4 Prevention of Bacterial, Viral, Fungal, and Parasitic Infections During the Early Post-transplant Period

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4.1 Evaluating the Pre-transplant Risk of Post-transplant Infections

The risk of infections in the first month post-SOT depends on the type of transplant, potential colonization of donor and recipient with multidrug resistant (MDR) bacteria, and the prolonged maintenance of indwelling vascular lines, chest or abdominal drainage tubes, and intubation devices [1]. Pre-transplant recipient conditions that impact the risk of infection include the underlying illnesses causing organ failure, their severity, and potential immunosuppressive role before transplant. For example, high MELD score (>30) liver transplant candidates have a significantly higher risk for post-transplant infections as compared to low MELD score liver transplant candidates. Chronic malnutrition predisposes to early post-SOT infections, and all efforts should be taken to correct nutritional defects before SOT. Pre-transplant use of steroids or occupational or recreational exposure to fungal pathogens (i.e., farming, gardening) might increase the risk for pre-transplant respiratory tract colonization by filamentous fungi. Similarly, the pre-transplant exposure of both donor and recipient to antibiotic therapies might lead to colonization by multidrug resistant (MDR) bacteria and yeasts and increase the subsequent risk of infection by these pathogens. In recent years, donor-derived infections with MDR bacteria have led to reports of devastating early post-SOT infections in the absence of specific prophylaxis [2]. As a consequence, both donor and recipient evaluation and screening for colonization by MDR pathogens may be indicated in order to tailor specific

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prophylactic measures for the recipient. This includes serologies for latent infections (i.e., *Treponema pallidum*, CMV, EBV, HSV, HIV, and, when indicated, *Toxoplasma gondii, Coccidioides, Trypanosoma cruzi*, *Strongyloides*); interferon gamma release assay (i.e., T-SPOT.TB or QuantiFERON-TB) testing for *Mycobacterium tuberculosis*; rectal, nares, and skin swabs to detect colonization with MDR bacteria; and transplant-specific cultures such as bronchial cultures for lung and urine cultures for kidney transplants. A careful travel history should be obtained whenever feasible to identify risks for infections with endemic pathogens. Table 4.1 summarizes the most frequent risk factors.

Pre-transplant evaluation should provide the opportunity to give vaccines (especially those live vaccines that can't be given after transplant, such as MMR or varicella), prophylaxis (i.e., latent tuberculosis), and treatment (i.e., latent syphilis, hepatitis B or C) when indicated.

Table 4.1 Risk factors for early post-transplant infections

Donor and recipient pre-transplant risk factors	Recipient per-transplant risk factors	Recipient early post-transplant risk factors
Colonization: – MDR bacteria (ESBL, CRAB, MRSA, VRE, etc.) – Yeasts (resistant *Candida* species) – Filamentous fungi (*Aspergillus*, *Mucor*, etc.) – Endemic fungi (*Histoplasma*, etc.) – *Pneumocystis jirovecii* – Respiratory viruses (influenza, RSV, etc.) Latent infections: – *Mycobacterium tuberculosis* – *Mycobacterium abscessus* – Viruses (CMV, HSV, EBV, HBV, HCV, West Nile virus, HIV, etc.) – *Toxoplasma gondii* Recipient specific: – Chronic malnutrition – Advanced organ failure (high MELD score) – Exposure to immunosuppressive agents (steroids, anti-TNF agents, etc.) – Palliative surgery – Mechanical ventilation, indwelling catheters, and drainage tubes	– Prolonged surgery – Extensive bleeding and high number of blood transfusions – Choice of surgical technique (Roux-en-Y biliary anastomosis for liver transplantation) – Technical problems affecting the transplant's functional integrity and vascular supply – Liver: hepatic artery thrombosis – Pancreas: duodenal leaks, splenic artery thrombosis – Kidney: vesicoureteral reflux – Heart: mediastinal bleeding – Lung: bronchial anastomotic leaks, etc.)	– Prolonged intubation and mechanical ventilation – Indwelling vascular catheters (central line catheters) – Abdominal and chest drainage tubes – Ureteral catheters – Persistent hematomas – Undrained collections – Persistent leaks (biliary, urinary, bronchial, etc.) – Prolonged renal replacement therapies (hemodialysis) – Repeated open surgery – Intense immunosuppression – Prolonged broad-spectrum antibiotic therapy – Nosocomial exposure (respiratory viruses, MDR pathogens, filamentous fungi, etc.)

