transplants are at significant and prolonged risk for opportunistic infections due to the high rate of GVHD associated with these allografts, resulting in prolonged T-cell–mediated immunosuppression and delayed immune reconstitution. Umbilical cord blood transplants are associated with prolonged neutropenia as a result of the small volume of cells available for transplantation, placing patients at risk for infection. The risk of GVHD, however, is lower with the use of umbilical cord cells.

Immediately after HSCT, stem cell recipients are neutropenic. During this pre-engraftment period, pathogens characteristic of prolonged neutropenia can cause infection. Prophylaxis with voriconazole or posaconazole, levofloxacin or ciprofloxacin, acyclovir, and penicillin is common to prevent infections during this vulnerable period.

After engraftment, the risk of GVHD is high for many allogeneic transplant recipients, resulting in administration of corticosteroids, methotrexate, cyclosporine, tacrolimus, sirolimus, or mycophenolate for prophylaxis. After GVHD develops, high-dose corticosteroids usually are given. In refractory cases, additional T-cell–active immunosuppressive agents, including antithymocyte globulin, tacrolimus, sirolimus, cyclophosphamide, and alemtuzumab, may be required. Patients on these agents are at risk for infections from CMV, *Nocardia*, HHV-6, *P. jirovecii*, adenovirus, invasive molds, and other opportunists. In some cases, patients require chronic immunosuppressive therapy to control GVHD, putting them at risk for infection indefinitely and requiring long-term antibiotic prophylaxis. If GVHD is successfully prevented, immunosuppressive therapy is discontinued after 6 to 12 months.

In uncomplicated HSCT cases without GVHD, functional humoral immunity takes 1 to 2 years to return to normal, and patients are at risk for encapsulated organisms despite being successfully engrafted. Standard immunizations are re-administered 1 to 2 years after transplantation because pretransplantation antibodies are often lost.

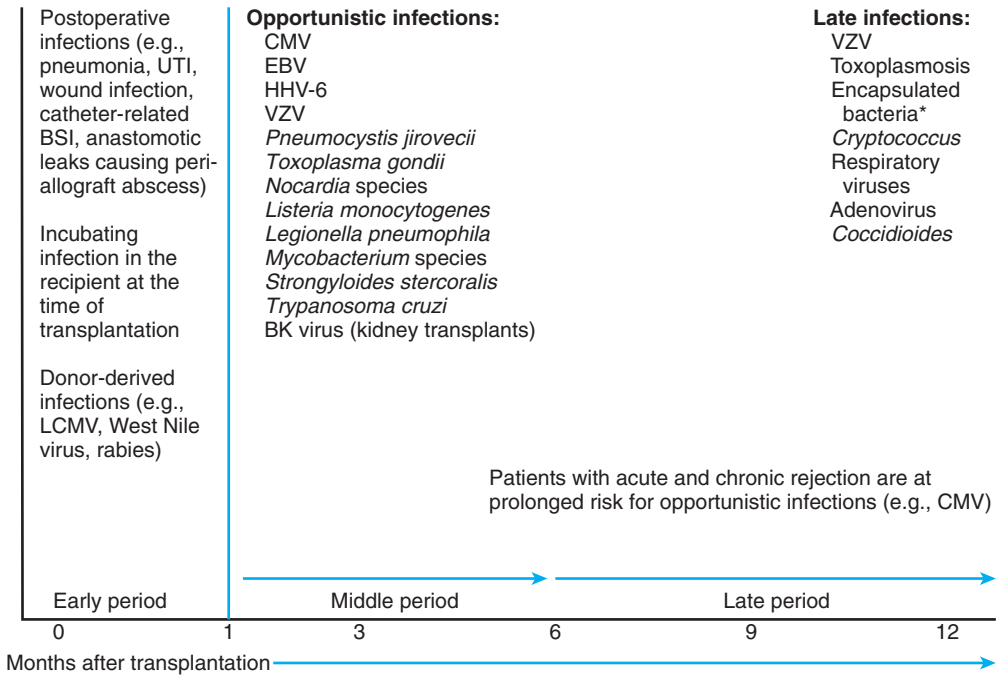
Human Immunodeficiency Virus Infection

Chapter 101 provides a detailed discussion of treatment and prophylaxis of infections occurring in HIV-infected patients.

Solid Organ Transplantation

Organ transplant recipients are at lifelong risk of infection, although the pathogens involved change over time (Fig. 102-2). In the first month after transplantation, patients develop infections related to the surgical procedures and hospitalization. Urinary tract infection in the renal transplant recipient, pneumonia in the lung transplant recipient, and sternal wound infection in the heart transplant recipient tend to manifest in the first 4 weeks postoperatively. Common nosocomial infections such as catheter-related bloodstream infection and *Clostridium difficile* may also occur. Anastomotic leaks in pancreas and liver transplant recipients may cause polymicrobial intra-abdominal abscesses.

Patients with incubating infection at the time of transplantation and induction immunosuppression can develop disseminated infection with viral or bacterial pathogens, which is associated with a high mortality rate. Careful evaluation of the recipient at the time of hospital admission for transplantation is



*Especially in patients with hypogammaglobulinemia complicating immune suppression

FIGURE 102-2 Timing of infections after solid organ transplantation. BSI, Bloodstream infection; CMV, cytomegalovirus; COPD, chronic obstructive pulmonary disease; EBV Epstein-Barr virus; HCV, hepatitis C virus; HHV-6, human herpesvirus 6; HIV, human immunodeficiency virus; LCMV, lymphocytic choriomeningitis virus; UTI, urinary tract infection; VZV, varicella-zoster virus.