either 1 patient at 1 month and 9 months after BMT, respectively, still exhibited the M-bcr rearrangement.

We conclude that mRNA extraction for diagnosis of the bcr-abl rearrangement in CML from dried blood spotted on filter paper is feasible. Filter papers enclosed in transparent storage sleeves may easily be filed with the patient’s records and thus allow comfortable access to DNA and RNA specimens collected at the time of diagnosis or during regular follow-up examinations for prolonged periods of time. In conclusion, simple spilling blood on filter paper may (1) facilitate the cooperation between laboratories and hospitals separated by long distances, (2) save transportation costs, and (3) contribute to standardize diagnostic procedures on a molecular level in CML and related diseases.

To the Editor:

Because of this, in Table 2 of the report, we described the results of 211 transfusions, which included donor-recipient pairs that had been used only once, donors not mismatched for HLA B12 or its splits, were not mismatched for any known strong cross reactive antigen groups, antigens against which recipient antibody is directed can favorably influence the results of one antigen mismatched platelet transfusions.

REFERENCES


Response

We agree with Dr. Murphy that careful selection of donors mismatched for cross-reactive antigens and avoidance of donors with antigens against which recipient antibody is directed can favorably influence the results of one antigen mismatched platelet transfusions.

Because of this, in Table 2 of the report, we described the results of 211 transfusions, which included donor-recipient pairs that had been used only once, donors not mismatched for HLA B12 or its splits, were not mismatched for any known strong cross reactive antigen groups, and given to patients without lymphocytotoxic antibody against the
mismatched antigen. The outcome was similar to the overall results shown in Table 1. Thus, the benefits of single antigen mismatched transfusions were apparent independent of these other strategies of donor selection and should be considered as a means of expanding the number of donors available for alloimmunized patients.

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Director of Multiple Myeloma Program

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Hemoglobin E and Pyrimidine 5'-Nucleotidase Deficiency

To the Editor:

We read with interest the report of Rees et al. Increased intraerythrocytic levels of reduced glutathione (GSH) caused by pyrimidine 5'-nucleotidase deficiency along with increased Fe³⁺ have been proposed as the possible cause of destabilization of mildly unstable hemoglobin E (HbE) in a Bangladeshi patient converting hemoglobin E disease into an unstable hemoglobinopathy-like disease.

Pyrimidine 5'-nucleotidase deficiency has been shown to interfere with hexose monophosphate shunt. This had prompted the investigators to wonder whether the combination of HbE and G6PD deficiency produce more severe clinical phenotype. Several investigators have studied the co-occurrence of HbE and HbE thalassemia with glucose 6 phosphate dehydrogenase (G6PD) deficiency without any apparent change in clinical phenotype. Furthermore it has also been pointed out earlier that the plasma hemoglobin level is increased in cases of HbE thalassemia, indicating some kind of intravascular hemolysis in these cases but not in HbE disease or in HbE traits.

G6PD deficiency normally tends to decrease the level of the reduced glutathione in the red blood cells contrary to pyrimidine 5'-nucleotidase deficiency, which tends to increase this level. Hence, if we accept the idea that the increased levels of reduced glutathione and Fe³⁺ together are causing the problem in the present case, co-occurrence of G6PD deficiency and HbE disease is unlikely to behave in that way.

To prove conclusively the correct hemolytic status, the Cr¹¹¹ survival study in the propositus would have been ideal. Furthermore, the demonstration of increased numbers of Heinz bodies in the peripheral blood smear after splenectomy would have provided morphologic evidence of unstable hemoglobin-like disease in the propositus described.

Moreover, pyrimidine 5'-nucleotidase deficiency interferes with many of the metabolic activities of red blood cells, reducing the pH of the milieu interior of red blood cells and precipitating ribonucleotides interfering with proper red blood cell function.

In some studies, G6PD deficiency has been shown to be more often associated with HbE than with the normal population (20% vs 3.4%). Hence, it appears that G6PD deficiency has no added part to play in making HbE trait clinically worse.

One wonders about the discrepancy in the globin chain biosynthetic ratio in the parents of the propositus despite the fact that both are carriers of HbE and pyrimidine 5'-nucleotidase deficiency. Coinheritance of nondeletional α thalassemia could be one explanation. Some other unexplored factors might have been co-inherited and could be responsible for producing the instability of HbE in the present case.

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Responses of Granulocyte Colony-Stimulating Factor–Mobilized Peripheral Blood Mononuclear Cells to Alloantigen Stimulation

To the Editor:

We have been much interested in the recent report by Mielcarek et al. that proposed one of the mechanisms explaining the unexpectedly low incidence of acute graft-versus-host disease (GVHD) after allogeneic peripheral blood stem cell transplantation (PBSCT) despite large numbers of T cells infused. They showed that T-cell proliferative responses to alloantigen stimulation were suppressed after granulocyte colony-stimulating factor (G-CSF) administration by a large number of monocytes in apheresis products. We also investigated alloantigen-stimulated immune responses of G-CSF–mobilized peripheral blood mononuclear cells (PBMCs) from normal donors using a mixed lymphocyte culture system. PBMCs before G-CSF administration (pre–G-CSF) and samples from leukapheresis (post–G-CSF) were obtained from six donors. Adherent...
Mismatched Platelet Transfusions to Alloimmunized Patients
Scott Murphy