tion of the four hour RUT for the detection of Campylobacter pylori. Two hundred and fifty six mucosal biopsy specimens were taken from the gastric antrum and duodenum for a four hour RUT, histological examination, and culture. Histology specimens were stained with Giemsa stain and examined for Campylobacter pylori. Specimens for culture were placed in 0·5 ml of 20% glucose, homogenised, and a drop taken for immediate Gram staining and the remainder placed on blood agar and campylobacter medium with Skirrow supplement. Plates were incubated in a microaerophilic environment at 37°C for six days. Campylobacter pylori colonies were examined for the presence of oxidase and urease and confirmation was obtained by Gram stain of the culture. One biopsy specimen (about 0·02 mg) was homogenised in 0·9% saline 0·5 ml, inoculated into 2% urea broth, incubated at 37°C, and examined after four hours. There was an excellent correlation between this modified four hour RUT, histological results, and culture. The table gives results of all biopsy specimens showing four hour RUT specificity at 100% and sensitivity at 89%. Specificity and sensitivity for antral biopsy specimens was 100% and 97% respectively.

False negative results for four hour RUT may be due to low numbers of bacteria within the specimen. We serially diluted Campylobacter pylori and inoculated dilutions into RUT broth which was incubated at 37°C and 50°C. We observed that the RUT test became positive within one hour at bacterial concentrations of >10^3 colony forming units/ml (cfu/ml) when incubated at 37°C and of >10^4 cfu/ml at 50°C. All biopsy specimens in which Campylobacter pylori could be detected showed evidence of acute gastritis or duodenitis.

These results indicated that the specificity of our test was higher than the CLO-Test (100% vs 97%) and that a positive four hour RUT is sufficient evidence to start treatment if appropriate. Preliminary results suggest that incubation at a higher temperature (50°C) may further enhance the sensitivity of the four hour RUT.

D Vaira,* J Holton‡

Results of antral and duodenal biopsy specimens

<table>
<thead>
<tr>
<th>Histology</th>
<th>Culture</th>
<th>Four hour RUT</th>
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<tbody>
<tr>
<td>Positive</td>
<td>91</td>
<td>63</td>
</tr>
<tr>
<td>Negative</td>
<td>165</td>
<td>191</td>
</tr>
<tr>
<td>Total</td>
<td>256</td>
<td>254</td>
</tr>
</tbody>
</table>

Letters to the Editor

References

1 Langenberg ML, Tygat GJN, Schipper MEI, Rietra PJGM, Zanen HC. Campylobacter-like organism in the stomach of patients and healthy individuals. Lancet 1984;i:1348.

Des-gamma-carboxyprothrombin and hepatoblastoma

Increased serum α fetoprotein is used as a diagnostic marker of hepatoblastoma in children, but in some cases, the serum α fetoprotein is normal.1 We wanted to know if the des-gamma-carboxyprothrombin concentration (DCP), a newly described marker of hepatocellular carcinoma in adults,2 could be used in the diagnosis of hepatoblastoma.

Raised plasma concentrations of DCP, the des-gamma-carboxyalted form of prothrombin (factor II), are seen in vitamin K deficiency, in patients taking oral anticoagulants, and in most patients with hepatocellular carcinomas.23 We assayed DCP value by a previously described method1 using staphylocoagulase (upper limit of normal, 15 mU/ml) in three children with histologically confirmed hepatoblastoma. None had received treatment. DCP, α fetoprotein, and vitamin K concentrations are shown in the table. The prothrombin time and serum vitamin K concentrations (normal range 0·2-0·8 μg/l) were normal in all three children.

The increased plasma DCP concentrations in these three patients was not due to vitamin K deficiency, and presumably results from the same (unknown) mechanism, observed in adult hepatocellular carcinoma. We found normal DCP values in other liver diseases of children associated with an increased α fetoprotein: hereditary tyrosinemia (n = 2), giant cell hepatitis (n = 4), fulminant hepatitis (n = 1), and neonatal hepatic necrosis with hepatic regeneration (n = 2).

Other studies are required to determine the respective value of DCP and α fetoprotein as assays for markers of hepatoblastoma in diagnosis and treatment, but we suggest that this marker may be useful in rare cases of hepatoblastomas with normal serum α fetoprotein. It should be noted that in adults with hepatic carcinoma, there is a weak correlation between increased DCP and increased α fetoprotein, the two markers thus being to some extent complimentary.

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References


Table α fetoprotein, DCP, and vitamin K concentrations in hepatoblastoma

<table>
<thead>
<tr>
<th></th>
<th>α-fetoprotein (μU/ml)</th>
<th>DCP (mU/ml)</th>
<th>Vitamin K (μg/l)</th>
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<tr>
<td>6 month old male infant</td>
<td>335 000</td>
<td>400</td>
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<tr>
<td>13 year old girl</td>
<td>600 000</td>
<td>70</td>
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<tr>
<td>6 year old girl</td>
<td>450 000</td>
<td>110</td>
<td>0·40</td>
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