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Severe Hemolytic Anemia Post–Renal Transplantation Produced by Donor Anti-D Passenger Lymphocytes: Case Report and Literature Review

Craig D. Ainsworth, Mark A. Crowther, Darin Treleaven, Denise Evanovitch, Kathryn E. Webert, and Morris A. Blajchman

The passenger lymphocyte syndrome (PLS), often associated with immune-mediated hemolytic anemia after solid organ and hematopoietic stem cell transplantation, is the result of concomitant transplantation of donor lymphocytes along with the donor allograft. Antibodies directed against recipient red blood cells (RBCs) are frequently found in ABO-mismatched solid organ transplants; however, passenger lymphocyte–mediated hemolysis due to Rh-incompatible antibodies has only rarely been reported. In this report, we present a case of severe hemolytic anemia related to the PLS in an ABO-matched renal allograft recipient. The recipient’s blood type was A Rh(D) positive; and the donor, who had been previously alloimmunized, was A Rh(D) negative. The renal allograft recipient’s hemoglobin abruptly decreased on postoperative day 12 in the setting of a newly positive direct antiglobulin test and anti-D antibodies in the plasma. The patient required intermittent RBC transfusions for ongoing hemolysis during the first 6 months post–renal transplant. Of all reported cases of anti-D–mediated PLS, our patient would seem to have been one of the most severe, as indicated by a nadir hemoglobin of 41 g/L and the need for 23 U of transfused RBCs. A hemolytic anemia occurring after organ transplantation should raise the possibility of donor-derived antibodies directed against the recipient RBCs. Passenger lymphocyte syndrome–associated hemolysis is occasionally severe as in our case, but can be effectively treated with compatible RBC transfusions.

CASE REPORT

A 49-YEAR-OLD woman with renal failure secondary to polycystic kidney disease underwent deceased donor renal transplantation. Her pretransplant blood type was A Rh(D) positive. The recipient’s medical history included hemodialysis dependence for 6 years, hypertension, right radical nephrectomy, type 2 diabetes mellitus controlled with oral hypoglycemics, remote viral meningoencephalitis, recurrent urinary tract infections, inguinal hernia repair, and 3 previous pregnancies. The donor was A Rh(D) negative and had been alloimmunized previously, as evident by the presence of anti-D detected in a blood sample drawn before her death.

No blood products were administered during an uneventful transplant operation. Postoperatively, the patient received immunosuppression including mycophenolate mofetil, tacrolimus, and prednisone. The postoperative course was initially complicated by delayed graft function requiring antithymocyte globulin (5 daily doses) and hemodialysis.

Before transplant, the patient’s hemoglobin was 109 g/L. Her hemoglobin remained stable until postoperative day (POD) 5 when it fell without explanation to 71 g/L and further decreased to 55 g/L by POD 11. Despite transfusion of 1 U of red blood cells (RBCs), the hemoglobin declined to 41 g/L on POD 12. A computed tomographic scan of the abdomen and pelvis as well as renal ultrasound failed to demonstrate evidence of bleeding. Additional investigations confirmed the presence of hemolysis, including a positive direct antiglobulin test (DAT) (2+ with immunoglobulin G [IgG] only) as well as hyperbilirubinemia and elevated lactate dehydrogenase, peaking at 100 μmol/L and 352 U/L, respectively. Anti-D antibodies were subsequently detected in the patient’s plasma, reactive to a titer of 32 by saline indirect antiglobulin test (SIDAT), from POD 19 to 37. Anti-D was also eluted from the patient’s RBCs. Anti-D was
undetectable by SIDAT by day 82; however, it was still detectable by MTS Gel (Micro Typing Systems, Pompano Beach, FL) until approximately POD 150 (Table 1). During some of this time (POD 15-46), the patient’s blood group typed as A Rh(D) negative. The patient received 11 U of Rh-negative RBCs from POD 11 to 19 and 23 U in total during the 6 months posttransplant. She received her last RBC transfusion on POD 181 and her hemoglobin has remained stable since (Fig 1). In addition to the RBC transfusions, the patient received ongoing transplant immunosuppression and intravenous immunoglobulin on 2 occasions during the period of acute hemolysis. Figure 1 summarizes the recipient’s clinical course.

