which will significantly reduce both materials and costs for laboratory tests.

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REFERENCES


Prognosis Value of Residual Disease Monitoring by Polymerase Chain Reaction in Patients With CBFβ/MYH11-Positive Acute Myeloblastic Leukemia

To the Editor:

Pericentric inversion of chromosome 16, translocation (16;16), and del(16q), resulting in a chimeric fusion of the CBFβ and MYH11 genes, are typically seen in the M4eo French-American-British (FAB) subset of acute myelogenous leukemias (AML). Karyotypic detection of these abnormalities can be difficult, particularly in the minimal residual disease (MRD) setting. To date, only 15 patients, in four publications,1-4 have been studied for MRD by reverse transcription-polymerase chain reaction (RT-PCR) detection of CBFβ/MYH11 transcripts. These studies showed the feasibility of the technique in MRD follow-up.

We have studied MRD by RT-PCR in peripheral blood, bone marrow, or cytapheresis samples from 10 patients (Fig 1). Nine were positive at diagnosis for the common A-type transcript and one (UPN1) for a variant of the E-type transcript. Patients UPN 198, 92, 91, and 319 were studied at Necker-Enfants Malades (Paris, France) using C1/M1/C2 primers and seminested PCR as previously described,1 whereas patients UPN 1, 5, 7, 9, 11, and 12 were tested at Institut Paoli-Calmettes (Marseille, France) with C4 (5'AGG-CCTTGTGAGGGGCTGGG-3') and M0 (5'-AGCGGGCCCTGG-AGACCCAG-3') primers in a single-round PCR followed by hybridization with an internal P3b CBFβ probe (5'-AGACAGGTC-TCTCATCGGGAGG-3') and autoradiography.6 The two laboratories achieved the same level of sensitivity (10-4 cellular equivalent ± 1 log) as controlled by dilution experiments of a positive control RNA (ME-1 cell line or patient diagnostic material) performed during each testing.

All patients achieved clinical and cytological complete remission (CR) after induction therapy (cytarabine and anthracycline-con-
GB Virus C Infection and Liver Transplantation: Increased Risk of Transfusion-Transmitted Infection

To the Editor:

GB virus C (GBV-C) and hepatitis G virus (HGV) are recently discovered RNA viruses that infect humans. Because GBV-C and HGV are most likely isolated strains of the same virus, the GBV-C nomenclature will be used throughout this letter. GBV-C seems to cause hepatitis unrelated to infection with hepatitis A, B, C, or D viruses. It has been reported that GBV-C infection may be linked to fulminant hepatitis. Therefore, in a retrospective study, we investigated 109 patients (41 women and 68 men; median age, 49 years) who underwent liver transplantation between 1992 and 1996. Seventeen (15.6%) of them had a history of fulminant hepatitis, 11 of unknown etiology, 5 of hepatitis B virus (HBV) infection, and 1 of hepatitis A virus (HAV) infection. The other 92 (84.4%) patients had liver cirrhosis due to autoimmune hepatitis (27 [24.8%]), chronic hepatitis C virus (HCV) infection (27 [24.8%]), chronic HBV infection (11 [10.1%]), HBV and hepatitis D virus (HDV) infection (4 [3.7%]), alcohol abuse (15 [13.8%]), unknown etiology (6 [5.5%]), or other (2 [1.8%]). GBV-C infection was investigated by reverse transcription-polymerase chain reaction (RT-PCR) using primers of the helicase-like region. At the time of liver transplantation, 7 (6.4%) of the 109 patients had detectable GBV-C viremia. Of these 7 patients, 4 patients had liver cirrhosis due to alcohol abuse, 2 patients had autoimmune hepatitis, and 1 patient had a cryptogenic cirrhosis. None of the 17 patients with fulminant hepatitis had detectable GBV-C viremia before or at the time of transplantation. Postoperatively, the 7 GBV-C–infected patients and additionally 42 patients (38.5%) had detectable viremia in sera drawn between days 6 and 14 after transplantation. These 49 patients showed a persistent GBV-C vire-
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