

Introduction

Among solid-organ transplants, kidney transplantation is associated with the lowest rates of infections, in part because of the elective or semi elective nature of kidney transplantation. In contrast, liver, and heart, or lung allograft recipients often have poor clinical and nutritional status before transplant that contributes to increased infection risk. Despite that, infection remains a significant cause of morbidity and mortality in renal transplant recipients (*Gabreil MD 2004*)

Cytomegalovirus (CMV) is a ubiquitous virus; the seropositivity in the population ranges worldwide from 40% to >90%. Because of its opportunistic behavior under immunosuppression, active CMV infections generally have a large impact on the clinical course of organ transplant recipients. The negative influence of CMV on the results of transplantation is beyond any doubt. Depending on donor-recipient (D/R) match, diagnostic techniques, and mode and magnitude of immunosuppression, the onset of the majority of active CMV infections after solid-organ transplantation ranges from 2 weeks to several months after transplantation. Active CMV infection occurs in 30%–75% of the transplant recipients at risk, with a mortality rate of \square 5%, even at present. Serious and often fatal secondary infections (especially fungal) are of great concern. At greatest risk are patients with a D_/R_match, patients undergoing lympholytic induction or rejection therapy, and patients with previous or concomitant (super-)infections. Whereas the incidence of CMV disease is 8%–35% in kidney, heart, and liver transplant recipients, its frequency is considerably higher in

pancreas or kidney-pancreas (50%) and lung or heart-lung transplant recipients (50%–80%).

A number of effects and sequelae have been linked to (active) CMV infection, such as CMV-related symptoms and organ dysfunction; contribution to the so-called net state of immunosuppression after organ transplantation; the clinical observation of a mutual influence between CMV infection and acute transplant rejection; a possible role of CMV in the development of chronic transplant dysfunction, such as accelerated coronary atherosclerosis after heart transplantation, vanishing bile duct syndrome after liver transplantation, and bronchiolitis obliterans syndrome after lung transplantation; and a role as an independent risk factor in the development of posttransplant lymphoproliferative disease.

Despite the introduction of newer antiviral medications for prophylaxis cytomegalovirus (CMV) infection is still the most common opportunistic infection following solid organ transplantation. The incidence of symptomatic CMV infection varies between 20 and 60% after transplantation and depends on the type of immunosuppressive treatment, the antiviral prophylaxis, the CMV status of the recipient and donor, and the methods used to detect CMV infection (*Brennan, 2001*) & (*Becker et al 2002*).

It is more common in patients treated with antilymphocyte antibodies, inadequate antiviral prophylaxis, or CMV seronegative recipients that receive transplants from CMV seropositive donors (*Brennan, 2001*) & (*Flechner, et al 1998*).

Cytomegalovirus disease significantly increases morbidity and mortality in transplant recipients, not only by severe primary infection but also by indirect effects on the allograft. Cytomegalovirus infection can initiate endothelial activation and vascular injury that may trigger acute rejection, chronic rejection, atherosclerosis, transplant glomerulopathy, or thrombotic microangiopathy (**Brennan , 2001**)

Acyclovir was initially used for CMV prophylaxis, but clinical trials demonstrated that high-dose acyclovir is not effective in preventing CMV disease in renal transplant recipients (**Kletzmayr et al 1996**)& (**Flechner , et al 1998**).

Valacyclovir, a prodrug of acyclovir, has a higher bioavailability and increased efficacy in CMV prophylaxis (**Lowance ,et al 1999**).

Ganciclovir was the first drug proven to be effective in life-threatening CMV infection (**Crumpacker ,et al 1996**).

Oral ganciclovir prophylaxis has decreased the incidence and severity of CMV infection in kidney and/or pancreas transplant recipients (**Brennan DC, et al 1997**)&(**Kaufman DB, et al 2001**).