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LETTERS TO THE EDITOR

RENAL TRANSPLANTATION AND IMMUNOSUPPRESSIVE THERAPY DO NOT CONTRAINDICATE STEM-CELL DONATION FOR BONE-MARROW TRANSPLANTATION

A 26-year-old man, who received a cadaveric kidney graft 7 years before, was considered as a donor for bone-marrow transplantation to his human leukocyte antigen (HLA)-identical 21-year-old sister. The diagnosis in both siblings was familial nephronophthisis. The sister received a cadaveric kidney transplantation 9 years before without complications. While receiving prednisone (PDN) (0.1 mg/kg per day), cyclosporine (CsA) (3.5 mg/kg per day), and azathioprine (AZA) (1 mg/kg per day), she developed a posttransplant lymphoproliferative disease, CD3+/CD56+, and natural killer cells lymphoma (*1*). The rapid deterioration of the clinical conditions of the young woman urgently required cytostatic therapy combined with bone-marrow transplant. The brother showed HLA to be full-matched (A, B, DR), with normal bone-marrow histology and immunophenotype. A search for an alternative donor of hematopoietic stem cells available in the immediate future was unfruitful. The donor gave his consensus to be treated with granulocyte colony stimulator factor (G-CSF) to mobilise his hematopoietic stem cells for infusing into his sister. He had a good renal function (serum creatinine [sCr] 132.6 mmol/L) and was on 2.8 mg/kg per day of CsA (trough level 150 ng/mL), 0.8 mg/kg per day of AZA, and 0.05 or 0.03 mg/kg per day of PDN.

Two days before G-CSF treatment, AZA was withdrawn and PDN doses were increased to 0.2 mg/kg per day. The stem-cell mobilisation was performed by subcutaneous administration of G-CSF at 10 μg/kg per day (*2*, *3*). A transient increase in sCr on the second day (sCr 190.9 mmol/L) was detected followed by a rapid reverse to baseline value on the fifth day (sCr 134.3 mmol/L), without any change in therapy regimen. Two sessions of stem-cell apheresis were performed, after 4 and 5 days of G-CSF administration, by using a Baxter CS, 3000 plus apparatus (Baxter-Fenwal, Deerfield, IL), yielding a total of 7.5×10^6 CD34+/Kg (CD3+4.12×10^8/Kg), which is considered satisfactory for stem-cell transplantation (*3*, *4*). Seven days after stem-cell apheresis, the peripheral count of leukocytes returned to pretreatment values.

The brother's stem cells were infused into the sister, who previously received a conditioning treatment with fludarabine 30 mg/m^2 for 5 days in association with total-body irradiation (200 rads). After transplantation, she received 20 mg/kg per day of mofetil mycophenolate, 3 mg/kg per day of CsA, and 1 mg/kg per day of methyl-prednisone for graft-versus-host-disease prevention. After 2 months, the bone-marrow histology showed a complete remission of the disease, with initial appearance of donor bone-marrow cells.

One month later, the clinical conditions of the young woman suddenly worsened with ascites, fever, acute renal failure, anaemia, and thrombocytopenia. The flow cytometry analysis on peripheral blood showed 95% of the peripheral lymphocytes XY and only 5% XX, suggesting a good result of stem-cell infusion, with a slight increase in CD3+/CD56+ cells (11%). The clinical conditions rapidly worsened, and after 15 days, the percentage of blood CD3+/CD56+ was up to 50%, whereas in the bone marrow, a clonality of T–cell-receptor gamma chain was demonstrated. She died 3 months after stem-cell transplantation.

After the treatment with G-CSF and hematopoietic stem-cell donation, the donor continued with 3 mg/kg per day of CsA and 0.05 mg/kg per day of PDN. The clinical conditions of the donor have been good with stable renal function (sCr 122.9 mmol/L) over the following 12 months. Our case, the first to our knowledge, suggests that patients receiving kidney transplants are not excluded as potential hematopoietic stem-cell donors in cases of exceptional need.

GIOVANNI CONTI
MAURIZIO GAIDO
ALESSANDRO AMORE
ROSANNA COPPO
Nephrology, Dialysis and Transplantation Unit
Regina Margherita Hospital
Turin, Italy

PAOLA SARACCO
FRANCA FAGIOLI
Hemato-Oncology Department
Regina Margherita Hospital
Turin, Italy

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Address for correspondence: Dr. Conti Giovanni, Nephrology Dialysis Transplantation Unit, Regina Margherita Hospital, Piazza Polonia 94, 10126 Turin, Italy. E-mail: nefrologia@oirmsantanna.piemonte.it

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