

Transplant infection

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THE STORY IN ONE SENTENCE

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“Transplant infection is a predictable calendar: month 1 brings hospital bugs, months 2–6 bring hidden viruses and fungi, and after 6 months come everyday colds plus slow stealth germs—because the anti-rejection drugs knocked out the immune army that normally stops them.”

Normal anatomy — the body's job

A healthy immune system has two layers that stop germs before they make you sick: the innate team (neutrophils, macrophages, and barriers like skin and mucous membranes) blocks invaders fast and crude, and the adaptive team (T-cells and antibodies) hunts down hidden viruses, fungi, and parasites with precision.

BODY'S JOB

The innate layer reacts in minutes—neutrophils swarm bacteria, macrophages eat debris, and tight skin and mucous membranes seal the body like a castle wall. The adaptive layer takes days to ramp up but has memory—T-cells recognize viruses hiding inside cells and kill infected cells, B-cells make antibodies that tag germs for destruction, and memory cells remember past invaders so the second attack is faster. Together, these two layers stop everyday colds, food bugs, and opportunistic germs (like *Pneumocystis* or CMV) that only grow when the immune system is weak.

1. Skin, mucous membranes, and stomach acid form the first wall—germs that cross it face neutrophils and macrophages that eat and kill them within hours.
2. If a germ survives, dendritic cells grab pieces of it and carry them to lymph nodes, where they wake up T-cells and B-cells.

3. Helper T-cells (CD4+) coordinate the attack, killer T-cells (CD8+) destroy infected cells, and B-cells pump out antibodies that stick to germs and mark them for cleanup.
4. Memory T-cells and B-cells stay in the blood for years, so if that same germ comes back, the immune system crushes it before you even feel sick.
5. This layered defense keeps opportunistic germs (Pneumocystis, CMV, Cryptococcus, Toxoplasma) locked down—they live in soil, water, or dormant in your body but never get a chance to grow because the immune army patrols constantly.

What breaks

Transplant patients take immunosuppressive drugs—calcineurin inhibitors (tacrolimus, cyclosporine), antimetabolites (mycophenolate, azathioprine), corticosteroids (prednisone), and monoclonal antibodies (basiliximab, alemtuzumab)—to stop T-cells from attacking the new organ, but these drugs also cripple the innate barriers and adaptive hunters that kill germs, leaving the body wide open to infections that healthy people never get.

CLINICAL TERM

Immunosuppression blocks T-cell activation and innate immune function, creating a dose- and time-dependent vulnerability to infection.

How the body compensates

The body tries to fight infection despite weak immunity by raising fever (to slow germ growth), increasing heart rate and breathing (to deliver more oxygen to inflamed tissue), and shedding infected cells (cough, diarrhea)—but these compensations can fail suddenly when the germ load overwhelms weakened defenses, leading to sepsis, respiratory failure, or organ dysfunction.

1. Fever rises because the hypothalamus (the brain's thermostat) resets higher in response to cytokines (inflammatory signals) released by the few active immune cells, creating heat that slows bacterial and viral replication and speeds enzyme activity in white blood cells.
2. Heart rate and respiratory rate climb to pump more oxygen-rich blood to infected tissue, trying to keep organs fed while the body battles the germ load.
3. Cough, coughed-up mucus (sputum), and diarrhea are the body trying to flush out infected mucus and cells from the lungs and gut, clearing the battlefield so new immune cells can move in.
4. Local lymph nodes swell as dendritic cells carry germ fragments there to activate any remaining T-cells and B-cells, trying to mount even a small adaptive response.

5. The danger zone: because the immune system is chemically blocked, these compensations are weak and short-lived—fever may be blunted by steroids, white blood cell count may stay normal despite infection, and the patient can look stable until the germ burden tips into sepsis, low oxygen (hypoxia), or multi-organ failure in hours.
6. Warning signs that compensation is failing: persistent fever despite antibiotics, oxygen saturation dropping, confusion or lethargy (brain low oxygen (hypoxia)), rising lactate (cells starving), falling blood pressure (vasodilation from sepsis), or worsening kidney or liver numbers (organs shutting down from poor blood flow (perfusion)).