The risk for reactivation of latent pathogens and infections with opportunistic infections diminishes gradually after transplant, with recovery from induction and gradual tapering of immunosuppression toward maintenance therapy. Treatment of rejection with increased immunosuppression augments the risk of infection, however, and prophylaxis against such infections may need to be reinitiated in such instances for some period of time, according to the type and intensity of immunosuppression.

4.2 Prevention of Bacterial Infections

Prophylaxis of bacterial infections is provided during the first days after transplant to prevent infections linked to the surgical act, amplified by the immunosuppression (Table 4.2). Usually antibiotic prophylaxis is kept as short as possible (24–72 h post-transplant) to avoid selection of resistant pathogens and only continued further in the case of patient-specific risks. This prophylaxis is targeted on the recipient's local flora and also potentially based on donor colonization by specific pathogens (MDR) [3]. Carbapenems should only be used in prophylaxis with documented colonization by ESBL-producing *Enterobacteriaceae* [4]. Some centers use organ transport fluid cultures as a means to target antibiotic prophylaxis. This should probably not apply for cultures growing potential contaminants (i.e., coagulase-negative *Staphylococci*). The common use of trimethoprim-sulfamethoxazole (TMP-SMX) to prevent *Pneumocystis jirovecii* infections may provide sufficient antibacterial prophylaxis to kidney transplant recipients against urinary tract infections; some centers add 24 h of prophylaxis with either a quinolone or a second-generation cephalosporin. For liver, heart, and lung transplants, reasonably broad Gram-positive and Gram-negative coverage (such as that provided by a second-generation cephalosporin or a penicillin with a beta-lactamase inhibitor) for 72 h might be sufficient in the absence of colonization by MRSA, VRE, or resistant Gram-negative bacilli. In case of pre-transplant colonization of kidney and liver recipients by carbapenem-producing *Enterobacteriaceae* (CPE), data about the benefit-risk of tailored prophylaxis is

Table 4.2 Prevention of early post-transplant bacterial infections

Transplant	24–96 h post-transplant[a]
Kidney	Ciprofloxacin or cefuroxime or cefazolin
Pancreas	Piperacillin-tazobactam + metronidazole for 5–7 days
Intestinal	Piperacillin-tazobactam or cefepime + metronidazole for 4 weeks
Liver	Cefuroxime or piperacillin-tazobactam
Lung	Cefuroxime
	Adapt to recipient/donor bronchial cultures
Heart	Cefuroxime or cefazolin

[a]Always adapt to local epidemiology, as well as pre- and per-transplant culture results of recipient to target patient-specific colonization. Keep duration of prophylaxis as short as possible

lacking. Therefore recent guidelines did not recommend targeted prophylaxis, except in centers with high incidence of surgical site infections [3]. For lung transplant recipients, the prophylaxis should be based on recipient pre-transplant cultures and adapted as soon as cultures of both donor and recipient main bronchi are available [5]. This is of particular importance in the case of cystic fibrosis (CF) recipients, frequently colonized pre-transplant by *Pseudomonas aeruginosa*, *Staphylococcus aureus* (MSSA and MRSA), *Burkholderia cepacia,* non-tuberculous *Mycobacterium* (NTM), and other potential pathogens. After transplant, these pathogens tend to seed the allograft from the recipient sinuses and lead to early severe infections in the absence of aggressive preemptive therapy [6]. Frequently patient-specific antibiotic therapy has to be provided for a few days post-transplant to such patients. In the case of NTM such as *Mycobacterium abscessus*, specific therapy is recommended for up to 12 months [7]. For liver transplant recipients in case of pre-transplant intra-abdominal infections, the prophylaxis should cover the previously identified pathogens. In the case of recurrent pre-transplant biliary infections (i.e., primary sclerosing cholangitis), the risk of peri-surgical intra-abdominal bacterial seeding is substantial and might require a targeted antibiotic prophylaxis for a few days post-transplant. In institutions with a high rate of VRE infections, specific prophylaxis might be used. For pancreas transplant recipients, most programs provide prophylaxis covering both Gram-positive and Gram-negative bacteria, as well as anaerobes for a few days. Intestinal transplant recipients have an extremely high risk of bacterial translocation due to the extensive mucositis during the first month following transplant. Broad-spectrum antibiotic prophylaxis (such as piperacillin-tazobactam or cefepime with metronidazole) is routine in these patients for 4 weeks post-transplant.

