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Use of Granulocyte Colony-Stimulating Factor (G-CSF) in Leukopenic Renal Transplant Recipients

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Abstract

Leukopenia secondary to drug effects or infection often occurs following renal transplantation and can lead to early withdrawal of immunosuppression. G-CSF causes a rise in granulocytes when given to patients with normal bone marrow function or to patients with bone marrow supression caused by cytotoxic drugs or AIDS. A potential use of G-CSF is in the treatment of leukopenic organ graft recipients. A major concern is that such treatment might exacerbate or precipitate a rejection process. We report the use of G-CSF in 3 leukopenic renal allograft recipients.

Two patients received cadaveric renal transplants (CRT) and one patient had a living-related donor renal (LRD) transplant. All the recipients and donors of the kidneys were positive for anti-CMV antibody. Immunosuppression consisted of cyclosporine (CsA), azathioprine (Az) and prednisone. Nifedipine or diltiazem were given as part of the protocol. Patients received prophylactic acyclovir, nystatin and sulfamethoxazole/ trimethoprim (S/T). Both CRT had delayed graft function. The LRD transplant began to function immediately. All 3 patients became progressively leukopenic despite Az and S/T being withheld. The WBC fell to < 0.9 x 10(3)/mm(3) and 2 patients were febrile. One patient had pseudomonas sepsis. Bone marrow biopsies were hypocellular in all patients. G-CSF was administered at 5 ug/kg/day. It resulted in a rise in the WBC count to >3,000/mm(3) by day 7 in all patients. There were no changes in graft function or other evidence of rejection.

Conclusion

G-CSF increases WBC counts without eliciting graft rejection in severely leukopenic patients undergoing solid-organ transplantation.