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LETTERS

Scintigraphic assessment of SO dysfunction

As diagnosis of sphincter of Oddi (SO) dysfunction may require perendoscopic manometric assessment of the sphincter, which is an invasive technique carrying a significant risk of pancreatitis, non-invasive scintigraphy has been proposed as an alternative diagnostic method. Craig et al (Gut 2003;52:352–7) reported their experience in assessing SO dysfunction in post-cholecystectomy patients with scintigraphy using the hepatic hilum-duodenum transit time (HHDT) of Cicala and colleagues1 as an abnormal threshold.

In the study of Craig et al, scintigraphic data after cholecystokinin octapeptide (CCK-OP) infusion were compared with SO basal pressure, the latter recorded at manometry in the absence of any stimulus.

Craig et al concluded that none of the above scintigraphic variables was sufficiently sensitive to diagnose SO dysfunction identified at SO manometry. Although it is evident that the scintigraphic method of Craig et al used in their study was indeed poorly sensitive, the conclusion of their study cannot be extended to interpret the validity of scintigraphy, as the scoring system of Sostre and colleagues2 that only HHDT performed with the correct description of the method it was pointed out was repeatable, compared with other scintigraphic variables related to hepatic uptake and clearance of radiolabelled bile.3 Noticably in the original HHDT method, ROIs were the heart, right external liver parenchyma, hepatic hilum, and duodenum, whereas in Craig’s study ROIs were the right lobe of the liver and the common bile duct (CBD), which are not sufficient reference sites to construct a transit measurement from the hepatic hilum to the duodenum. In addition, an ROI placed on the CBD cannot correctly discriminate CBD activity from that of the major bile ducts at the hepatic hilum.

Furthermore, analysis of time threshold performed on visual assessment of three minute composite images implies a measurement error of several minutes. In addition, assessment of radiolabelled bile at different sites cannot be performed correctly with a visual method that is significantly delayed in comparison with assessment based on time activity curves (TAC), provided that frame timing is adequate. For example, this methodological error is clearly described and illustrated in figure 3 of the article by Cicala and colleagues,4 with the original description of the HHDT method where the time period assessed with static images at 2.5 minutes apart was delayed by 2.5 minutes in comparison with the assessment based on the TAC constructed on 15 second frame timing. Use of a cholecystokinin stimulus to assess HHDT is also questionable as CCK is known to affect hepatic bile secretion and SO motor activity, either accelerating the transit of bile under normal conditions or slowing it in the case of SO paradoxical response. Madaux and colleagues,5 comparing measurement of HHDT without any stimulus and after caerulein administration, showed that the 89% sensitivity of the test without the cholecystokinin stimulus decreased to 0% after the cholecystokinin stimulus. In addition, it is not acceptable to derive any conclusions on test sensitivity from a comparison between a scintigraphic assessment performed after a cholecystokinin stimulus and manometric recordings performed in the absence of a stimulus. Craig et al’s study refers to >9 minutes as an abnormal threshold of HHDT, as indicated in the study of Cicala and colleagues.6 Use of a reference threshold from another centre does not apply when a different technique is used. The technique of Craig et al should be validated with correct reference standards defined in a control group, which was lacking in their study.

Several studies have used timed but not comparable scintigraphic techniques to assess SO dysfunction by means of the hepatic duodenum transit time or a score (see table 1).

All but one of the studies in table 1 have compared scintigraphy with manometry and have shown a high specificity of the test and, with the exception of Craig et al, have also shown a satisfactory sensitivity in the absence of a cholecystokinin stimulus. Reliability of the HHDT test is supported by the following studies:

1) it is reproducible in asymptomatic controls (Cicala and colleagues1) and in patients with SO dysfunction (Cicala and colleagues2); and

2) it discriminates asymptomatic controls from SO dysfunction patients (Corazziari and colleagues3).

Finally, in common with the validation process used for SO manometry, validity of a scintigraphic diagnostic test for SO dysfunction is proved if the test (that is, HHDT or a score) normalises after, and predicts the outcome of, treatment of the abnormality that the test is designed to detect. Both of the above demonstrations for HHDT have been presented in the study by Cicala and colleagues.4

We are aware that information concerning reliability and outcome prediction of HHDT derive from a single group of investigators and confirmation studies with comparable techniques performed in other centres would be welcome. However, we would caution against making comparisons and drawing conclusions with techniques that are not comparable and have not been submitted to proper validation studies to ascertain their reliability.