During the first few weeks after transplant, bacterial infections occur essentially as consequence of surgical wound infections and technical problems such as anastomotic leaks, urethral reflux, and biliary, bronchial, or urethral stenosis. Source control, including drainage of all accessible sites, is essential. Secondary prophylaxis might be required in the case of recurrent infections but should always be targeted and timely restricted to its minimum to avoid selection of resistant pathogens.

4.3 Prevention of Viral Infections

Prevention of viral infection after SOT may involve either routine monitoring of viral loads by periodic blood testing or prophylaxis with an antiviral agent or immunoglobulin (i.e., hepatitis B virus (HBV) immunoglobulin, cytomegalovirus (CMV) immunoglobulin; these are used less frequently in the era of directly acting antiviral therapy). For those recipients with donors at increased risk of transmission of HBV, hepatitis C virus (HCV), and HIV, routine post-transplant testing by both serology and nucleic acid testing in the first year is recommended [8].

Vaccination prior to transplant can help prevent many viral infections, including hepatitis A virus (HAV) and HBV, measles, mumps, rubella, varicella/zoster virus

(VZV), polio, human papilloma virus, and, for travelers and those with risk for certain exposures, rabies, yellow fever, and Japanese encephalitis. After transplant, non-live vaccines may be given, although they generally have less immunogenicity. The live viral vaccines that should generally be avoided after transplant include measles, mumps, rubella, varicella, zoster (live; recombinant may be safe), polio (oral), rotavirus, and yellow fever.

The human herpes viruses (HHV) are the most common viral infections after transplant and are the predominant preventable viral pathogens, primarily herpes simplex virus (HSV), VZV, and CMV; Table 4.3 summarizes details of routine prophylaxis. For CMV, some groups use preemptive therapy for prevention, with frequent (often weekly) blood checks for several months and initiation of treatment-dose antivirals when a certain threshold is reached [9]; to prevent varicella zoster

Table 4.3 Human herpes virus prophylaxis after kidney, liver, heart, or pancreas transplant [9]

Induction agent	Donor CMV antibody	Recipient CMV antibody	Prophylaxis	Monitoring with CMV viral load
Antithymocyte globulin	Positive	Positive	Valganciclovir × 3 months	Monitoring while on prophylaxis only if clinically indicated by symptoms; consider weekly monitoring after prophylaxis × 8–12 weeks in higher-risk patients and those on more potent immunosuppression
	Negative	Positive		
	Positive	Negative	Valganciclovir × 6 months (plus consider weekly monitoring afterward × 8–12 weeks in higher risk D+R− on more potent IS)	
	Negative	Negative	Acyclovir, famciclovir, or valacyclovir × 3 months[a]	
Basiliximab None	Positive	Positive	Valganciclovir × 3 months	
	Negative	Positive		
	Positive	Negative		
	Negative	Negative	Acyclovir, famciclovir, or valacyclovir × 3 months[a]	

Notes on viral prophylaxis:

- Dosages of all antiviral agents need to be adjusted for renal function. The eGFR or creatinine clearance should be used (not simply the serum creatinine)
- For prophylaxis, the first doses may be oral valganciclovir or intravenous ganciclovir, converting IV to oral as soon as patient tolerating oral medications There is recent data supporting the safety and efficacy of either approach in the treatment (not prophylaxis) setting [23, 24]
- While most kidney transplant recipients (given lower GFR) will need valganciclovir 450 mg a day (or less), some may have GFR > 60 and need valganciclovir 900 mg a day. Minidosing not recommended
- Lung transplant prophylaxis would be similar, although generally with longer courses of prophylaxis

[a]In case of either HSV or VZV, D+ or R+; if all are negative, no need for prophylaxis

and herpes simplex viruses, clinicians may wish to add acyclovir, valacyclovir, or famciclovir. Other HHV, such as Epstein-Barr virus (EBV), HHV-6, and HHV-8, are less amenable to prophylaxis. Vaccination with varicella and live viral zoster vaccines should be done before the onset of immunosuppression [10]; the recombinant zoster vaccine may be useful after transplantation.

