Acute antibody-mediated rejection

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Acute antibody-mediated rejection (AMR) occurs in <5% of renal transplants but is more serious and more di ffi cult to treat than cell-mediated rejection. It is caused by HLA DSA, which are produced by a sensitising episode from a previous transplant, a blood transfusion or pregnancy . Binding of DSA to mismatched HLA antigens on the surface of allograft endothelial cells leads to activation of the complement system and tissue injury . In the kidney , transplant biopsy will show inflammation of the vessels (vasculitis) and deposition of the complement component C4d in the peritubular capillaries (Figure 88.9). Treatment is by plasma exchange to remove circulating antibodies. Rituximab, an anti-CD20 monoclonal antibody , can also be used to destroy B cells and prevent further production of DSA. The pathophysiology is not well understood but the long-lived indirect antigen presentation pathway is likely to be important. CD4 T-cell activation also promotes donor-specific alloanti - body production, and this causes ongoing allograft damage.

Figure 88.9 Acute antibody-mediated renal allograft rejection. There is widespread staining for the complement component C4d within the peritubular capillaries (arrows), which indicates alloantibody binding to the graft vasculature. Depleting antibodies Calcineurin blockers ATG Ciclosporin Tacrolimus Alemtuzumab Resting Early T cell activation Costimulatory blockade CTLA-4Ig Figure 88.10 Site of action of immunosuppressive agents on T cell. ATG, antithymocyte globulin; CTLA-4Ig, cytotoxic T-lymphocyte-associated protein 4 immunoglobulin; MPA, mycophenolic acid derivatives; mTOR, mammalian target of rapamycin.

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