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 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 treatment (surgery, chemotherapy) and combination, 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, (confine of lymphoma to the mucosa and submucosa) from stage I (tumour extension beyond the submucosa according to Radaszkiewicz and colleagues) nor discrimination of stage II, (involvement of regional lymph nodes) from stage II, (node involvement beyond the regional area, as assessed in the Munsell modification) is sufficiently serving the demand for documenting all features of lymphoma. To meet these shortcomings, the Lugano classification was constructed by Roitman and colleagues introducing stage IIIE for “serosa penetration” without lymph node involvement into the Ann Arbor system. This represents a change in meaning of stage II that originally indicated lymph node involvement. Therefore, the Lugano system is causing more confusion than benefit.

TNM staging for tumours of epithelial origin has also been proposed as an alternative in gastrointestinal lymphoma to describe localised disease. The “T” part of this system pertains to the anatomical structure of the organs and sufficiently fulfills the requirements for staging of local extent of the disease.

The European Gastro-Intestinal Lymphoma Study Group (EGILS) is a multidisciplinary group of investigators, including clinical investigators such as gastroenterologists, medical oncologists, radiotherapists, and pathologists, as well as basic researchers such as cellular and molecular biologists. Several groups from the UK, France, Germany, the Netherlands, Spain, and Austria now take part and have come together regularly since 1999 to discuss and study subjects in epidemiology and molecular and cell biology of gastrointestinal lymphoma. Clinical protocols and trials have been developed and performed 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.

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

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>TX</td>
<td>Lymphoma extent not specified</td>
</tr>
<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>T1sm</td>
<td>Lymphoma confined to submucosa</td>
</tr>
<tr>
<td>T2</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 lymph nodes beyond the regional area</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 sites (eg, peritoneum, pleura) or organs (eg, tonsils, parotid gland, ocular adnexa, lung, liver, spleen, kidney, breast etc.)</td>
</tr>
<tr>
<td>BX</td>
<td>Involvement of bone marrow not assessed</td>
</tr>
<tr>
<td>B0</td>
<td>No evidence of bone marrow involvement</td>
</tr>
<tr>
<td>B1</td>
<td>Lymphomatous infiltration of bone marrow</td>
</tr>
</tbody>
</table>

TNM Clinical staging: status of tumour, node, metastasis, bone marrow

pT: Histopathological staging: status of tumour, node, metastasis, bone marrow

pN: The histopathological 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 Retumor 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 caecocolic 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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A Morger Medical Department 1, Technical University Hospital, Dresden, Germany
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 with high serum levels of CA19.9. In particular, obstruction of the body of the pancreas without hepatobiliary abnormalities can cause a fall in CA19.9. Unspecific elevation of CA19.9 in serum is frequently associated with inflammatory hypersecretion of CA19.9 by normal epithelial cells.

The constituent flavonoids of tea beverage are known to be potent antioxidants. It appears that flavonoids impact on a wide range of molecular targets that influence cell growth and pathways of angiogenesis. In this case, heavy tea consumption lowered blood levels of CA19.9 by serum levels of CA19.9 were ruled out before a link with the tea beverage was suspected. After tea consumption was stopped, a dramatic improvement occurred and the patient became asymptomatic. The diagnosis of tea intoxication was confirmed by the fall in serum CA19.9 levels. The patient reported no clinical abnormalities and gained the 2 kg weight loss. Laboratory data demonstrated normal ranges for electrolytes, liver function tests, and 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 found mild enlargement of the body of the pancreas without hepatobiliary abnormalities. Endoscopic ultrasonography showed no pancreatic malignancy or biliary abnormalities. The pancreas was homogenous and mildly enlarged in the body without pathological significance. Spirometry, chest x ray, bronchoscopy, and bronchoalveolar lavage fluid examination were normal.

The patient was advised to stop tea consumption. Four weeks later she became symptom free and gained the 2 kg weight loss. Another serum CA19.9 assay showed a considerable drop in levels to 42 U/ml. A rechallenge test was then attempted. The patient restarted tea consumption as previously. Four weeks later, serum 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
CA19.9 (sialyl lewis-a) is a monosialoganglioside with small increases in a number of benign diseases, with highest levels in pancreatic adenocarcinoma, hepato-cellular, 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 with 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 in 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.