DISCUSSION

Allograft passenger lymphocytes have been reported to cause hemolysis after solid organ and hematopoietic stem cell transplantation. This process, termed the passenger lymphocyte syndrome (PLS), results in immune-mediated hemolysis due to the concurrent transplantation of donor plasma cells, or B-lymphocytes, along with the allograft. These cells often produce antibodies that are reactive against recipient RBC antigens. Passenger lymphocyte syndrome–associated antibodies are detected relatively frequently; and the resultant antibody-induced hemolysis is a well-recognized consequence in ABO-mismatched solid organ transplants, occurring in 9% of kidney, 29% of liver and 70% of heart-lung transplants. However, in patients with ABO-compatible transplants, PLS-mediated hemolysis due to Rh-incompatible antibodies is exceedingly rare and not usually associated with clinically important hemolysis. Our case represents a particularly severe hemolytic disorder, associated with a transient switch from A Rh(D) positive (native) to A Rh(D) negative (donor) RBC phenotype, a

Table 1. Summary of the Serologic Investigations, Transfusion Requirements, and Hemoglobin Level in the Patient Described in This Report

<table>
<thead>
<tr>
<th>POD</th>
<th>Hb</th>
<th>Group</th>
<th>DAT</th>
<th>Product</th>
<th>Anti-D</th>
<th>Titer</th>
</tr>
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<tbody>
<tr>
<td>−1</td>
<td>112</td>
<td>A+</td>
<td>N/A</td>
<td>None</td>
<td>Negative</td>
<td>N/A</td>
</tr>
<tr>
<td>+2</td>
<td>85</td>
<td>N/A</td>
<td>N/A</td>
<td>150 mg anti-ATG</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>+3</td>
<td>86</td>
<td>N/A</td>
<td>N/A</td>
<td>150 mg anti-ATG</td>
<td>N/A</td>
<td>N/A</td>
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<tr>
<td>+4</td>
<td>84</td>
<td>N/A</td>
<td>N/A</td>
<td>100 mg anti-ATG</td>
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<td>N/A</td>
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<tr>
<td>+5</td>
<td>71</td>
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<tr>
<td>+6</td>
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<td>N/A</td>
<td>N/A</td>
<td>50 mg anti-ATG</td>
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<td>N/A</td>
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<tr>
<td>+8</td>
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<td>N/A</td>
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<tr>
<td>+11</td>
<td>55</td>
<td>A+</td>
<td>Positive (2+ with IgG)</td>
<td>1 U RBCs (A−)</td>
<td>Positive (2+ by MTS and weak by SIDAT)</td>
<td>0</td>
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<tr>
<td>+12</td>
<td>41</td>
<td>A+</td>
<td>Positive (2+ with IgG)</td>
<td>4 U RBCs (A−)</td>
<td>Positive (2+ by MTS)</td>
<td>N/A</td>
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<tr>
<td>+13</td>
<td>72</td>
<td>N/A</td>
<td>N/A</td>
<td>2 U RBCs (A−)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>+15</td>
<td>93</td>
<td>A−</td>
<td>Negative</td>
<td>2 U RBCs (A−)</td>
<td>Positive (2+ by MTS)</td>
<td>N/A</td>
</tr>
<tr>
<td>+19</td>
<td>91</td>
<td>A−</td>
<td>Negative</td>
<td>2 U RBCs (A−)</td>
<td>Positive (2+ by MTS)</td>
<td>32</td>
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<tr>
<td>+21</td>
<td>106</td>
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<tr>
<td>+33</td>
<td>94</td>
<td>A−</td>
<td>Positive (weak with IgG)</td>
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<tr>
<td>+35</td>
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<td>None</td>
<td>None</td>
<td>N/A</td>
<td>32</td>
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<tr>
<td>+46</td>
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<td>A+</td>
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<td>Positive</td>
<td>16</td>
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<td>+56</td>
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<td>Positive (weak with IgG)</td>
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<td>+57</td>
<td>63</td>
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<td>N/A</td>
<td>2 U RBCs (A−)</td>
<td>None</td>
<td>N/A</td>
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<tr>
<td>+61</td>
<td>86</td>
<td>N/A</td>
<td>N/A</td>
<td>None</td>
<td>N/A</td>
<td>4</td>
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<tr>
<td>+70</td>
<td>68</td>
<td>A+</td>
<td>Positive</td>
<td>1 U RBCs (A−)</td>
<td>Positive</td>
<td>1</td>
</tr>
<tr>
<td>+82</td>
<td>77</td>
<td>A+</td>
<td>Positive</td>
<td>1 U RBCs (A−)</td>
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<tr>
<td>+83</td>
<td>81</td>
<td>N/A</td>
<td>N/A</td>
<td>2 U RBCs (A−)</td>
<td>N/A</td>
<td>N/A</td>
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<tr>
<td>+124</td>
<td>63</td>
<td>A+</td>
<td>Positive (weak with IgG)</td>
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<td>Positive</td>
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<tr>
<td>+127</td>
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<td>N/A</td>
<td>N/A</td>
<td>1 U RBCs (A−)</td>
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<td>N/A</td>
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<tr>
<td>+131</td>
<td>N/A</td>
<td>A+</td>
<td>Positive (weak with IgG)</td>
<td>None</td>
<td>Positive (2+ by MTS; negative by SIDAT)</td>
<td>0</td>
</tr>
<tr>
<td>+133</td>
<td>81</td>
<td>N/A</td>
<td>N/A</td>
<td>2 U RBCs (A−)</td>
<td>None</td>
<td>N/A</td>
</tr>
<tr>
<td>+182</td>
<td>64</td>
<td>A+</td>
<td>Positive (1+ with IgG)</td>
<td>2 U RBCs (A−)</td>
<td>Negative</td>
<td>0</td>
</tr>
<tr>
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<td>91</td>
<td>A+</td>
<td>Positive (1+ with IgG)</td>
<td>None</td>
<td>Negative</td>
<td>0</td>
</tr>
<tr>
<td>+214</td>
<td>96</td>
<td>A+</td>
<td>Positive (1+ with IgG)</td>
<td>None</td>
<td>Negative</td>
<td>0</td>
</tr>
<tr>
<td>+228</td>
<td>123</td>
<td>A+</td>
<td>Positive (1+ with IgG)</td>
<td>None</td>
<td>Negative</td>
<td>0</td>
</tr>
</tbody>
</table>

