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Conrad Seoul, Korea
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Session Title : Updated prophylaxis for Infection

P. jirovecii prophylaxis

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Pneumocystis jirovecii, previously known as *Pneumocystis carinii,* is an opportunistic pathogen that causes severe pulmonary infection in immunocompromised hosts. In the transplantation setting, symptoms of *P. jirovecii* pneumonia (PJP) often develop over a few days up to 1-2 weeks, usually dominated by hypoxemia out of proportion to pain radiographic imaging. Corticosteroids, calcineurin inhibitors, and sirolimus may mask the signs and symptoms of PJP. In addition to more rapid onset, the resulting pneumonia and lung involvement are often more severe, and lower arterial-oxygen tension and respiratory failure are more frequently developed in transplanted patients with PJP. A mortality rate was up to 50% among kidney transplant recipients without prophylaxis despite aggressive antibiotic therapy. Before the introduction of prophylaxis, the incidence of PJP was in approximately 5-24% of post-transplant patients, depending on the transplanted organ or transplant center, with higher prevalence in lung and simultaneous lung and heart transplant recipients. In the era of prophylaxis, this infection rate has been dramatically decreased. Trimethoprim-sulfamethoxazole (TMP-SMX) is efficacious in decreasing the risk of developing PJP post-transplant, and it is the drug of choice for prophylaxis in patients after solid organ transplantation (SOT). The incidence of PJP infection peaked between 3 and 6 months post-transplantation. Therefore, several guidelines-such as the Kidney Disease Improving Global Outcomes (KDIGO) guidelines, the American Society of Transplantation (AST) Infectious Disease Community of Practice, and other reports-usually recommend PJP prophylaxis by using TMP-SMX for 3-12 months after SOT. SOT recipients at high risk of PJP should be considered for an extended duration of prophylaxis. This table summarizes the risk factors associated with PJP in SOT recipients (modified by 2019 AST)

Risk factor	Comments
Immunosuppressive therapies	
Corticosteroids	The median dose and duration of therapy in one series equivalent to 30 mg/day of prednisone for 12 week.
Antibody therapies	antilymphocyte antibodies for graft rejection or induction are linked to the highest risk of PJP being

	in the 1–6 month post-transplant time period. Alemtuzumab may confer the highest risk for PJP of antibody therapies.
Mycophenolate mofetil	Mycophenolate may have anti-pneumocystis effects in animal models and uncontrolled human data, leading to theories about it being protective; definitive data are lacking.
Calcineurin inhibitors	Limited data suggest greater risk with cyclosporine A compared with azathioprine in renal transplantation. Retrospective study with higher incidence of PJP among renal transplant recipients on tacrolimus-based regimens compared with cyclosporine A.
Sirolimus	Sirolimus is associated with a clinical syndrome of interstitial pneumonitis that may be confused with PJP.
Other clinical factors	
CMV disease	Systemic immunosuppressive effects of CMV are an independent risk factor for PJP; CMV, and PJP coinfection well-reported.
Allograft rejection	PJP has been related to the degree and intensity of immunosuppression in transplant recipients and directly linked to treatment for and number of episodes of acute rejection.
Low CD4+ T-cell	About 73% of PJP diagnoses in SOT recipients occurred in patients with CD4+ T-cell counts of <200 cells/ml and were associated with absolute lymphocyte count <500 × 106 cells.
Neutropenia	Prolonged neutropenia has been suggested as a potential risk factor for developing PJP in transplant recipients.