PVT ($P = .24$) and was significantly higher in the group of 31 *JAK2VF*-positive PV patients (0.52; $P = .0005$), in agreement with previous studies.[2-4] However, we found no difference in the frequency of the 46/1 haplotype between *JAK2VF*-positive (0.27; n = 75) and *JAK2VF*-negative patients (0.28; n = 95) ($P = .90$). This finding is in contrast with the study by Smalberg et al, which reported a significantly higher frequency of the 46/1 haplotype in 54 patients with *JAK2VF*-positive SVT compared with controls (0.43 vs 0.27, respectively; $P < .01$).

The discrepancy between the 2 studies may depend on the populations studied. The proportion of *JAK2VF*-positive versus -negative patients is higher in our study (75/95 vs 54/145), making it possible that the high 46/1 haplotype frequency found by Smalberg et al would not be confirmed in a larger cohort with greater statistical power. Next, it recently appeared that the 46/1 haplotype frequency is correlated with the *JAK2VF* allele burden, VF-positive patients with low mutation burden displaying 46/1 haplotype frequency similar to VF-negative patients.[7-9] In our study, concordantly with the literature,[10] mutant allele burden was < 50% in 89% of patients (median: 19%) whereas in the study by Smalberg et al, JAK2VF allele burden was available in 45 patients only. In summary, in a larger cohort of *JAK2VF*-positive patients with comprehensive mutant allele burden data, we did not confirm the higher frequency of the 46/1 haplotype in SVT patients with MPNs found by Smalberg et al.

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To the editor:

West Nile virus infection transmitted by granulocyte transfusion

Eleven cases of transfusion-transmitted West Nile virus (WNV) infection and associated illness have been reported from 9 blood donors since the introduction of minipool nucleic acid testing (MP-NAT) in 2003.[1-3] This number of breakthrough infections is against a background of more than 2800 cases of WNV infection identified in blood donors in the United States through 2010. No cases of transfusion-transmitted WNV have been reported following the transition to individual-donation NAT (ID-NAT) in locations experiencing WNV activity. However, even with the use of ID-NAT,[4,5] transfusion transmission is theoretically possible from blood units having low viral loads.[1] We report the first case of WNV infection acquired via apheresis granulocyte (granulocyte) transfusion.

A 25-year-old white woman with a history of precursor B-lymphoblastic leukemia was admitted on July 9, 2010, for postchemotherapy fever and found to be neutropenic. *Escherichia coli*–positive blood cultures were identified and antibiotic therapy initiated. Neutropenia persisted and *Candida* endocarditis developed. Antifungal therapy was initiated and daily granulocyte transfusions began on August 7. Infectious disease testing results obtained on August 10 revealed that the August 9 granulocyte collection was positive for WNV. On August 25, the patient developed sudden onset of confusion with extremity weakness. A lumbar puncture revealed no malignant cells. CSF was WNV-NAT– and IgM-positive. Intravenous immunoglobulin, hydration, and nutritional support were administered and the patient discharged.

Table 1. Granulocyte donor WNV infectious disease test results

Sample type	Collection date	WNV RNA TMA S/CO*	WNV IgM S/CO†	WNV IgG S/CO	WNV RNA by PCR in copies/mL‡
Index donation	8/9/10	29.53	NT	NT	700
Index retention	8/9/10	33.79	< 0.67	< 1.30	3600
Follow-up	10/18/10	0.02	4.73	3.74	< 5

TMA indicates transcription-mediated amplification; S/CO, signal-to-cutoff; PCR, polymerase chain reaction; and NT, not tested.

*TMA was done using the Gen-Probe/Novartis WNV assay on the TIGRIS automated platform. S/CO ratios of 1.00 or greater indicate a reactive test result.

†Antibody testing (IgM and IgG) was performed by Focus Laboratories. S/CO ratios of 0.67 or greater for IgM and 1.30 or greater for IgG indicate a positive test result.

‡PCR testing was performed by National Genetics Institute using a quantitative assay with a 95% lower limit of detection of 5 copies per milliliter.

The patient was readmitted and died on November 6 from complications of sepsis secondary to febrile neutropenia.

The multi-time donor is a 45-year-old man who received dexamethasone before donation. He was asymptomatic at donation, but subsequently became ill on August 22 with fever, chills, severe fatigue, headache, joint and bone pain, tremor, rash, and difficulty thinking. He was hospitalized on August 26 for treatment of WNV meningitis. His symptoms were consistent with those reported in WNV-infected donors.[6] He cleared his WNV infection and seroconverted at follow-up (Table 1).

Granulocytes must be transfused as soon as possible after collection and thus transfused before completion of infectious-disease testing.[7] The collecting facility performed WNV ID-NAT from August 3 through October 11 because of WNV activity in the area, consistent with recommendations in the United States.[3] The WNV infecting unit had a high viral load sufficient for detection by MP-NAT. Even though ID-NAT results are available sooner than those from MP-NAT, they were not available before the need for transfusion. The time required for generation of test results by any licensed screening test, including WNV NAT, prevents their availability before the need for transfusion of highly time-sensitive components. This case illustrates the need to evaluate the benefits of granulocyte transfusion for critically ill, neutropenic patients in the face of the rare possibility of WNV transmission during epidemic periods. Health care providers should be aware that granulocytes may transmit WNV, and thus health care providers should consider WNV as a potential cause of neurologic complications after granulocyte transfusion.

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To the editor:

Permissive, nonpermissive HLA-DPB1 epitope disparities and the specificity of T cells infiltrating the skin during acute graft-versus-host disease

Human leukocyte antigen (HLA)–DPB1 functions as a classic transplantation antigen.[1] In the context of hematopoietic stem cell transplantation (HSCT), when donor T cells recognize host HLA-DP, they can induce a graft-versus-host disease (GVHD) and/or a graft-versus-leukemia (GVL) effect, whereas in the opposite direction, host T cells recognizing the donor can induce rejection (HVG). Accordingly, any possibility to anticipate the nature and strength of an anti-DP T-cell response is crucial in this context.

Based on the HLA-DP recognition pattern of several HLA-DPB1*0901-specific T-cell clones, Crocchiolo et al classified HLA-DPB1 alleles according to their predicted "immunogenicity,"[2] and using an algorithm deduced from this classification (Figure 1), they showed that the presence of "nonpermissive" HLA-DPB1 mismatches correlated with a significantly increased hazard of acute grade 2-IV GVHD. Unexpectedly, the authors reported that the increased risk of aGVHD was detectable independently of the predicted direction (GVH/GVL or HVG) of the T-cell response. To reconcile the statistical observation with the immunologic hypothesis (the increased risk of GVH when the algorithm predicted the recognition of donor HLA-DP by host T cells), these authors considered the possibility of an indirect pathway for GVH because of cytokine release by host T cells recognizing "immunogenic" HLA-DPB1 on donor antigen-presenting cells (APC).

In 4 successive studies,[3-6] we have in the past assessed the specificities of T-cell clones infiltrating skin biopsies during aGVHD (Table 1). In each situation, T-cell clones specific for host HLA-DP were isolated from the skin at the onset of aGVHD. These studies demonstrated that no mismatch could theoretically be