EBV infection augments the risk of EBV-positive post-transplant lymphoproliferative disease (PTLD), especially in those who are EBV D+R− and those who undergo multivisceral/small bowel and thoracic transplants. In these EBV D+R− recipients, post-transplant monitoring periodically for the first 1–2 years can identify those at higher risk for PTLD; when possible, reduction of immunosuppression may sometimes help diminish the viremia [11]. Antiviral medication has not been found to be effective in either preventing or decreasing EBV viremia.

HBV can be prevented by pre-transplant diagnostic testing, including HBV core and surface antibody (both IgG), surface antigen, and sometimes viral load (to detect the rare cases when the surface antigen is negative but viral load positive). If any of those are positive, additional studies can be sent (HBVe antigen and antibody, hepatitis D antibody and antigen). Pre-transplant vaccination is recommended for all nonimmune organ transplant recipients; higher doses of vaccine are more likely to provide protection in those with chronic organ disease. Antiviral treatment can be given for acute or chronic active infection or when there is a risk of reactivation or transmission from the donor (i.e., HBV core antibody positive) [12].

HCV management has changed rapidly in recent times, given the advent of highly active therapies. Although some recipients are treated prior to transplant, some are now treated after transplant, in part to allow them to undergo transplant from donors with HCV, which may shorten the waiting time for organs and provide access to organs from younger donors with fewer comorbidities [13]. Numerous programs are now using HCV-positive donors in recipients without HCV and treating after transplant (often initiating therapy immediately, and primarily in research settings); early work demonstrates acceptable outcomes [14]. Prevention approaches for HCV involve both monitoring by viral load and serology and use of various treatment methods when indicated.

Hepatitis E virus (HEV) causes acute and chronic hepatitis in SOT recipients, especially in endemic regions, where pre-transplant screening of donors and recipients may be useful.

BK polyomavirus (BKPyV) causes nephropathy primarily in kidney transplant recipients and often reflects relative over-immunosuppression. Over 85% of adults have prior exposure and latent viral infection. Prevention is best done through periodic (every several months) urine and/or blood viral load testing during the first 2 years after transplant [15, 16]. Urine viral loads are often positive before blood. When a certain threshold has been achieved, clinicians may wish to reduce immunosuppression, as the best method to help clear BKPyV infection. With extensive, unremitting infection, some programs use antiviral therapy [16].

4.4 Prevention of Fungal Infections

In recent years, efforts have been made to identify risk factors for post-transplant fungal infections allowing risk stratification and to tailor antifungal prophylaxis individually to each recipient [17]. This takes into account center-specific epidemiologic data, potential pre-transplant and post-transplant environmental exposure to filamentous or endemic fungi, and pre-transplant colonization (Table 4.4).

Table 4.4 Prevention of fungal infections

Transplant	Fungus	Risk factors	Prophylaxis	Suggested duration[a]
Kidney	*Pneumocystis* *Candida* *Aspergillus*	All patients Candiduria Proven colonization, high-dose steroids, acute rejection, CMV infection	TMP-SMX Fluconazole Aerosolized amphotericin B, voriconazole	6 months 10–14 days 4–6 weeks
Pancreas	*Pneumocystis* *Candida* *Aspergillus*	All patients All patients Proven colonization, high-dose steroids, acute rejection, CMV infection	TMP-SMX Fluconazole, echinocandin Aerosolized amphotericin B, voriconazole	12 months 14 days 4–6 weeks
Intestinal	*Pneumocystis* *Candida* *Aspergillus*	All patients All patients Proven colonization, high-dose steroids, acute rejection, CMV infection	TMP-SMX Fluconazole, echinocandin Aerosolized amphotericin B, voriconazole	12 months 4 weeks 4–6 weeks
Liver	*Pneumocystis* *Candida* *Aspergillus*	High MELD score (>30), ATG, CMV disease, second transplant >2 risk factors: broad-spectrum antibiotics >5 days, yeast colonization >3 body sites, ICU >5 days, post-transplant hemodialysis, retransplantation or need for second surgery, choledocojejunostomy, high transfusion requirement, and pancreatitis Proven colonization, high-dose steroids, primary allograft failure or severe dysfunction, hemodialysis, retransplantation, acute rejection, CMV infection	TMP-SMX Echinocandin followed by fluconazole IV or Aerosolized amphotericin B, mold-active azoles (voriconazole, posaconazole, or isavuconazole)	6–12 months 2–4 weeks 4–6 weeks

