NATIONAL CONGRESS OF DIGESTIVE DISEASES

22-27 November 1997 - Bologna (Italy)

ABSTRACTS OF SCIENTIFIC PRESENTATIONS
171. Lack of correlation between Helicobacter pylori infection and both primary and secondary Raynaud phenomenon

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It has been recently reported that H. pylori gastric infection is strongly correlated with primary Raynaud phenomenon1. In order to confirm this observation, we decided to assess the prevalence of this infection in a larger population of patients with both primary and secondary Raynaud phenomenon. Methods: We evaluated consecutively 18 patients with primary (16 F and 2 M, mean age 41 ± 18 yrs) and 26 patients with secondary Raynaud phenomenon (26 F, mean age 53 ± 16 years). They were compared with 28 and 30 control subjects, respectively, matched for age and sex. Primary Raynaud phenomenon was characterized by the absence of underlying diseases and laboratory findings. The diagnosis of secondary Raynaud phenomenon was based on the presence of architectural abnormalities of the nailfold capillary bed (assessed by videocapillaroscopy), clinical aspects of associated connective tissue diseases and serological abnormalities including antinuclear, antitopoisomerase I (Scl 70) and anticientromere antibodies. H. pylori infection was diagnosed by urea breath test, which was performed collecting duplicate samples before and 30 min after the ingestion of 13C labelled urea dissolved in 150 ml 0.033 mol/L citric acid (Cortex Italia, Milano). Analysis of breath samples was performed by isotope ratio mass spectrometry and a value of delta-over-baseline higher than 5‰ was considered positive. Chi-square test was used for statistical comparison. Results: The proportions of H. pylori infection in both primary and secondary Raynaud phenomenon did not differ from those of respective controls (8/18 = 44% vs 10/28 = 35% and 15/26 = 57% vs 16/30 = 53%). Conclusions: Our data do not confirm the already reported high association between primary Raynaud phenomenon and H. pylori infection. The same lack of correlation is true for secondary Raynaud phenomenon. Differences in age and diagnostic criteria may be responsible for the discrepancy between our results and those previously published. However, the evaluation of anti-H. pylori antibodies is under way in all patients.

References

173. Duodenogastric reflux and bile flow rates in cholecystectomized patients with functional dyspepsia


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Background: Several patients with gallstone disease referred, after cholecystectomy, dyspeptic symptoms or abdominal discomfort. Aim: The purpose of this study was to evaluate whether a duodenogastric reflux and an anomaly of the bile flow could be demonstrated in such patients. Methods: Twenty two cholecystectomized patients, without endoscopic and ultrasound evidence of organic complications of the biliary tree and with negative cholestasis laboratory tests, were studied. 10 patients were asymptomatic (control patients) and 12 had a persisting functional dyspepsia (symptomatic patients). The two groups of patients were matched for age and sex. The duodenogastric reflux and the filling and emptying kinetics of the biliary tree were studied by sequential cholecintigraphy using a 99mTc trimethyl-Bromo-IDA and a computerized gamma-camera. The exam was performed for sixty minutes during fasting state and for sixty minutes after a standard cholecystokinetic meal.
**Results:** (m ± se)  

<table>
<thead>
<tr>
<th></th>
<th>Control pts</th>
<th>Symptom. Pts</th>
<th>P</th>
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<tbody>
<tr>
<td>Fasting reflux index</td>
<td>1.92 ± 0.5</td>
<td>12.1 ± 3</td>
<td>0.03</td>
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<tr>
<td>Postprandial reflux index</td>
<td>3.1 ± 0.6</td>
<td>27.8 ± 12.3</td>
<td>0.01</td>
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<tr>
<td>Hepatic peak time (min)</td>
<td>16.9 ± 0.2</td>
<td>11.3 ± 0.4</td>
<td>&lt; 0.002</td>
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<tr>
<td>Hepatic half-life (min)</td>
<td>52.5 ± 3.0</td>
<td>37.6 ± 2.6</td>
<td>0.001</td>
</tr>
<tr>
<td>Duodenal Time (min)</td>
<td>9.9 ± 0.4</td>
<td>8.1 ± 1.9</td>
<td>0.5</td>
</tr>
<tr>
<td>Biliary peak time (min)</td>
<td>49.6 ± 7.6</td>
<td>37.2 ± 9.9</td>
<td>0.1</td>
</tr>
<tr>
<td>Biliary half-life (min)</td>
<td>93.3 ± 7.4</td>
<td>69.3 ± 4.2</td>
<td>0.008</td>
</tr>
<tr>
<td>% Residual biliary activity at 90°</td>
<td>50.5 ± 1.1</td>
<td>28.0 ± 4.2</td>
<td>0.07</td>
</tr>
<tr>
<td>% Residual biliary activity at 120°</td>
<td>23.8 ± 4.6</td>
<td>10.0 ± 1.7</td>
<td>0.02</td>
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</table>