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Universita La Sapienza, Rome, Italy

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References


Table 1 Scintigraphic tests for sphincter of Oddi (SO) dysfunction

<table>
<thead>
<tr>
<th>Reference No</th>
<th>Stimulus</th>
<th>Frame/t</th>
<th>Analysis</th>
<th>Reproducibility (controls/pts)</th>
<th>Versus manometry (no stimulus)</th>
<th>Versus sphincterotomy (sensitivity)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Sensitivity (%)</td>
<td>Specificity (%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Scintigraphic HHDT (choledochoscintigraphy)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1, 4, 5</td>
<td>None</td>
<td>15 s</td>
<td>TAC</td>
<td>Rel/rel</td>
<td>83</td>
<td>100</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>1 min</td>
<td>TAC</td>
<td>Not assessed</td>
<td>89</td>
<td>100</td>
</tr>
<tr>
<td>6</td>
<td>CRL</td>
<td>1 min</td>
<td>Static</td>
<td>Not assessed</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>Craig et al</td>
<td>CCK-OP</td>
<td>1 min</td>
<td>Static</td>
<td>Not assessed</td>
<td>13</td>
<td>95</td>
</tr>
<tr>
<td>Scintigraphic score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>CCK-OP</td>
<td>1 min</td>
<td>Static +TAC</td>
<td>Not assessed</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Craig et al</td>
<td>CCK-OP</td>
<td>1 min</td>
<td>Static +TAC</td>
<td>Not assessed</td>
<td>38</td>
<td>90</td>
</tr>
</tbody>
</table>

CRL, caerulein; CCK-OP, cholecystokinin octapeptide; TAC, time activity curve; Rel, reliable; Pts, patients.


Proposal for a new histological grading system for post-treatment evaluation of gastric MALT lymphoma

Gastric MALT lymphoma (GML) development is closely associated with Helicobacter pylori infection cases. The majority of stage IE GML regress following H pylori eradication but assessing cure of the disease requires long follow-up. Residual lymphoid infiltrate in post-treatment gastric biopsies can be very difficult to interpret and histological criteria for the diagnosis of minimal residual disease or complete remission are not clearly defined. Molecular follow up by polymerase chain reaction (PCR) for the rearranged immunoglobulin heavy chain variable region shows that persistent monoclonality in the absence of histological features observed in post-treatment gastric biopsies are due to the persistence of H pylori infection cases.

Thus histological evaluation of gastric biopsies remains the cornerstone to assess cure of the disease requiring long follow-up data using the new follow up system. Three to six sequential gastric biopsies were analysed for all patients with a mean follow up of 19 months after H pylori eradication therapy. Interobserver agreement evaluated by the weighted kappa value gave excellent results, with values over 0.84, indicating very good agreement among the seven observers. Assessing the lymphoma remission status is of great importance for clinical practice. Developing tools to evaluate residual disease are needed, not only for clinical practice but also to conduct clinical trials that aim to define therapeutic guidelines. We propose in this study a histological grading system for the evaluation of post-treatment gastric biopsies. Testing of this scheme in a small number of cases within the group developing this scheme has shown it to be highly reproducible. These results encourage further evaluation of this scheme on larger series, as well as investigation of its clinical significance and impact on clinical guidelines. In combination with molecular studies, this scheme could provide an interesting tool for the evaluation of residual disease in prospective studies on GML.

Table 1 GELA histological grading system for post-treatment evaluation of gastric MALT lymphoma

<table>
<thead>
<tr>
<th>Score</th>
<th>Lymphoid infiltrate</th>
<th>LEL</th>
<th>Stromal changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>CR (complete histological remission)</td>
<td>Absent or scattered plasma cells and small lymphoid cells in the LP</td>
<td>Absent</td>
<td>Normal or empty LP and/or fibrosis</td>
</tr>
<tr>
<td>pMRD (probable minimal residual disease)</td>
<td>Aggregates of lymphoid cells or lymphoid nodules in the LP</td>
<td>Absent</td>
<td>Empty LP and/or fibrosis</td>
</tr>
<tr>
<td>rRD (responding residual disease)</td>
<td>Dense, diffuse, or nodular extending around glands in the LP</td>
<td>Focal LEL or absent</td>
<td>Focal empty LP and/or fibrosis</td>
</tr>
<tr>
<td>NC (no change)</td>
<td>Dense, diffuse, or nodular</td>
<td>Present, “may be absent”</td>
<td>No changes</td>
</tr>
</tbody>
</table>

MM, muscularis mucosa; LP, lamina propria; SM, submucosa; LEL, lymphoepithelial lesions.

References


Remarkable resemblance in the mode of transmission of HCV infection among haemodialysis patients and IVDAs

Hepatitis C virus (HCV) infection is widespread among patients on long term haemodialysis (HD) and among intravenous drug abusers (IVDAs). However, there appear to be striking similarities in the mode of...
transmission between the two groups as both are at high risk for parenterally transmitted HCV infection.

