Abstract

Cellular rejection after renal transplantation, in general, occurs as a result of an interaction between immunologic processes that maintain graft tolerance versus allograft rejection. A potential mechanism that triggers such processes might be through the activation of the innate immune response initiated during organ procurement and ischemia/reperfusion injury, contributing to delayed graft function or graft dysfunction. Our goal was to test the impact of molecular markers that have key roles in innate immunity such as cytokines, Toll-like receptors (TLRs), and allograft inflammatory factor-1 (AIF- 1) at early times after transplantation. Blood samples from a total of 90 patients who received kidney transplants were included in this study. Three samples from each patient at different time intervals (pretransplantation, day 3. and day 6 after transplantation) were tested using a quantitative reverse transcriptase polymerase chain reaction. The mRNA transcripts were tested in association with glomerular filtration rates (GFR) as a measure of allograft function. Surgical samples obtained from transplant nephrectomy were used in a tissue array for immunohistochemistry testing. In peripheral blood mononuclear cells, the mean ± standard error of mean (SEM) for interleukin 18 (IL-18), and IL-10 mRNA expression were increased and interferon-y was decreased in association with high GFR post-transplantation as compared with the pretransplantation expression levels. The mean ± SEM for expression level of AIF-1 was increased 1.5fold and for TLR-2 and TLR-4 were increased 1.2 to 1.4-fold in samples obtained on day 6 posttransplantation in association with low GFR (P < .05). In neutrophils, the mean ± SEM levels of TLR-2 mRNA was increased 2-fold on day 6 in association with high GFR (P < .005), but was reduced 2.8-fold in association with low GFR (P < .002). In conclusion, the mRNA profiles of biomarkers presented here appeared to be informative for prediction of allograft status and outcome.