Post-transplant infections

Sir,

We read with interest the article "Post-transplant infections: An ounce of prevention".[1] We have encountered a few patients of new-onset diabetes mellitus after transplantation (NODAT) on tacrolimus-based immunosuppression with asymptomatic hepatitis C virus (HCV)/Cytomegalovirus (CMV) infection detected on polymerase-chain-reaction assays. It is unclear whether HCVRNA and CMVDNA should be done in all transplant recipients at initial diagnosis of NODAT to detect asymptomatic HCV/CMV infection with or without other known risk factors especially when NODAT incidence is usually high?

A meta-analysis of ten studies of 2502 patients found that anti-HCV positive patients were nearly four times more likely to have NODAT compared with uninfected individuals.[2] The relationship between HCV infection and NODAT may be heightened with tacrolimus versus cyclosporine-based immunosuppression CMV infection has also been reported to increase the risk of NODAT.[3] In one study, an asymptomatic CMV infection was associated with a lower median insulin release and a fourfold increased risk of NODAT.[4]

Also unclear is how CMV, BK polyomavirus, herpes simplex virus (HSV) infection and others should be monitored in patients who had undergone desensitization protocol with multidrug regimen?Desensitized patients receive more immunosuppression treatment, including rituximab, plasmapheresis, and anti-thymocyte globulin, compared with nonsensitized patients, which might increase the risk of infection (especially CMV and BK polyoma virus).

For all highly sensitized patients who received a kidney transplant, polymerase-chain-reaction assays for CMV, and polyomavirus BK were performed on whole-blood specimens monthly for the first 3–6 months after transplantation and then every 3 months until the end of the first post-transplant year, or whenever there is an unexplained rise in serum creatinine, and after treatment for acute rejection, with appropriate clinical features. The methods used for monitoring viral replication have been described previously.[5-7] A Cedars Sinai group monitored their desensitized patients by monthly CMV, Epstein-Barr virus, parvovirus B-19, and BK virus (BKV) testing.[7] Antiviral prophylaxis should be considered in patients treated with bortezomibas herpes zoster virus (HZV) infections are common, especially in cancer patients. Vaccination against HZV before bortezomib use should be contemplated in HZV-naïve patients. Weekly monitoring of CMV antigenaemia should be performed in seropositive patients at risk of reactivation or disease.[7-10]

Whether the pre-emptive approach would be optimum in low risk (D-/R-) recipients who had received blood transfusions pre-transplant? The risk of CMV infection is rare in solid organ transplants who are at risk for severe morbidity from CMV infection and who receive CMV reduced risk products. Two methods to supply CMV reduced risk products, which appear to have equal efficacy are: CMV seronegative cellular components (red cells, platelets) or leukoreduced components. Such facility may not be available in all hospitals.

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