The indispensable requirement of having a vascular access site possibly adds to the risk of acquiring HCV infection among patients on long term HD through nosocomial transmission, especially in high HCV prevalence units. Preliminary data suggest that among various types of vascular access used for HD, arteriovenous fistula and polytetrafluoroethylene grafts which require extra skillful handling, perhaps play a more significant role in the transmission of HCV than permanent or temporary central venous catheters. Sharing of dialysis equipment, equipment reuse, and the physical proximity of an infected patient during HD are additional important factors incriminated in the transmission of HCV in the busy HD unit. Gilli et al. reported an outbreak of HCV in an Italian HD facility when a multidose saline vial was contaminated with blood from a HCV infected patient in a multidose saline vial was contaminated and reported an outbreak of HCV occurring when blood from a HCV infected patient during HD are additional important factors incriminated in the transmission of HCV in the busy HD unit. Gilli et al. reported an outbreak of HCV in an Italian HD facility when a multidose saline vial was contaminated with blood from a HCV infected patient in a multidose saline vial.

Another recent study from the USA reported an outbreak of HCV occurring when a multidose saline vial was contaminated with blood from a HCV infected patient in a Florida hospital. Breakdown in the implementation of standard infection control safety measures recommended by the CDC is essentially responsible for the rapid rise in HCV infection among HD patients worldwide.

Likewise, sharing of contaminated equipment (needles and syringes) among IVDAs is also the primary concern attributed to the continuous increase in HCV infection. However, in a recent report from Kolkata, India, dissemination of HCV accelerated, paradoxically from a baseline prevalence rate of 17% in 1996 to 66% in 2002 and to 80% during the next year, regardless of the supply of fresh needles and syringes on a daily basis, under the supervision of trained field workers, with the equipment being taken away from IVDAs on the next day after use. Most of the IVDAs did not share their syringes or needles, none the less, they shared the multidose vials of the drugs. Indirectly sharing of the drug ampoules suggested contaminated body fluids/blood being the means of transmission of HCV through direct access to the blood circulation, including an infected patient during HD as both groups did not reach statistical significance.

What will all the technicians do? However, Dr Neale has perhaps been a little too cautious in condemning colonoscopy in recurrent melaena without a definite cause in case No 1, with a marked microcytic anaemia and very doubtful indication. However, in case No 1, with a marked microcytic anaemia and recurrent melena without a definite cause in the upper gastrointestinal tract, many would regard visualisation of the colon by whatever means prudent, although given the likely comorbid nature of the patient, a non-invasive test might have been better, depending on resources. It is clear that in both of these typical cases appropriate thought had not gone into the diagnostic approach but also interestingly that in these days of great service pressures it was possible to perform oesophagogastro-duodenoscopy and colonoscopy before haematologic estimations. In both cases the colonoskopist must share the blame for taking an overly technical view; bowel preparation may be rather unpleasant but that is no reason to continue and perform an unnecessary investigation if the patient turns up on a list. I would echo Dr Neale’s thoughtful suggestion that we need to concentrate more on efficacy and safety of care.

Author’s reply I thank Dr Beales for his comments. Clearly, it is always easy to be wise in retrospect. However, we teach students to make a diagnosis by listing the positive findings and linking these to build a coherent diagnosis. In case No 1, the house officer noted ascitis ingestion, melaena, a hard liver edge, and thrombocytopenia. He suggested cancer of the gut with hepatic metastases. This was reasonable enough even though it did not include thrombocytopenia.

The next logical step might have been scanning of the upper abdomen in which case splenomegaly would have been added to the list and from there it was only a short step to hepatic cirrhosis and possible reinterpretation of the erythematous/exudative gastritis.

We also teach that patients be told the risk-benefit ratio of any procedure. Frank melaena is a rare presentation of cancer of the colon and the risk of colonoscopy is perhaps 0.2%.

I leave the reader to decide if the present day gastroenterologist should concentrate on honing specialist technical skills to gather information or should develop as a consultant who weighs the evidence as it unfolds.

G Neale
Clinical Safety Research Unit, Academic Department of Surgery, 10th Floor QEQM, St Mary’s Hospital, Praed St, London W2 1NY, UK; g.neale@ic.ac.uk
Entrants must be 40 years old or less on 31 December 2004 but 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 Glasgow in March 2004. Applications (TWENTY COPIES) should be made to the Honorary Secretary, British Society of Gastroenterology, 3 St Andrews Place, London NW1 4LB by 1 December 2003.

British Society of Gastroenterology

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- 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.

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British Society of Gastroenterology

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Successful applicants will be expected to provide a brief written report to the Endoscopy Committee of the outcome of their visit.

Application forms are available from the British Society of Gastroenterology Office, 3 St Andrew's Place, London NW1 4LB. Email: bsg@mailbox.ulcc.ac.uk

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

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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

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This meeting will be held on 15–19 April 2004 in Berlin, Germany. Further details: Secretariat, c/o Kenses International, 17 rue du Cendrier, PO Box 1726, CH-1211 Geneva, Switzerland. Tel: +41 22 908 0488; fax: +41 22 732 2850; email: info@easl.ch; website: www.easl.ch/easl2004

- Deadline for receipt of abstracts: 16 November 2003
- Deadline for early registration 10 February 2004