LONG-TERM MANAGEMENT OF LUNG TRANSPLANT RECIPIENTS

While survival after lung transplantation continues to improve by era, the survival rates in this group are lower than in other solid-organ cohorts. Approximately 50% of lung transplant recipients will experience at least one episode of acute rejection in the first posttransplant year, and by 5 years posttransplant, approximately half will have developed chronic rejection. As a result, posttransplant immune suppression regimens may be more aggressive than in other solid-organ recipients, as described above. The immunosuppressive regimen must be balanced against the potential toxicities that accrue with these medications over time.

Acute cellular rejection in lung transplant recipients is most common in the first posttransplant year, with a decreased but not absent frequency thereafter. Infections can stimulate cellular rejection, most clearly demonstrated in the setting of CMV infection, but also noted after other infections. Most programs incorporate a schedule of routine surveillance bronchoscopy to assess for acute cellular rejection posttransplant. Acute cellular rejection manifests as a lymphocytic infiltrate involving the distal small vessels and capillaries and/or a lymphocytic bronchiolitis involving the distal airways of the lung. Acute cellular rejection, a risk factor for the development of chronic lung allograft dysfunction (CLAD), is treated with augmented immune suppression. Antibody-mediated rejection in its classic form is a neutrophilic vasculitis associated with the small vessels and capillaries of the lung, with associated deposition of by-products of the complement cascade, in the setting of allograft dysfunction and circulating donor-specific HLA antibodies in the blood. The manifestations of antibody-mediated rejection in the lung allograft are less specific than in other organs. Further research is ongoing into the diagnostic and treatment considerations of this entity in lung transplantation.

CLAD is an overarching description of the syndrome of long-term allograft rejection. The classic manifestation of CLAD is obliterative bronchiolitis, the development of fibrinous material within the distal airways that leads to small-airways obstruction. As transbronchial biopsies are insensitive for diagnosing obliterative bronchiolitis, a