Anti-HCV IgM as predictor of response to interferon therapy in schistosomal patients with chronic hepatitis C

The appearance of anti-HCV IgM in chronic hepatitis C is associated with viral replication and ongoing hepatitis.1,2 We evaluated anti-HCV IgM as predictor of response to combined therapy with interferon-α2b (Intron A; Schering-Plough, Kenilworth, NJ, USA) 3 MU three times weekly subcutaneously with 1200 mg daily oral dose of ribavirin (Rebetron; Schering-Plough) in 29 patients with chronic hepatitis C (age range 42-58 y; 17 men) and 28 patients with concomitant hepatitis C virus (HCV) and bilharzial infections (15 men) of similar demographic data. ALT, anti-HCV IgM (EIA, Equipar Diagnostic, Italy) and HCV RNA (RT-PCR based assay, COBAS Amplipcr HCV Monitor 2.0, Roche Molecular System, USA) were assessed in serum pre-treatment, at the end of treatment and 6 months later (Table).

Table: ALT levels and HCV RNA load in the two treatment groups

<table>
<thead>
<tr>
<th></th>
<th>HCV alone</th>
<th>HCV + bilharzial infections</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALT (U/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-treatment</td>
<td>96.7 (15.3)</td>
<td>111.8 (32.2)*</td>
</tr>
<tr>
<td>12 mo</td>
<td>72.2 (40.2)</td>
<td>86.8 (52.7)</td>
</tr>
<tr>
<td>18 mo</td>
<td>99.5 (49.2)</td>
<td>113.2 (48)</td>
</tr>
<tr>
<td>HCV RNA (x 10^5 copies/mL)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-treatment</td>
<td>6.5 (5.7)</td>
<td>13.9 (8)*</td>
</tr>
<tr>
<td>12 mo</td>
<td>4.7 (6.4)</td>
<td>8.82 (7.7)*</td>
</tr>
<tr>
<td>18 mo</td>
<td>7.6 (8.2)</td>
<td>14.7 (7.3)*</td>
</tr>
</tbody>
</table>

p **<0.05, **<0.01 as compared to group with HCV infection alone

The end-of-treatment (ETR) response, as defined by normalization of ALT and loss of detectable serum HCV RNA, in bilharzial compared to non-bilharzial patients (37.9% and 28.6%, respectively; p=0.2), and sustained response 6 mo later (24.1% and 10.7%, p=0.18) were similar. Pre-treatment anti-HCV IgM was detected in 12 non-bilharzial patients (41.3%) compared to 16 bilharzial cases (57.1%). Most pretreatment anti-HCV IgM seropositive patients elicited no ETR (non-responders 10 non-bilharzial and 15 bilharzial patients). Patients who responded relapsed 6 mo later. On the other hand, 10 patients with pre-treatment anti-HCV seronegative non-bilharzial and 9 bilharzial patients had ETR. Sustained response 6 mo later was seen in 7 and 3 patients, respectively.

A major limitation of our study has been the absence of genotype testing; the dose of interferon used is also empiric. Based on the finding that none of the anti-HCV IgM seropositive patients pretreatment could achieve ETR it can be suggested that anti-HCV IgM when positive may be used as a predictor of poor response to combined interferon and ribavirin therapy irrespective of the presence of concomitant bilharzial infection.

Moutaz F Derbala, Aliaa M Amer,*
Inas E El Defrawi**

Departments of Gastroenterology and Hepatology, *Laboratory Medicine and Pathology, and **Microbiology, Hamad Medical Corporation and Theodore Bilharz Research Institute, Egypt

References

Correspondence to: Dr. Derbala, P O Box #3050, Doha, Qatar. E-mail: moutazderbala@hotmail.com, derbalamf@yahoo.com

Diagnostic value of gall bladder wall thickness in patients with ascites

Gall bladder wall thickening is related not only to intrinsic gall bladder pathology but also to non-biliary pathology like hepatic dysfunction, hypoalbuminemia, ascites, hepatitis, congestive heart failure, renal disease, AIDS, malignancy and sepsis.1 Several investigators have suggested that ultrasonography may be useful for distinguishing benign from malignant ascites based on thickness of the gall bladder wall.2,3 Malignant ascites is usually associated with normal gall bladder wall thickness whereas benign etiologies are associated with abnormally thickened gall bladder wall.

Over a one-year period, gall bladder wall thickness was prospectively evaluated in 94 consecutive patients with sonographically established ascites. All studies were carried out with real-time ultrasonographic scanner using 3.5 MHz and 7.5 MHz transducers. Sonographically collapsed gall bladders were excluded, as were cases with cholelithiasis, focal thickening or clinically suspected biliary pathology. Gall bladder wall thickness was determined on the posterior wall with measurement perpendicular to the wall. Wall thickness 3 mm or more was considered thickened.1 Patients were categorized into portal hypertension ascites and non-portal hypertension ascites based on