LETTER TO THE EDITOR

Alloimmune hemolytic anemia after liver transplantation from an ABO-identical and Rh-nonidentical donor in a patient with postpartum Budd–Chiari syndrome

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Severe alloimmune hemolytic anemia can occur as a rare complication after orthotopic liver transplantation (OLT) with an ABO compatible organ [1]. The transplantation of liver and herewith the transfer of lymphocytes from a rhesus-negative female donor, presumably immunized during deliveries of rhesus (D)-positive children, can lead to a severe hemolytic crisis in rhesus (D)-positive recipients. This phenomenon is described to occur with both solid organ and (prior to engraftment) bone or peripheral blood progenitor cell (PBPC) transplantation [2,3], even if the marrow is T-cell depleted. The perioperative use of donor group-specific blood may reduce ABO hemolysis.

We report on a 25-year-old woman (blood group O, rhesus D+, C+, c+, E+, e+; K1-negative), who presented with a progressive, therapy-refractory ascites, initially detected 4 months prior to admission, in the third week postpartum. A Budd–Chiari syndrome was diagnosed by ultrasound and computerized tomography scan and a prior liver disease was excluded on serologic and histologic criteria; thrombophilia as a potential cause of liver veins obstruction was ruled out. As liver function parameters and neurological status worsened after a portocaval shunt and the patient developed a hepato-renal syndrome, OLT was performed. The cadaveric donor organ was explanted from a 41-year-old female (blood group O, rhesus D-negative, mother of two children). Both parties were negative for atypical red blood cell antibodies and perioperative transfusion was performed with 10 packed cells group O, rhesus D positive and 9 units of fresh frozen plasma.

After an initially uncomplicated postoperative course, with normal hemoglobin values (Hb) and continuously decreasing transaminases and bilirubin levels, indicating normal graft function, the patient developed on day 8 a dramatic drop of serum Hb level to 5.5 g/dl without any indication of bleeding.

The direct anti-globulin test was strongly positive (1:1000) for immunoglobulin (Ig)G and was negative for complement, IgG1 and IgG3 anti-D titers were elevated (>1:100) (Fig. 1). Lactate dehydrogenase was 1024 U/l (ref. <480 U/l), haptoglobin <0.2 g/dl (0.3–2 g/dl), pointing to a hemolytic anemia. The bone marrow showed erythropoietic hyperplasia, according to the suspicion of hemolysis. The anemia was reversible after transfusion of 15 units of group O rhesus (D) negative blood. Hemoglobin, haptoglobin, alanine aminotransferase and aspartate aminotransferase returned to normal levels around day 20, total bilirubin levels subsequently decreased and normalized within 2 months (Fig. 2). Graft function was sustained by therapy with cyclosporine and methylprednisolone and the outcome at the 1-year follow-up control showed good transplant function.

This is, to the best of our knowledge, the first report on post-transplant hemolytic anemia in a young woman with postpartum Budd–Chiari syndrome. Our case of severe hemolysis illustrated high IgG anti-rhesus (D) production from passenger lymphocytes [4], with self-limited outcome [5]. As lymphocytes do not engraft and have a finite lifespan, the antibody titers declined with hemolytic consumption [6]. Although a survival benefit was described in ABO-identical vs. ABO-compatible liver transplants [7], strict rhesus compatibility cannot always be provided in emergency situations [8–10]. Careful monitoring of the direct antiglobulin test can lead to early recognition of post-transplant hemolysis.

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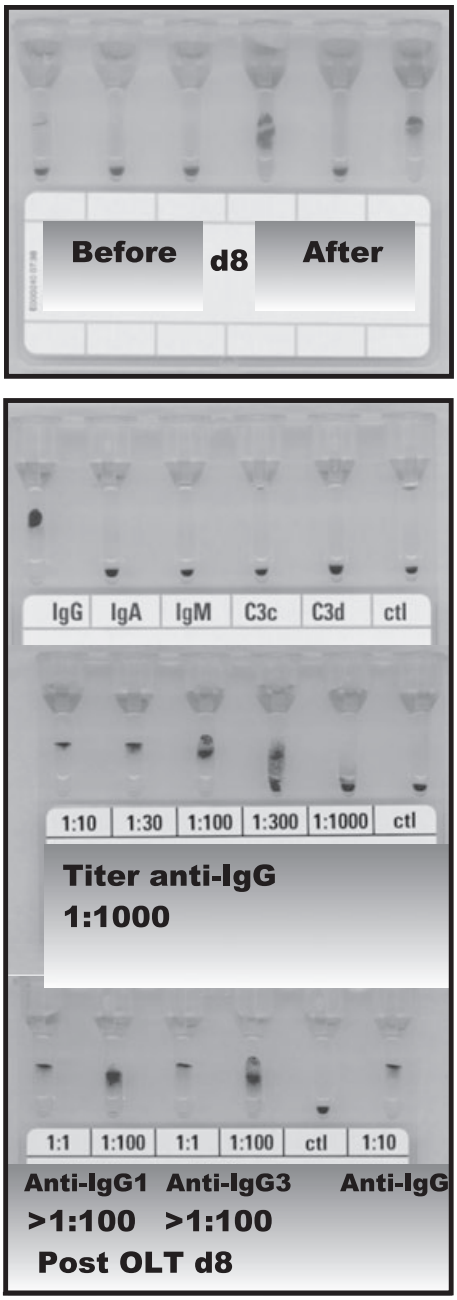

Figure 1 Direct antiglobulin test: results of immunoglobulin (Ig)G coating and complement coating, anti-D titers for IgG1 and IgG3 subclasses.

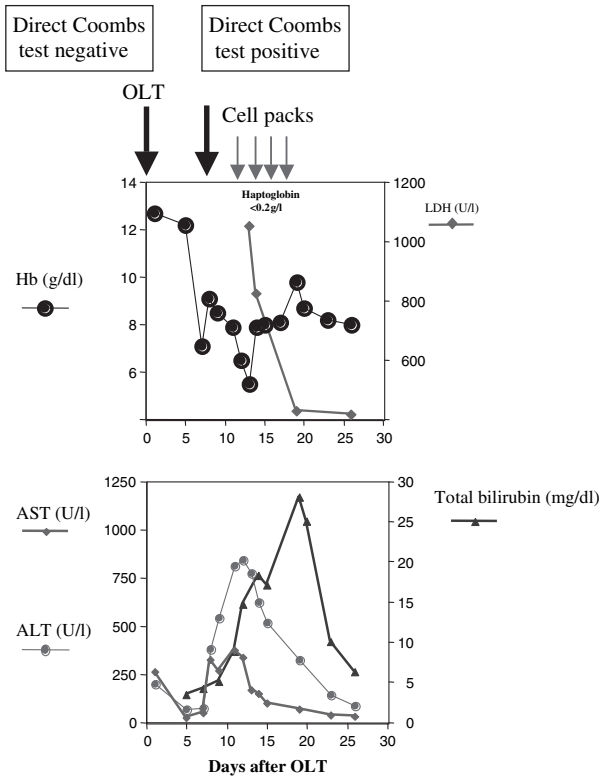

Figure 2 Postoperative values of hemoglobin, haptoglobin, lactate dehydrogenase, total bilirubin and transaminases, illustrating the hemolytic crises.

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