Abbreviations: Hb, hemoglobin level; ATG, antithymocyte globulin; N/A, not available.
reflection of the large amount of A Rh(D) negative RBCs transfused.

The anemia induced by the PLS is usually abrupt in onset and associated with a positive DAT and the presence of serum antibodies in addition to evidence of hemolysis. Hemolysis usually becomes apparent between days 3 and 24 posttransplant and the timing does not appear to be dependent on the antibody specificity. In some cases, hemolysis precedes any serologic abnormalities by 1 to 2 days; and therefore, the diagnosis is considered only after a significant source of bleeding has been excluded. In ABO-incompatible transplants, the DAT results reveal approximately equal numbers with IgG, complement or both present on the RBCs. Most PLS cases caused by Rhesus antibodies are DAT IgG positive only. In our case, the initial precipitous drop in hemoglobin (79 to 55 g/L) occurred on POD 11, in concert with elevated lactate dehydrogenase (344 U/L) and bilirubin (67 μmol/L) levels as well as a positive DAT. Anti-D titers rose to 32 on POD 19. These findings, along with an unremarkable result of computed tomography of the abdomen and pelvis, alerted us to the diagnosis of immune-mediated hemolysis.

Most cases of passenger lymphocyte-mediated hemolysis are mild and self-limited; however, RBC transfusions are sometimes necessary. Most experience in patients with hemolysis requiring transfusion comes from case reports after ABO-mismatched organ transplants. In a review of all published cases up to 1991, Ramsey reported that the median number of RBC units transfused was 6.5 (range, 1-18) in 18 renal transplant patients. In 7 liver transplant patients, the range was 2 to 11 U; and in 4 heart-lung patients, it was 16 to 24 U. Transfusion requirements due to hemolysis from anti-ABO and/or anti-D have been comparable, although experience with Rh antibody-induced hemolysis has been limited. Ramsey identified 9 reported cases of hemolysis resulting from donor Rh antibodies, all involving renal transplants and most caused by anti-D (5 of 9 patients). In these patients, the number of RBC units transfused was 0 to 9, whereas the lowest hemoglobin levels ranged from 47 to 77 g/L (median, 62 g/L). Cases implicating anti-D in posttransplant hemolysis have subsequently been published, including 7 renal, 1 heart-lung, 1 pancreas, 1 kidney-pancreas, 6 liver, and 2 lung transplant recipients. The number of RBC transfusions for anemia resulting from such hemolysis was 0 to 17, 15, 14, 14, 0 to 20 and 1 to 5 U, respectively. One of the renal cases was a dual-kidney recipient who required 16 U of RBCs and had a nadir hemoglobin of 42 g/L, whereas the heart-lung transplant patient achieved a nadir hemoglobin of 38 g/L and required 15 U of RBCs. Considering all documented cases, transfusion requirements as high as 20 U of RBCs over the duration of immune hemolysis have been reported. Of all the reported cases of anti-D-mediated PLS (Table 2), our patient’s degree of hemolysis would seem to have been one of the most severe as indicated by a nadir hemoglobin of 41 g/L and the receipt of a total of 23 U of RBCs.
Table 2. Cases of Anti-D Rh-Incompatible Antibody-Mediated Hemolysis Post–Solid Organ Transplantation Reported in English Literature

