Congenital parvovirus infection

I M R Wright, M L Williams, B J Cohen

Abstract

A case of congenital parvovirus (B19) viraemia with associated thrombocytopenic purpura and platelet antigen incompatibility in an infant is reported. Results of laboratory investigations indicated that the baby was infected in utero.

A recent report has shown evidence of the sequelae of congenital parvovirus (B19) infection. Others have shown haematological consequences of infection in an otherwise normal infant. We report a confirmed intrauterine infection with B19 in an infant, which was associated with thrombocytopenic purpura and platelet antigen incompatibility.

Case report

An infant boy was born at 37 weeks’ gestation after a normal delivery. Pregnancy had been
Investigations for parvovirus B19

<table>
<thead>
<tr>
<th>Time sample taken after birth</th>
<th>B19 IgM*</th>
<th>B19 IgG*</th>
<th>B19 DNA†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 weeks</td>
<td>&gt;100</td>
<td>35</td>
<td>Negative</td>
</tr>
<tr>
<td>1 week</td>
<td>31</td>
<td>&gt;100</td>
<td>Negative</td>
</tr>
<tr>
<td>Baby</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Birth</td>
<td>33</td>
<td>6/1</td>
<td>Positive</td>
</tr>
<tr>
<td>2 weeks</td>
<td>&gt;100</td>
<td>6/5</td>
<td>Negative</td>
</tr>
<tr>
<td>4 weeks</td>
<td>6</td>
<td>19</td>
<td>Negative</td>
</tr>
</tbody>
</table>

*Radioimmunoassay units: <1 negative, 1-3 equivocal, and >3 positive.
†Dot blot hybridisation assay with digoxigenin labelled probe.

uneventful with no evidence of maternal illness. He weighed 2600 g and his Apgar scores were 8 at birth and 10 at five minutes. Three hours after birth the infant was noted to have a widespread petechial rash over his trunk, limbs, and palate and larger bruises were evident over bony prominences. There was no hepatosplenomegaly and examination was otherwise unremarkable. Initial investigations showed a haemoglobin concentration of 169 g/l, white cell count 20.7 x 10^9/l (74% neutrophils), and platelets 19.0 x 10^9/l. Clotting screen and bacterial infection screen were normal. Recovery was uneventful except for moderate jaundice requiring four days of phototherapy. By day 8 the platelet count was 550 x 10^9/l. Cranial ultrasound scan was normal.

Further serological investigation showed the father to be platelet antigen (PlA^1) positive and the mother PlA^1 negative. The mother also demonstrated anti-PlA^1 antibody. The infant was PlA^1 positive and his serum cross reacted with that of his mother.

Discussion

The results of B19 investigations (table) indicated intrauterine infection with a high concentration of specific IgM and a rising concentration of specific IgG in mother and infant. In addition DNA studies demonstrated a viraemia in the first infant sample that cleared as the IgM rose. The recent survey by the Public Health Laboratory Service on B19 in pregnancy suggested a 30% intrauterine infection rate but no case showed the presence of the viraemia in the infant and no thrombocytopenia was noted. A recent report showed viraemia in a 2 month old infant but this was persistent and associated with erythroid hypoplasia. Maternal infection was not reported in this case. Others have demonstrated antiplatelet antibodies and thrombocytopenic purpura in previously normal children with viral infections, including parvovirus. This case shows not only confirmed intrauterine infection with B19 and transient viraemia in the newborn infant, but the association with thrombocytopenic purpura and platelet antigen incompatibility; this has not previously been demonstrated with congenital parvovirus B19 infection.

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