Reproducible test for detecting Helicobacter pylori in frozen samples

Retrospective investigations of Helicobacter pylori in stored samples might help us to understand the aetiologic role of Helicobacter pylori in different lesions with which it has been associated.1 Helicobacter pylori, however, is a fastidious micro-organism, and it is difficult to store it without pronounced loss of viability.2 Some investigators have studied the presence of Helicobacter pylori in stored samples using stained sections,3 but no one has evaluated the reproducibility of a method to detect Helicobacter pylori in stored samples. The aim of this study was to evaluate prospectively the accuracy of a simple and rapid urease test and culture techniques for the detection of Helicobacter pylori in infected frozen gastric biopsy specimens. A total of 32 antral gastric biopsy specimens were included in the study. The biopsy specimens were ground in 1 ml of 20% sterile glucose chilled transport medium with the pH adjusted to 6.8. A fraction of each specimen was processed for culture and rapid urease test assay. The remaining parts of the homogenized specimens were divided into three groups and stored at −70°C. The groups were reanalysed at different times (two, four, and six months after endoscopy) by repeating the rapid urease test and culture.

The culture was performed on Skirrow's Campylobacter agar (Difco Laboratories, Detroit, Michigan), trypticase soy agar (Difco) supplemented with 7% horse blood, 1%, IsoVitalex, and with or without antibiotics (10 mg/l vancomycin, 5 mg/l trimethoprim lactate, and 2500 IU/l polymixin B), chocolate agar, and Thayer-Martin agar (BBL) with 1%, IsoVitalex and 2%, haemoglobin. Plates were incubated at 37°C for up to seven days in a Campy Pak (BBL) generating microaerophilic or a Campylobacter gas generating Kit (Oxoid Ltd, London, England). The rapid urease test used in a modification of that used by Ahrndt et al,4 and recently validated in our laboratory,5 can be performed with very small amount of homogenised tissue incubated at 55°C.

Characteristics of 45 patients with deep lower limb venous thrombosis

The acute phase response with fever, a neutrophilic leucocytosis and increased concentrations of plasma proteins may be induced by different infectious, ischaemic, immunological, malignant, or traumatic stimuli. One of the most practical methods for monitoring it is to quantify the serum C-reactive protein (CRP) concentration.1 The purpose of the present work was to compare the acute phase response in different types of deep lower limb thrombosis.

Forty-five patients with deep lower limb thrombosis verified by ascending phlebography were studied. These patients did not have the rapid urease test was 100%, reproducible at any time, a decrease in the activity of the enzyme was observed.

The culture of the stored specimens that were Helicobacter pylori positive in the fresh analysis identified the micro-organism in 14 of 16 (87%) samples after two months, 14 of 15 (93%) after four months, and 10 of 11 (91%) after six months. The viability deter-

Maximum serum C-reactive protein concentration in all cases of acute deep lower limb venous thrombosis. Group 1, iliofemoral, group 2, femoro-popliteal thrombosis. The horizontal lines denote means of serum CRP. The difference between the medians was significant p = 0.02 (Mann-Whitney U-test).

any other cause for the acute phase response. Treatment protocol at our hospital includes the combination of heparin and coumarine and only coumarine after discharge.

Periperal blood leucocyte counts on admission were determined with an automated, and auxiliary temperatures and CRP concentrations were monitored throughout. Serum CRP was determined by an immunoturbidimetric method (Orion Diagnostica, Espoo, Finland). The detection level for CRP at our hospital varied from <10 mg/l to <15 mg/l during the period concerned, but in those cases a serum CRP value of 5 mg/l was used for statistical purposes. Data were compared with the non-parametric Student's t test, Mann-Whitney U-test, and Pearson's correlation analysis.

The 19 cases with iliofemoral thrombosis (group 1) were older than the 26 with the femoro-popliteal thrombosis, and the serum CRP response was higher in the former, but no such difference was seen in leucocyte responses or auxiliary temperatures (table). Age itself did not seem to explain the differences in CRP response between these two groups when examined by Pearson correlation analysis (r = 0.24, p = 0.24 in group 1; r = 0.24, p = 0.33 in group 2).