Table 1 Non-malignant causes of CA19.9 elevation (median value)

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Serum CA19.9 level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild elevation</td>
</tr>
<tr>
<td></td>
<td>(&lt;200 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>

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

References

5. Liu H, Ye H, Ruskone-Fourmestraux A et al. 7(11,13) is a marker for all stage gastric MALT lymphomas that will not respond to H pylori eradication. Gastroenterology 2002;122:1286-94.
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 characterized 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 kg/m²), serum triglyceride level (TG mg/dl), and serum total cholesterol (T-ch mg/dl) were similar in patients with or without GH deficiency (table 1). The significance of the per cent hepatic steatosis was evaluated by χ² test. BMI, TG, and T-ch were analysed using an unpaired Student’s t-test. The male patient on whom we performed a liver biopsy was diagnosed with GH deficiency 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 significant risk factor for hepatic steatosis and NASH. 

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References


Expression of thioipurine 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-thiouric acid 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. Previous reports in Caucasians have shown that approximately 0.3% are homozygously and 10% heterozygously deficient in TPMT. One study...
suggests that the frequency of individuals with the mutant TPMT allele, TPMT*3A, is lower in Southwest Asians than in Caucasian; allele causes reduced enzyme activity in vitro. 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 homozygously 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 serum 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 leptin concentrations that are at least two fold greater than men. Therefore, leptin 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.

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References

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 only 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. 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, firstly, 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 colon 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 soon.

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:3 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 in which both markers are performed prospecively, may carry the least risk of methodological flaws.

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BOOK REVIEW

Radiological Imaging of the Small Intestine

Springer Verlag are producing an extensive range of books entitled Radiological Imaging 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 outstanding and the more than 800 in this Springer publications, the illustrations are its distinguished editor. This is now a very latest in the line. Professor NC Gourtsoyiannis the general editorship of Professor AL Baert, range of books entitled Springer Verlag are producing an extensive process where observations need to be care- for any radiologist with an interest in the fore remains more of a deductive radiological “knee jerk” response of biopsy it. It there- well as the fact that it is not so easy to initiate elude it. Study of the small bowel however to be congratulated on producing a superb copy from both ends of the alimentary tract; a “second bite of the cherry”. Is there competition in the market place and how does this book hold up? The inevita- ble comparison has to be made to Clinical Imaging of the Small Intestine by Heirinlig, Maglinite, 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 Radiologi- cal Imaging is primarily a European work with some distinguished North American con- tributors. Indeed, two of the editors of Clinical Imaging are authors. Both books go for the same layout—that is, chapters which are based on their own experiences, this can both MR imaging—the reader can appre- ciate the difference. It has to be said that both books fulfil their remit extremely well but your reviewer, if he had to choose, would opt for spending the extra Euros. Professor Gourtsoyiannis and his team have to be congratulated on producing a superb book that graces the swelling ranks of medical radiology. A must for every radiology depart- ment and a continuing source of information 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 Général Leclerc, 91401 Orsay Cedex, France. Tel: +33 169 867 779; fax: +33 169 867 739; email: tavitian@lil.jrf.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 sugges- tions on pathophysiology, diagnostics, and therapy of liver diseases in ten programmes. Further details: Dr 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, Depart- ment 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@cmcc.nhs.uk

Falk Symposia—New Findings in Pathogenesis and Progress in Management of IBD
This symposium will be held on 16–18 June 2003 in Brus- sels, Belgium. Further details: Nancy Beauf- reez, 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: beaufrezn@ulb.ac.be

Gastroenterology and Endotherapy: XXIst European Workshop
This will be held on 16–18 June 2003 in Brus- sels, Belgium. This annual meeting will be held on 7–10 July 2003 in Edin- burgh, UK. Further details: Conference Secretariat, The AGBI at the Royal College of Surgeons of Edinburgh, 35–43 Lin- coln’s Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acpgbi@asgbi.org.uk; website: www.acpgbi.org.uk

The Association of Coloproctology of Great Britain & Ireland
This annual meeting will be held on 7–10 July 2003 in Edin- burgh, UK. Further details: Con- ference Secretariat, The AGBI at the Royal College of Surgeons of Edinburgh, 35–43 Lin- coln’s Inn Fields, London WC2A 3PE. Tel: +44 (0)20 7973 0307; fax: +44 (0)20 7430 9235; email: acpgbi@asgbi.org.uk; website: www.acpgbi.org.uk

European Helicobacter Study Group (EHSG)
This meeting, on Helicobacter infections and gastrroduodenal pathology, will be held on 3–6 September 2003 in Stockholm, Sweden. Fur- ther details: Professor Torkel Wadstrom, President- EHSG, Lund University, Depart- ment of Infectious Diseases & Medical Micro- biology, Division of Bacteriology, Solvagatan 23, SE-223 62 Lund, Sweden. Tel: +46 46 173 241; fax: +46 46 152 564; email: torkel.wadstrom@mmi.lnb.se; website: www.w-helicobacter.org

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