**Observations:** In cholecystectomized patients with functional dyspepsia a more rapid hepatic clearance of the compound, a faster biliary wash-out and a greater duodenogastric reflux were found. **Conclusions:** In some cholecystectomized patients the greater bile output in duodenum is likely to be a risk of a pathologic duodenogastric reflux; 2) the duodenogastric reflux may be responsible for dyspeptic symptoms, but this could only be the epiphemomenon of functional changes which may involve both the biliary-duodenal-motility function and the hepatic secretory function.

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**174. Lamivudine treatment in HBV liver transplanted patients**

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Orthotopic liver transplantation (OLTx) in HBV patients is conditioned by the high recurrence rate due to the endogenous reinforcement of the graft liver. Here we report our experience in the management of the new antiviral drug lamivudine (Lam) in HBV liver transplanted pts. Five pts, transplanted for end stage liver disease (HBsAg positive, HBV-DNA negative) were treated with Lam (100 mg/day orally) for a mean treatment period of 9.2 months (range 3-15) in an open compassionate use basis. Among them, 2 pts received Lamivudine also in the pre-transplant setting in order to obtain the normalization in DNA. In 3 pts, DNA negative and not treated before OLTx, antiviral therapy was administered because of recurrent hepatitis. HBV recurrence occurred a median of 5.3 months after OLTx despite immunosuppression and was diagnosed as the appearance of high serum transaminases level (mean reported ALT 10 times the upper normal limit) and the positivity in HBsAg and HBV-DNA. Histologic evidence of B hepatitis was obtained in all patients. After 1.2 months of therapy HBV-DNA (mean level 2076 pg/ml-range 83-6056) completely disappeared in two of them; the pt with the higher titre reported a reduction in 90% of DNA in only 1 month while ALT normalized in all the patients. In the other two pts, who received Lamivudine and were DNA negative at the moment of transplant, treatment was continued prophylactically for at least 1 year post-OLT in conjunction with HBIG. They neither presented hypertransaminasemia nor positivity in HBV-DNA post-OLTx. No adverse events or rejection were reported also during the long-term treatment thus excluding the occurrence of viral mutation during therapy. In conclusion, Lam is efficacious and well tolerated even during long-term treatment in HBV recurrent grafted pts and seems that the association with HBIG efficaciously prevents viral recurrence. Further studies are needed to investigate the viral clearance also at tissue level and to define the best treatment duration.

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**175. Assessment of intestinal permeability in patients with ileal pouch (IPAA)**

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A small number of pts affected by Ulcerative Colitis (UC) does not respond to medical treatment and needs surgery. In such IPAA pts, intestinal permeability to 51-Cr-EDTA directly administered into the pouch, has been recently found to be augmented irrespective of pouchitis. Aim of this study was to investigate intestinal permeability - through a double probe oral load test - in UC patients before (T0), two months (T1) after the proctocolectomy with the patients having a protected IPAA, and two further months (T2) after the abolition of the protective ileostomy, when the pouch was functioning. Data are drawn from eight patients with UC (F = 6, M = 2; mean age ± SEM = 40.8 ± 10.2) with a disease duration ranging 2-15 yrs (mean ± SEM = 7.9 ± 6.3 yrs). All patients underwent surgery for failure of medical treatment. Small intestine permeability was evaluated by Cellulose-Mannitol (CE/MA) test at T0, T1 and T2. Cellulose (5g) and Mannitol (2g), given as oral isomolar load, were recovered in urine collected for 3 hrs and the CE/MA of % urinary excretions was considered as an index of intestinal permeability. From 32 healthy controls in our lab the CE/MA upper limit of normal was calculated to be 0.028 (mean ± 2SD). Basal values of CE/MA were in the normal range in 5 pts, and higher in the other 3 pts (T0: median 0.012, range 0-0.130). At T1 most pts (6 of 8) showed CE/MA values similar or reduced in respect to their basal values (T1: median 0.011, range 0-0.066). One patient died due to heath crisis, before completing the study. In 6 of 7 patients who have completed the study up today, with functioning pouches in absence of any macroscopic signs of pouchitis, the CE/MA was augmented in two cases (i.e.: 0.029 vs 0.014 and 0.056 vs 0.010). These results seem to show that permeability of both small intestine and pouch is unaltered in most cases; further follow up controls might suggest evolution to pouchitis in pts with altered permeability of an apparently normal pouch.

**References**