(continued)

Table 4.4 (continued)

Transplant	Fungus	Risk factors	Prophylaxis	Suggested duration[a]
Lung	*Pneumocystis* *Candida* *Aspergillus*	All patients None All patients or according to risk factors: proven colonization, high-dose steroids, retransplantation, acute rejection, CMV infection	TMP-SMX – Aerosolized amphotericin B, voriconazole	12 months, lifelong – 4–6 weeks, lifelong
Heart	*Pneumocystis* *Candida* *Aspergillus*	All patients None Proven colonization, high-dose steroids, primary allograft failure or severe dysfunction, hemodialysis, retransplantation, acute rejection, CMV infection	TMP-SMX – Aerosolized amphotericin B, voriconazole	12 months, lifelong (depending on toxoplasma status) – 4–6 weeks

[a]Suggested durations from the University Hospitals Geneva, Switzerland

Except for low-risk liver transplant recipients, prophylaxis against *P. jirovecii* is recommended for all SOT recipients for 6–12 months, and some centers provide livelong prophylaxis for lung transplant recipients. Most centers use TMP-SMX (also providing protection against *Toxoplasma*, *Nocardia*, and urinary tract infections). In case of intolerance, oral atovaquone, dapsone, and aerosolized pentamidine are alternatives.

Candida sp. infections mainly occur as nosocomial infections during the first month post-transplant. Lung, heart, and kidney transplant recipients do not generally require systemic yeast prophylaxis; oral amphotericin B or nystatin is frequently provided to prevent thrush. Liver transplant recipients should be evaluated in a risk stratification to decide whether systemic anti-yeast prophylaxis is justified. Prophylaxis is given in the presence of more than two of the following risk factors: broad-spectrum antibiotics for more than 5 days, yeast colonization of more than three body sites, ICU for more than 5 days, post-transplant hemodialysis, retransplantation or need for second surgery, choledocojejunostomy, high transfusion requirement, and pancreatitis [17]. Early after liver transplant, an echinocandin might be preferred, once the liver function has recovered, and taking into account local epidemiology, a switch toward fluconazole might be considered. The duration should take into account the persistence of the risk factors. Pancreas and intestinal transplant recipients are at highest risk and should receive systemic anti-yeast prophylaxis for 2 and 4 weeks, respectively, post-transplant.

Aspergillus and *Mucor* infections are a serious concern following SOT because of their high-associated mortality. Given the toxicity, side effects, and drug interactions of antifungal prophylaxis, efforts should be made to identify SOT recipients at increased risk for invasive fungal infections. Recipients colonized at the time of transplant should receive prophylaxis for at least 4–6 weeks. Risk factors common

to all transplants include proven colonization, high-dose steroids, primary allograft failure or severe dysfunction, hemodialysis, retransplantation, acute rejection, and CMV infection. Lung transplant recipients, especially CF patients, are at high risk for *Aspergillus* infections. Some centers provide universal prophylaxis with either aerosolized amphotericin B or mold-active azoles (voriconazole, posaconazole, or isavuconazole); others prefer to give prophylaxis only in the presence of documented colonization or other risk factors. Voriconazole has been associated with a higher incidence of skin cancer in lung transplant recipients [18]. The strategy should be adapted according to local epidemiology.

Cryptococcus neoformans infections may occur before (especially with liver disease) or after transplant; prophylaxis with fluconazole is only recommended in the presence of positive cryptococcal antigen detection or with a documented history of disease. Similarly, those at risk for *Coccidioides* after transplant should be given prophylaxis with fluconazole.

4.5 Prevention of Parasitic Infections

Parasitic infections are less common after transplant and may be more challenging to diagnose. Acknowledging the risk of reactivation and donor-to-recipient transmission, based on donor and recipient exposures, may be the first step in preventing these infections. While most transplant recipients would be given *Toxoplasma* prevention, prevention of *Trypanosoma cruzi*, *Schistosoma*, *Leishmania*, malaria, *Babesia*, and others would only occur when risk was identified.

