Exacerbation of chronic liver disease due to hepatitis B surface antigen after delta infection

SIR,—The report by Dr G Raimondo and others (12 March, p 845), in which the δ agent was implicated as the causal factor in three cases of fatal hepatitis, together with the fulminant cases reported by Smedile et al. are of much concern since they show that, in Italy at least, δ acquired by a hepatitis B carrier is severely detrimental to the patient.

We have evidence, however, that the simultaneous acquisition of δ with hepatitis B does not cause an appreciable increase in severity in drug abusers with serologically acute hepatitis B. We have examined 212 drug abusers who developed hepatitis during a continuing outbreak of hepatitis B among drug abusers in Dublin and five long term drug abusing carriers of hepatitis B surface antigen (HBsAg) for δ antigenaemia, using enzyme immunoassay after detergent treatment, and for anti-δ, using enzyme immunoassay with serum as the δ antigen source; the specificity of our test has been confirmed on a sample by Dr Rizzetto (table).

There was no significant difference in the clinical findings between those with or without S antigenaemia, and there has been one fatal case in this group so far. Six of the 212 patients have become HBsAg carriers and four of these have anti-δ; the fatality occurred in one of these 14 months after the acute episode. Additionally, five have been carriers for several years; we have not found S antigen retrospectively in stored sera from these five, but three have anti-δ. It is not known whether these three acquired their δ infections simultaneously with hepatitis B or subsequently.

It appears that in Ireland the acquisition of S and hepatitis B simultaneously is not usually associated with severe hepatitis, and this is similar to the experience in Sweden, where Moestrup et al. (8 January, p 87) found only one fulminant case in 37 with acute hepatitis B and δ. The severe δ associated hepatitis reported from Italy seems to have occurred only in patients who were carriers of hepatitis B

Results of tests for presence of δ markers, δ antigen, and anti-δ in 217 drug abusers

<table>
<thead>
<tr>
<th>Drug abusers tested</th>
<th>No with δ markers</th>
<th>No with δ antigen</th>
<th>No with anti-δ</th>
<th>Seropositive conversions</th>
</tr>
</thead>
<tbody>
<tr>
<td>212 with acute hepatitis B</td>
<td>72 (34%)</td>
<td>53 (25%)</td>
<td>19 (9%)</td>
<td>14</td>
</tr>
<tr>
<td>5 HBsAg carriers</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>–</td>
</tr>
</tbody>
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and who acquired δ subsequently. The possibility of an ethnic (genetic) factor in δ pathogenicity must also be considered.

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