AUTOIMMUNE-LIKE THROMBOCYTOPENIA AFTER BONE MARROW TRANSPLANTATION

To the Editor:

Recently, Anasetti et al[1] have published an analysis of 20 patients with thrombocytopenia (TCP) 60 or more days after allogeneic bone marrow transplantation (BMT). Patients were selected by their attending physician before analysis. The investigators concluded that persistent TCP after BMT is most often secondary to increased platelet destruction mediated by multiple mechanisms, and that platelet autoantibodies are found in patients with acute or chronic graft-versus-host disease (GVHD).

Between April 1985 and November 1986, we prospectively analyzed the evolution of platelet-associated immunoglobulin G (PaIgG) in 48 patients after BMT: five were excluded because of the lack of PaIgG follow-up and four others because of death within 2 months posttransplant. Among the 39 evaluable patients, 14 received autologous and 25 allogeneic BMT (genotypically identical for 18, human leukocyte antigen nonidentical for 6, and syngeneic for 1). Conditioning regimen included TBI and chemotherapy in 29 cases. In 10 other cases, patients were treated with high-dose polychemotherapy. Indications for transplant were: acute leukemia (18), chronic granulocytic leukemia (13), Hodgkin's disease (5), non-Hodgkin's lymphomas (2), and one myelofibrosis. Bone marrow was purged with monoclonal antibodies and complement in 22 cases, with mafosfamide in 3, and untreated in 14. Platelet surface-bound IgG assay was evaluated with a direct radioactive Coomb's test (I^{125}-labeled goat antihuman IgG).

TCP were separated in two groups: prolonged TCP, when the platelet count did not recover over 100×10^9/L beyond 2 months after BMT; and late TCP, when after an initial stable recovery of a normal count, platelets dropped under 100×10^9/L after D + 60 postgraft. Among 39 evaluable patients, 17 (43%) were thrombocytopenic after D + 60: 9 had prolonged TCP and 8 had late TCP (TCP occurred after 8 of 18 allogeneic, 5 of 14 autologous, and 4 of 6 mismatched BMT). All were associated with a PaIgG positive test, except two cases of late TCP. Twenty-six patients developed at least one viral infection, 16 developed TCP, and only one patient developed TCP without viral infection ($P = .005$, Fisher's exact test). Eleven of the 26 viral infections were due to cytomegalovirus, and only 4 of 25 patients developed GVHD $\geq$ II (at that time we used T-cell depletion systematically).

The physiopathology of TCP after BMT is polyfactorial: the role of GVHD has been emphasized by Bierling et al,[2] First et al,[3] and Anasetti et al[1]. We and Verdonck et al[4] found a striking correlation between TCP and viral infections. Overall, platelet reconstitution remains an important problem after BMT, and the explanation of the phenomenon remains unclear.

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