<table>
<thead>
<tr>
<th>Ref</th>
<th>Organs</th>
<th>Donor/Recipient</th>
<th>Nadir Hb (g/L)</th>
<th>RBC Units Transfused*</th>
<th>DAT</th>
<th>Clinical course</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pomper et al</td>
<td>Kidney</td>
<td>B+/&gt;B+</td>
<td>42</td>
<td>16</td>
<td>Positive (IgG)</td>
<td>Hemolysis noted POD 13, Hb nadir POD 23, last transfusion POD 78</td>
</tr>
<tr>
<td>Ramsey et al</td>
<td>Kidney A†</td>
<td>O−/&gt;O+</td>
<td>73</td>
<td>4</td>
<td>3+ (IgG)</td>
<td>DAT positive POD 23, last transfusion approximately POD 28, nephrectomy for acute rejection POD 27</td>
</tr>
<tr>
<td></td>
<td>Kidney B†</td>
<td>O−/&gt;O+</td>
<td>90</td>
<td>0</td>
<td>3+ (IgG)</td>
<td>DAT positive POD 24, hemolysis not significant as bilirubin normal throughout Hemolysis described as &quot;intermediate,&quot; evidence of hemolysis and anti-D anti-A detected POD 15</td>
</tr>
<tr>
<td>Solheim et al, Soheim et al</td>
<td>Kidney A†</td>
<td>O−/&gt;A+</td>
<td>Not specified</td>
<td>4</td>
<td>Positive (IgG)</td>
<td>Hemolysis noted POD 78, positive DAT POD 23, last transfusion POD 28, nephrectomy for acute rejection POD 27</td>
</tr>
<tr>
<td></td>
<td>Kidney B†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>9</td>
<td>Positive</td>
<td>Hemolysis noted 2 wk posttransplant, last transfusion POD 28, nephrectomy for acute rejection POD 27</td>
</tr>
<tr>
<td></td>
<td>Kidney C†</td>
<td>O−/&gt;B+</td>
<td>Not specified</td>
<td>1</td>
<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
</tr>
<tr>
<td></td>
<td>Kidney D†</td>
<td>O−/&gt;B+</td>
<td>Not specified</td>
<td>2</td>
<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
</tr>
<tr>
<td></td>
<td>Kidney E†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>3</td>
<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
</tr>
<tr>
<td></td>
<td>Kidney F†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>5</td>
<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
</tr>
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<td>Kidney G†</td>
<td>O−/&gt;O+</td>
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<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
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<td>Kidney H†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
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<td>Kidney I†</td>
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<td>Not specified</td>
<td>8</td>
<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
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<tr>
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<td>Kidney J†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
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<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
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<td>Kidney K†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>10</td>
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<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
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<tr>
<td></td>
<td>Kidney L†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>11</td>
<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
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<tr>
<td></td>
<td>Kidney M†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>12</td>
<td>Positive</td>
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<td>Kidney N†</td>
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<td>Not specified</td>
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<td>Kidney O†</td>
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<td>Kidney P†</td>
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<td>Kidney Q†</td>
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<td>Kidney R†</td>
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<td>Kidney S†</td>
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<td>Kidney T†</td>
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<tr>
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<td>Kidney X†</td>
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<td>Kidney Y†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>24</td>
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<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
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<td>Kidney Z†</td>
<td>O−/&gt;O+</td>
<td>Not specified</td>
<td>25</td>
<td>Positive</td>
<td>Hemolysis noted POD 10, Hb stable by POD 20</td>
</tr>
</tbody>
</table>

Abbreviations: ICU, intensive care unit; D/C, discharged; POD, postoperative day.

* Number of RBC units transfused for anemia related to hemolysis.

† Cases included in original Ramsey review (fifth anti-D case excluded from table because it was not published in English literature).
Passenger lymphocyte syndrome generally does not persist longer than several months, as lymphocytes in general, including those transplanted along with the donor organ, are only capable of a brief period of proliferation and antibody production. However, some long-lived plasma cells can survive for longer periods of time in sheltered sites such as the bone marrow or inflamed kidney tissue. In ABO-unmatched kidney transplants, initially positive DATs became negative at 2 to 13 weeks posttransplantation (median, 5 weeks); and the last reactive serum detection of antibodies ranged from 2 to 23 weeks (median, 5.5 weeks). Anti-D–mediated hemolysis tends to last longer than hemolysis induced by ABO antibodies and can persist for up to 6 months, as seen in our patient.

The discovery of hemolytic anemia in the early posttransplant period should raise the possibility of donor-derived antibodies directed against recipient RBCs. Hemolysis in this setting may be severe and is potentially life threatening, but can be effectively treated by compatible RBC transfusions. Whether immunosuppression hastens resolution (as a result of lymphocytoxicity) or worsens hemolysis (by reducing immune surveillance of the abnormal population) is unknown. Recognizing this disorder as a potential cause of posttransplant anemia may allow for earlier diagnosis, avoidance of unnecessary investigation and prompt management.

Table 2 provides a summary of all reported cases of anti-D antibody-mediated hemolysis post–solid organ transplantation.

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