Case Report

Concomitant parvovirus B19 and cytomegalovirus infections after living-related renal transplantation

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Introduction

Parvovirus B19 was first identified serendipitously in human sera from blood donors in 1975. Shortly thereafter it was found to be responsible for erythema infectiosum in children and transient aplastic anemia in patients with chronic haemolysis. It can also cause chronic marrow hypoplasia or aplasia in patients with immunosuppression [1–3]. It has recently been reported to cause anemia following cadaveric renal transplant [4–12]. We describe an unusual case of concomitant parvovirus B19 and cytomegalovirus (CMV) infection in a patient who underwent living related renal transplantation. The literature on parvovirus B19 and CMV infection in renal transplant is reviewed.

Case

The patient was a 21-year-old Caucasian female with end-stage renal disease (ESRD) secondary to focal segmental glomerulosclerosis (FSGS) since 1993. She underwent a living, related renal transplant from her father in October 1995. Both the patient and her father were CMV-antibody positive before the transplant. She required transfusion of 4 units packed red cells perioperatively. The patient received cyclosporin A 200 mg bid, mycophenolate mofetil 1000 mg bid and methylprednisolone (later prednisone) 60 mg daily. OKT3 infusion 5 mg daily was also given for 3 days postoperatively. She also received prophylactic oral acyclovir 200 mg every 8 h and sulfamethoxazole/trimethoprim 800/160 mg daily. She was discharged on day 9 with serum creatinine 1.1 mg/dl. An episode of early rejection was successfully abrogated with radiation of 900 centigrays over 6 fractions over 2 weeks. The patient was readmitted on day 80 with abnormal liver function, ALT 182 IU/l, AST 136 IU/l, alkaline phosphatase 218 IU/l and γ-GT 1188 IU/l. Her serum creatinine was 1.1 mg/dl, WBC was 5800/µl, Hgb 10.4 g/dl, MCV 93.1 fl and platelet 176 000/µl. Hepatitis A, B and C virus antibodies were all negative. Parvovirus B19 was first identified serendipitously in human sera from blood donors in 1975. Shortly thereafter it was found to be responsible for erythema infectiosum in children and transient aplastic anemia in patients with chronic haemolysis. It can also cause chronic marrow hypoplasia or aplasia in patients with immunosuppression [1–3]. It has recently been reported to cause anemia following cadaveric renal transplant [4–12]. We describe an unusual case of concomitant parvovirus B19 and cytomegalovirus (CMV) infection in a patient who underwent living related renal transplantation. The literature on parvovirus B19 and CMV infection in renal transplant is reviewed.

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There appears to be a single and stable antigenic type of B19 virus such that infection is usually followed by life-long immunity. Parvovirus B19 usually occurs between the ages of 4 and 11 years. Over 50% of adults are seropositive indicating past infection [13]. However, in patients with chronic haemolysis or with an immunosuppressed state, parvovirus B19 infection may become protracted and cause prolonged anemia and even aplastic anaemia [3].

Parvovirus B19 has been reported in different solid-organ transplants such as renal, heart and liver [14–16]. Parvovirus B19 infection is relatively uncommon in bone marrow transplantation presumably because of the routine use of intravenous immunoglobulin that contains protective parvovirus B19 antibodies [16]. The case reports of parvovirus B19 infections complicating renal transplants are summarized in Table 1. Most cases involved cadaveric donors except the current case. The age of the recipients ranged from 7 to 62 years with a median of 45 years and the donors ages ranged from 18 months to 57 years.

While the mode of transmission of parvovirus infection in a normal host is mostly through the respiratory tract, it is less defined in the transplant setting. Donor tissues were investigated in cases 2, 4 and 5. Parvovirus DNA was detected only in the donor serum of case 5. In case 4, while parvovirus DNA was not detected in the donor serum, it was present in the patient serum taken just before transplant suggesting that the infection was not donor-related. Similarly in cases 6 and 9, parvovirus infection was documented in the patient sera stored prior to renal transplant. It is conceivable that the mode of transmission may or may not be donor related. Reactivation of parvovirus infection, as with cytomegalovirus infection, is also possible.

Table 1. Case reports of parvovirus B19 infection after renal transplant

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Sex/age</th>
<th>Donor source/age</th>
<th>Onset of unexplained anaemia after transplant</th>
<th>Documentation of parvovirus infection after transplant</th>
<th>Remarks</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M/57</td>
<td>Cadaveric/—</td>
<td>4</td>
<td>4</td>
<td>Remit spontaneously</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>M/62</td>
<td>Cadaveric/1.5</td>
<td>52</td>
<td>52</td>
<td>Donor tissue-negative for parvovirus DNA; treated successfully with IVIG</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>M/7</td>
<td>Cadaveric/—</td>
<td>8</td>
<td>8</td>
<td>HHV6 coinfection; remit spontaneously</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>M/48</td>
<td>Cadaveric/—</td>
<td>3</td>
<td>0</td>
<td>Donor serum-negative for parvovirus DNA; treated successfully with IVIG</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td>F/47</td>
<td>Cadaveric/20</td>
<td>2</td>
<td>2</td>
<td>Donor serum-positive for parvovirus DNA; treated successfully with IVIG</td>
<td>8</td>
</tr>
<tr>
<td>6</td>
<td>F/57</td>
<td>Cadaveric/—</td>
<td>27</td>
<td>—13</td>
<td>Treated successfully with IVIG</td>
<td>9</td>
</tr>
<tr>
<td>7</td>
<td>M/62</td>
<td>Cadaveric/5</td>
<td>6</td>
<td>6</td>
<td>Treated successfully with IVIG</td>
<td>10</td>
</tr>
<tr>
<td>8</td>
<td>M/38</td>
<td>Cadaveric/57</td>
<td>4</td>
<td>4</td>
<td>Treated successfully with IVIG</td>
<td>10</td>
</tr>
<tr>
<td>9</td>
<td>F/43</td>
<td>Third cadaveric/29</td>
<td>4</td>
<td>—4</td>
<td>Treated successfully with IVIG; associated with FSGS</td>
<td>10</td>
</tr>
<tr>
<td>10</td>
<td>M/13</td>
<td>Cadaveric/—</td>
<td>6</td>
<td>6</td>
<td>Treated successfully with IVIG</td>
<td>11</td>
</tr>
<tr>
<td>11</td>
<td>M/37</td>
<td>Cadaveric/—</td>
<td>11</td>
<td>11</td>
<td>Treated successfully with IVIG</td>
<td>12</td>
</tr>
<tr>
<td>Current</td>
<td>F/20</td>
<td>Living related/45</td>
<td>12</td>
<td>12</td>
<td>CMV coinfection; remit spontaneously</td>
<td>—</td>
</tr>
</tbody>
</table>
ciclovir in protecting against HSV-1- and HSV-2-induced cutaneous lesions in a mouse model. It was suggested that the depletion of guanine monophosphate by mycophenolate mofetil favours the inhibitory action of the drugs on the viral DNA polymerase. A further prospective study is required to elucidate the role of mycophenolate mofetil in CMV infections in kidney transplantation.

References

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