Bone Marrow Transplantation, (1998) 21, 533–535
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Fulminant hepatitis in aplastic anemia: hepatitis G or ALG?

After reading the article by Killick *et al*[1] in the February 1997 issue of *Bone Marrow Transplantation* we wish to report a case of aplastic anemia and fulminant hepatic failure. An 18-year-old girl with a diagnosis of severe aplastic anemia was admitted to the Department of Hematology, Ibni Sina Hospital, Ankara University. She received cyclosporine (10 mg/kg/day 6 months), antilymphocyte globulin (ALG) (Pasteur Merieux, Lyon, France) (20 mg/kg/day 8 days), prednisolone (2 mg/kg/day 8 days) and GM-CSF (5 μg/kg/day) according to the protocol arm to which she was assigned. Two days after the last dose of ALG she developed bone pain and abdominal pain, and her transaminases and bilirubin levels which were normal before therapy started to rise dramatically. ALT levels rose to 2837 U/l, AST levels increased to 4796 U/l and bilirubin levels rose to 23.46 mg/dl. Urinary output diminished followed by a rise in creatinine and blood urea nitrogen levels. The maximum level for creatinine was 7.6 mg/dl. She died 3 days after this. Hepatic biopsy revealed massive hepatic necrosis. G virus RNA was detected using RT-PCR as described before.[2] Hepatitis B antibody was positive and serological markers for HCV and HAV were negative at that time. She was hepatitis B antibody positive before treatment. It is premature to say that this hepatic failure resulted from G hepatitis because a case of severe hepatic failure that occurred after the administration of ALG + cyclosporine has been reported.[3] Also, hepatitis G might or might not be the cause of aplastic anemia in this patient as described before.[4,5] Nevertheless, this is a documented case of severe hepatitis in the presence of G virus. Recent articles indicate that G virus is not a cause of hepatitis, and does not alter the clinical course of hepatitis C.[6,7] We still feel that one must be cautious in interpreting G virus until it is clearly documented that it is innocent.

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Is there a relationship between G-CSF response to conditioning regimen and engraftment after bone marrow transplantation?

Busch *et al*[1] recently published an article in the Journal. In a selective group of patients with CML, they showed that patients with later engraftment presented higher G-CSF levels than the patients with earlier engraftment. While both groups started with identically low G-CSF levels 1 day prior to bone marrow transplantation, levels differed at day +1 after bone marrow transplantation and this difference continued until day +10. It has been shown that peripheral stem cell infusion has a cytokine triggering effect.[2] We have previously reported very high levels of G-CSF following peripheral blood stem cell transplantation used to rescue engraftment failure (Figure 1).[3] In the study by Busch *et al*, the difference in mean G-CSF levels at day +1 may be caused by differing amounts of the infused cell dose. In our study, serum G-CSF which was measured by the same enzyme immunoassay principle (Amgen Diagnostics, Thousands Oaks, CA, USA) reached its highest levels at day 0 just after the end of conditioning. In contrast to the finding of Busch *et al*, the G-CSF levels at day 0 were lower in three of our patients with engraftment failure compared to those with successful engraftment (Figure 1). This finding is in concordance with Cairo *et al*'s results.[4] Our