The analysis of the individual responses showed more than one third (35%) of the 26 patients in group 2 to have no CRP response (figure). However, while only three had a temperature of

Characteristics of 45 patients with deep lower limb venous thrombosis

<table>
<thead>
<tr>
<th>Tidal/Popliteal (n = 26)</th>
<th>Iliofemoral (n = 19)</th>
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</thead>
<tbody>
<tr>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
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<tr>
<td>Age (years)</td>
<td>52.6 (14.7)</td>
</tr>
<tr>
<td>Leucocytes*</td>
<td>8.7 (1.9)</td>
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<tr>
<td>Test†</td>
<td>30.7 (6.5)</td>
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</tbody>
</table>

* = maximum of peripheral blood leucocyte counts, x 10⁷/μl.
† = maximum of auxiliary temperature, °C.

Acute phase response and deep lower limb venous thrombosis

The acute phase response with fever, a neutrophilic leucocytosis and increased concentrations of plasma proteins may be induced by different infectious, ischaemic, immunological, malignant, or traumatic stimuli. One of the most practical methods for monitoring it is to quantify the serum C-reactive protein (CRP) concentration.1 The purpose of the present work was to compare the acute phase response in different types of deep lower limb thrombosis.

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> 37.5°C and only two out of 25 patients had a leucocyte reaction of >11.0 × 10^9/l during acute phase of thrombosis. Serum CRP did not rise in three cases out of the 19 (15.8%) patients in the iliofemoral group, and only one patient had a temperature of > 37.5°C or a leucocyte response of > 11.0 × 10^9/l. The sensitivity of serum CRP to show the presence of thrombosis was 72% (95% confidence interval 54 to 90%) in group 1 and 32% (12 to 52%) in group 2.

A recent study has proposed a 100% sensitivity of serum CRP for detecting deep venous thrombosis. According to our results, deep lower limb venous thrombosis seems to elicit only a slight or even undetectable acute phase response. The serum CRP was normal in more than one third of the cases with the thrombosis in the tibial or popliteal veins and undetectable in about 16% of the cases of iliofemoral thrombosis. In our series the sensitivity of serum CRP was low in the cases with the thrombosis in the tibial or popliteal vein (32%), but clearly higher (77%) in the cases of iliofemoral thrombosis. Perhaps most cases had femoral or iliac vein thrombosis. This could partly explain the differences between their results and ours. Whole blood leucocyte counts and axillary temperatures were usually normal in both of our groups. Thus deep lower limb thrombosis seems to be a weak inducer of the acute phase response and some other cause for induction of the acute phase response should be considered if serum CRP concentration is over 100 mg/l.

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To try to cover the whole of contemporary knowledge on cancer in a series of 10 books is an audacious project. Hans Kaiser has gathered together nine distinguished colleagues and has tried to do the impossible. There is much in these volumes that will be of relevance to those interested in the biology of human, animal, and plant neoplasia. It should be said at the outset that it is rather difficult to identify the target audience for this series, and that such a project is inevitably going to suffer from the staggering rate at which new data are generated and interpretations of old data change.

In the first volume (Fundamental aspects of cancer. RH Goldfarb, ed), Goldfarb and colleagues have reviewed much of the basic information regarding the biology of cancer seen in most standard texts. In this and in the other volumes some contributions are rather esoteric and some poorly written. For example, what does the sentence, "Phylogeny is the accumulation of major ontogenies in the sense of hologenesis" mean? The mechanisms of carcinogenesis are reviewed in volume 2 (Mechanisms of carcinogenesis. EK Weissberger, ed) but it is inevitable that the rapidly growing field of molecular oncology has ensured that many of the chapters are already rather dated. The burgeoning field of antioncogenes and tumour suppressor genes is scarcely mentioned. In the third and fourth volumes (volume 3 Influence of tumour development on the host. LA Liotta, ed; volume 4, Influence of the host on tumour development. RB Herberman, ed), the interactions between host and tumour are considered. Liotta’s review of the mechanisms of cancer invasion and metastasis is masterful, yet has recently been covered in many other reviews. Similarly, Nicholson’s coverage of the tumour cell surface is admirable, but has also been covered elsewhere.

The major part of the fifth volume (volume 5, Comparative aspects of tumour development. HE Kaiser, ed) is devoted to comparisons of taxonomy and morphology in tumours from different species, including plants. There are fascinating accounts of tumours in molluscs, arthropods, patterns of spread in fish and amphibians, cancer in reptiles and detailed discussion of better known tumours such as...