Symptomatic toxoplasmosis has been well described, primarily after heart transplant, and may present with myocarditis, brain abscess, pneumonitis, or disseminated disease. Without prophylaxis, those who are D+/R− have a 50% to 75% risk of symptomatic infection within the first few months. While rates of positivity are low in the United States, they can be much higher in Europe, Brazil, and elsewhere [19]. TMP-SMX is the most common prophylaxis. While pyrimethamine with sulfadiazine is effective and has been used for high-risk cardiac recipients, it does not seem to be essential based on clinical data and experience, as TMP-SMX alone has been sufficient and better tolerated; Table 4.5 outlines further details.

Strongyloides infections can be latent for decades, due to the autoinfection loop, and develop into clinically significant disease, from gastrointestinal to disseminated [20]. Both donors and recipients from endemic regions should be screened, with a plan to initiate treatment with ivermectin, thiabendazole, or albendazole if needed. In deceased donors, screening usually involves serology; there have been numerous cases of donor-derived infection [21]. Living donors and recipients may be screened by serology or by several stool specimens, as the sensitivity of an individual stool study is low. Those who have concomitant microfilarial disease and who are given ivermectin may experience the Mazzotti reaction, with fever, adenopathy, pruritus, abdominal pain, and even angioedema; it is best to screen those from endemic regions within the past 5–7 years (primarily Africa and Asia) for microfilaria by

Table 4.5 Duration of toxoplasmosis prophylaxis after heart transplant based on serologic combinations (Massachusetts General Hospital)

Serologic combination (donor/recipient)	Risk group	Treatment and dosing	Duration of therapy
D+R−	Highest risk	TMP-SMX DS[a] (some centers use SS) every day (if DS dose reduce to Bactrim SS q day if GFR < 30) × 12 months and then TMP-SMX SS every day (no need for dose reduction with renal insufficiency even ESRD/dialysis)[b]	Lifetime, if possible (otherwise discuss with infectious disease)
R+ (D+ or D−)	Moderate risk	TMP-SMX SS[a] every day (no need for dose reduction with renal insufficiency even ESRD/dialysis)	Can stop at 1 year, or when on low-dose immunosuppression (i.e., prednisone 5 mg a day), whichever is *later/longer* Restart during intensification of immunosuppression (i.e., pulse-dose steroids, ATG, or Rx of AMR) for same period as after transplant
D−R−	Lowest risk	Same as for R+ for *Pneumocystis* and other preventions, although not needed for toxoplasmosis	

[a]Trimethoprim-sulfamethoxazole (TMP-SMX) DS (double strength) is sulfamethoxazole 800 mg and trimethoprim 160 mg, while TMP-SMX SS (single strength) is half that dose
[b]If true TMP-SMX allergy documented, second-line prophylaxis would be with atovaquone1500 mg a day or dapsone 100 mg a day. With dapsone, breakthrough toxoplasmosis infection could occur, as could methemoglobinemia, and G6PD should be checked before starting treatment to avoid hemolysis if deficient

blood smear. For latent *Strongyloides* infection, ivermectin is often given as one or two daily doses, with a repeat series 2 weeks later, due to the autoinfection cycle and the efficacy of ivermectin at only certain stages in the parasite lifecycle; the optimal regimen has not been defined [20]. Those who are seropositive for HTLV are at risk for recurrent *Strongyloides,* sometimes necessitating repeat treatment; HTLV serology should be checked when positive *Strongyloides* serology is found.

Chagas disease is caused by *Trypanosoma cruzi* and generally occurs in those from Central and South America or who have received organs or blood products from infected people. Pre-transplant serologic testing of donors and recipients from endemic regions is recommended [22]. Rates of transmission from positive donors to recipients are significant; acceptance of hearts from positive donors is not recommended, and recipients of other organs from positive donors should undergo transplant only after informed consent. If either are positive, post-transplant screening by blood PCR or smear weekly for the first few months may detect early infection, at which point preemptive treatment with benznidazole (or nifurtimox) would be indicated [22]. Prophylaxis with these agents is not generally done, due to lack of efficacy data and significant toxicity.

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