Table Diagnoses and results of the radionuclide tests in the 50 infants

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No</th>
<th>DISIDA</th>
<th>Rose-Bengal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No excretion</td>
<td>Rapid excretion</td>
</tr>
<tr>
<td>Biliary atresia</td>
<td>21</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Bile duct plug</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>α1 Antitrypsin deficiency</td>
<td>6</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Hepatic disease</td>
<td>22</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>50</td>
<td>23</td>
<td>6</td>
</tr>
</tbody>
</table>

Discussion

A skilfully interpreted percutaneous liver biopsy specimen and 131I Rose-Bengal faecal excretion test have been shown to discriminate between biliary atresia and hepatic disease once genetic disorders have been excluded. Neither test alone is reliable. The use of 131I as a tracer in infancy is not ideal as it is a beta emitter with a long half life, although the dose is very small. The test also involves collection of stools for three days, carefully avoiding urinary contamination that may invalidate the result. This study suggests that DISIDA scintigraphy after treatment with phenobarbitone is as accurate as the Rose-Bengal test. Whether it is necessary to use phenobarbitone is uncertain. It was used in this study because five of seven infants with hepatic disease had no excretion of paraisopropyl IDA when tested without phenobarbitone but did excrete after treatment with phenobarbitone.

DISIDA scintigraphy has the drawback that it is subjective, allowance having to be made for isotope in the kidneys and bladder, and time consuming. Assessment of hepatic uptake of DISIDA may increase diagnostic accuracy. Nevertheless, when used in conjunction with ultrasonography, tests for infective and genetic causes of liver damage, and percutaneous liver biopsy DISIDA scintigraphy represents a considerable advance in distinguishing complete from partial cholestasis in early infancy.

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References


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Splenuncleulectomy in thrombocytopenic purpura

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SUMMARY In six patients with thrombocytopenic purpura not cured by splenectomy platelets fell to $<150 \times 10^9/l$ within six weeks of splenectomy. They 'failed to respond'. Two underwent splenuncleulectomy without improvement. Splenuncleulectomy offered little chance of improvement to published cases who failed to respond but may well be helpful after true relapse.
A small proportion of children with persisting idiopathic thrombocytopenic purpura are not cured by splenectomy. It has long been postulated that enlarged accessory spleens may be responsible for recurrent thrombocytopenia in some splenectomised patients, and splenunculectomy has cured or improved several such patients.

With this aim we reinvestigated seven patients who were not cured by splenectomy and offered splenunculectomy to those with persistent bruising in whom an accessory spleen was shown.

Methods

Platelets were labelled with $^{111}$In-oxine under aseptic conditions. Eight MBq of labelled platelets were injected intravenously and anterior and posterior abdominal scintigraphs obtained at one hour and one, two, and three days with a gammacamera.

Patients

The seven patients were aged between 4 and 12 years (mean 8 years) at the onset of the purpura. Splenectomy was performed between one and four years (mean two years) after diagnosis. The response to splenectomy in the seven patients is shown in the Figure. Case 7 became thrombocytopenic and started bruising again at one year after splenectomy. Indium scans were obtained between two and 19 years (mean 10 years) after splenectomy. Enlarged accessory spleens were found in cases 1 to 4. Cases 3 and 4 underwent splenunculectomy. Neither was improved.

Discussion

The response to splenunculectomy in cases 3 and 4 was disappointing and caused us to review more critically the published reports. The role of splenunculectomy in patients with idiopathic thrombocytopenic purpura who have not been cured by splenectomy has been obscured by loose use of the term 'recurrent' and by widely differing interpretations of what constitutes a remission. For example, Wallace et al required a platelet count $>150 \times 10^9/l$ at an unspecified time after splenectomy, and Verheyden et al., in their survey of cases in the English reports, included two whose post splenectomy 'remissions' lasted only 12 and 30 days.

In our experience some rise in the platelet count occurs in almost all children with idiopathic thrombocytopenic purpura immediately after splenectomy, but it is unlikely to be sustained unless the count exceeds $300 \times 10^9/l$ in the first week. It is also clear from the patients shown here that such a rise does not guarantee cure. We classified empirically those patients not cured by splenectomy whose counts fell below $150 \times 10^9/l$ within six weeks, irrespective of their peak concentrations, as having...
Pancytopenia caused by iron-dextran

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**SUMMARY** Pancytopenia after intramuscular iron-dextran treatment occurred in an infant with Down's syndrome. Haematological abnormalities recurred on subsequent challenge. Positive migration inhibiting factor and mast cell degranulation tests support an allergic pathogenesis for the pancytopenia. These side effects have not been reported previously.

Severe, even fatal, allergic reactions are known to occur after parenteral administration of iron-dextran (Imferon). We describe a patient who developed thrombocytopenia, leucopenia, and haemolytic anaemia after intramuscular injection of iron-dextran. These haematological abnormalities recurred on subsequent challenge.

**Case report**

A 1 year old girl with Down's syndrome was admitted to our department. Blood count showed an iron deficiency anaemia with haemoglobin concentration 83 g/l, packed cell volume 0.28, mean corpuscular volume 71 fl, leucocytes 8 x 10⁹/l (70% neutrophils, 12% band forms, 16% lymphocytes, 2% monocytes), and platelets 165 x 10⁹/l. Serum iron concentration was 2.3 mM/l (12.7 µg%). Iron binding capacity was 55 mM/l (307 µg%). Intramuscular iron-dextran (30 mg/kg) was given in three divided doses over six days because of feeding problems and poor compliance. Ten days later the haemoglobin concentration rose to 98 g/l and the reticulocyte count was 4.8%. The leucocyte count was 9.7 x 10⁹/l with normal differential count, and the platelet count was 180 x 10⁹/l. She was discharged home but was readmitted to hospital one month later. Her blood count showed pancytopenia with haemoglobin concentrations 74 g/l, packed cell volume 0.24, mean corpuscular volume 85 fl, leucocytes 3.3 x 10⁹/l (49% neutrophils, 4% band forms, 43% lymphocytes, 2% monocytes, and 2% eosinophils), and platelets 50 x 10⁹/l (Figure). Direct and indirect Coombs' tests were negative, her glucose-6-phosphate concentration was normal, and...