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Commentary

Effects of Viral Infections during Renal Transplantation

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DESCRIPTION

Liver, kidney, heart, and lung transplantation have become standard treatments for certain terminally ill patients. However, complications such as infection and rejection of the allograft due to immunosuppressant's do occur. Treatment remains a major cause of morbidity and mortality after organ transplantation. Viral infections are the result of community exposure (influenza, adenovirus) and some are commonly transmitted in allografts (Cytomegalovirus, Epstein-Solid organ transplantation is a treatment option for so many human diseases nowadays Barr virus). The result of more distant exposures that are reactivated as part of immunosuppression (chickenpox). Viral and non-viral infections are also common. The effects of CytoMegaloVirus (CMV) and human herpes virus 6 or cytomegalovirus and pneumocystis virus infection appear to be direct, indirect febrile and neutropenia syndromes and invasive diseases such as pneumonia, enteritis, meningitis or encephalitis considered a direct effect. Cytokines, chemokine's and growth factors are reduced in response to viral infections in the body, deepening immunosuppression and increasing the risk of other opportunistic infections. In addition, viral infection can alter and induce expression of surface antigens (eg, histocompatibility antigens) which causes graft rejection and/or deregulated cell proliferation later contributes to carcinogenesis. Risk factors for CMV disease and CMV infection, Reactivation of HHV 6 and HHV 7, Co-infection with polyomavirus and CMV has been reported in renal transplant recipient. CMV is the most common viral infection in solid organ transplant recipients. It usually occurs in the first few months after transplantation and is associated with clinical infections (eg: fever, pneumonia, gastrointestinal ulcer, hepatitis, retinitis) and acute or chronic graft failure. UTI affects about 1 in 3 transplant recipients, especially when reflux nephropathy or diabetes is the main causes of renal failure. Urinary tract infections usually cause pain when urinating . Infection is a major cause of morbidity and mortality in renal transplant recipients. Careful pre-transplant screening, vaccination, and post-transplant prophylactic antibiotics can reduce the risk of post-transplant infections. The chronic immunosuppression required to maintain allograft function after organ transplantation predisposes transplant patients to a variety of viral infections. These can occur at any stage of the post-implantation process. However, some infections, such as CytoMegaloVirus (CMV), Epstein-Barr Virus (EBV), or BK Virus (BKV), tend to occur within months after transplantation. CMV infection can be readily prevented by prophylactic therapy, whereas EVB or BKV infection can be prevented by reducing immunosuppression. Some viral infections can lead to post-transplant lymphoproliferative disease (EBV), Kaposi's sarcoma (human herpes simplex virus type 8), or skin and/or cervical cancer (papillomavirus). Other viral infections, such as influenza virus, are mostly acquired by environmental infection. All of these can be easily recognized in the early stages and treated effectively. CMV is the most common viral infection in solid organ transplant recipients. It usually occurs in the first few months after transplantation and is associated with clinical infections (eg, fever, pneumonia, gastrointestinal ulcer, hepatitis, retinitis) and acute or chronic graft failure. Causes of CMV infection in transplant recipients include latent reactivation, donor-derived virus, and virus residing in donor leukocytes. Approximately 20% to 60% of all transplant recipients develop symptomatic CMV infection. Treatment of established CMV disease requires a multifactorial approach, including immunosuppressive agents, antiviral agents and possibly reduced adjuvant therapy. Intravenous ganciclovir is considered the mainstay of treatment. Valganciclovir is an oral prodrug of ganciclovir with 10-fold greater bioavailability than oral ganciclovir. Recent studies suggest that valganciclovir may replace both oral and intravenous ganciclovir in many settings. Both HSV and VZV are alpha herpesviruses with a double-stranded DNA core. Infection in renal transplant patients is usually caused by latent viral reactivation. HSV infection usually presents as oral or genital lesions, but in some cases can cause esophagitis, hepatitis, encephalitis, or pneumonia. EBV is a ubiquitous herpes virus and maintains its latent period. Under severe immunosuppression, severe impairment of cytotoxic T-cell responses leads to unsustainable latency, leading to uncontrolled EBV-driven B-cell proliferation and Post-Transplant Lymphoproliferative Disease (PTLD). This is why this virus is clinically important. Polyomaviruses are DNA viruses, three of which are known to infect humans. BK virus, JC virus, simian virus, SV40. Initial infection is usually asymptomatic and likely

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to be respiratory or bloodborne. Known risk factors in transplant recipients are multiple episodes of rejection and seropositive donors and/or recipients. Reports of PolyomaVirus Nephropathy (PVAN) are also increasing with the increased use of potent immunosuppressants such as tacrolimus, mycophenolate mofetil, and sirolimus. HHV-6 and HHV-7 are increasingly recognized as pathogens in transplant recipients. HHV-6 and his HHV-7 are homologous to CMV. HHV-6 is associated with fever, rash, encephalitis, hepatitis, myelo suppression, and interstitial lung disease. Careful detection and management of opportunistic viral infection enable better graft survival and quality of life in renal transplant patients.