Domino effect

1. Transplant surgery and induction immunosuppression (monoclonal antibodies, high-dose steroids) wipe out most T-cells and slow neutrophils and macrophages right away. — The anti-rejection drugs hit the immune system like a sledgehammer to protect the new organ, but that leaves the body defenseless—neutrophils are sluggish, macrophages do not patrol well, and T-cells are depleted or blocked from activating.
2. In month 1, hospital-acquired bacteria (MRSA, Pseudomonas, E. coli, Klebsiella) and Candida yeast invade through catheters, surgical wounds, and breathing tubes. — Surgical sites and device insertion points are fresh wounds with broken skin barriers, and the weakened neutrophils cannot swarm fast enough to kill the germs that hospitals harbor—so resistant bacteria and yeast colonize lines, lungs, and bladder and cause bloodstream infections, pneumonia, and wound abscesses.
3. Between months 2 and 6, opportunistic pathogens explode: CMV (cytomegalovirus) reactivates, Pneumocystis jirovecii (PJP) grows in the lungs, EBV (Epstein-Barr virus) can trigger lymphoma (PTLD), and fungi (Aspergillus, Cryptococcus) or parasites (Toxoplasma) invade. — T-cells are the hunters that kill cells infected with viruses and stop slow-growing fungi and parasites—without them, CMV hiding in blood cells wakes up and spreads to gut and lungs, Pneumocystis fills lung air sacs with frothy fluid, EBV-infected B-cells multiply out of control forming tumors, and environmental fungi inhaled from soil or bird droppings grow into deadly brain or lung masses.
4. After 6 months, two risks persist: community-acquired infections (influenza, RSV, COVID, pneumococcus) and late opportunistic infections (chronic CMV, endemic fungi like Histoplasma or Coccidioides, mycobacteria). — Long-term maintenance immunosuppression keeps T-cells and antibody responses low, so seasonal viruses that healthy people shake off can cause severe pneumonia, and slow germs from the environment (fungal spores in certain regions, TB in endemic areas, or persistent CMV that never fully cleared) reactivate or progress because the immune system never recovers full strength.

5. Each infection can trigger a cascade: viral infections raise immunosuppression indirectly (the team dials up drugs to protect the organ during stress), bacterial superinfections follow viral lung damage, and fungal infections seed the bloodstream and brain. — Infection stresses the transplanted organ, so doctors may increase immunosuppression to prevent rejection—but that makes the infection worse, creating a vicious cycle. A flu infection damages lung lining, letting bacteria invade and cause pneumonia. Fungal spores inhaled into damaged lungs spread through blood to the brain (cryptococcal meningitis) or form abscesses, and without T-cells to wall them off, fungi disseminate and kill.
6. Prevention (Prophylaxis) (preventive antibiotics and antivirals) and early detection (regular CMV PCR, fungal markers, chest imaging) are the only strategies that break the domino chain before it reaches sepsis, respiratory failure, PTLT, or disseminated fungal infection. — Because the immune system cannot protect itself, doctors give trimethoprim-sulfamethoxazole to prevent PJP, valganciclovir to suppress CMV, and sometimes antifungals in high-risk patients—these drugs act as a chemical immune system while the real one is shut down. Screening blood for CMV DNA every week in the first months catches reactivation before it spreads, and any new fever or symptom gets worked up fast (cultures, imaging, bronchoscopy if needed) because waiting even a day can let a small infection explode into organ failure.
7. When a serious infection is confirmed, the clinical team must balance two hard goals: treat the infection aggressively (antibiotics, antivirals, antifungals) while reducing immunosuppression enough to let the immune system help, but not so much that the transplanted organ gets rejected. — It is a tightrope walk—lower the immunosuppression too much and the body attacks the new organ (acute rejection), but keep it too high and the infection kills the patient. The team monitors drug levels daily, watches for rejection signs (rising creatinine in kidney transplant, liver enzymes in liver transplant), and adjusts doses in real time, often in the ICU, because both rejection and infection can cause organ failure and death within days.

8. The endpoint of untreated or late-treated transplant infection is multi-organ failure: septic shock (blood pressure crashes from bacterial toxins), ARDS (lungs fill with fluid and cannot oxygenate), encephalitis (viruses or fungi invade the brain), or graft loss (the transplanted organ fails from infection or rejection), often leading to ICU care, ventilator support, dialysis, and high mortality. — When the immune system is chemically blocked and the germ load grows unchecked, the infection spreads everywhere—bacteria release endotoxins that dilate blood vessels and crash blood pressure (septic shock), lungs leak fluid into air sacs from inflammation (ARDS), CMV or fungi cross into the brain causing confusion and seizures (encephalitis), and the transplanted organ itself gets infected or inflamed and stops working, forcing the patient back onto dialysis or a transplant list. The body is trying to fight with the few immune cells left, but without T-cells and functional neutrophils, the effort fails, and supportive care (pressors, oxygen, dialysis, ICU antibiotics) is all that stands between the patient and death while waiting for the drugs to kill the germs.

Symptoms · what the patient feels, and why

- Fever (temperature above 100.4°F or 38°C): The transplanted organ or nearby tissue is fighting an infection. The immune system — even though it's dialed down by anti-rejection drugs — still releases signals called cytokines (messenger proteins that turn up the body's thermostat) when germs invade.
- Pain, redness, warmth, or swelling at the surgical site or around the transplanted organ: Germs (bacteria, viruses, or fungi) have gotten into the wound or tissue. Even with weaker defenses, the body sends white blood cells and fluid to the area, which makes it swell, feel hot, and hurt.
- The transplanted organ stops working as well (like rising creatinine in a kidney transplant or rising liver enzymes in a liver transplant): Infection inside or around the new organ damages its cells directly, or the inflammation clogs tiny blood vessels so the organ can't get enough oxygen and nutrients to do its job.
- Cough, trouble breathing, or low oxygen levels (in lung transplant or widespread infection): Germs — especially fungi like *Aspergillus* or viruses like CMV (cytomegalovirus, a common virus that hides in the body and wakes up when defenses are low) — infect the lungs. The air sacs fill with fluid and pus, blocking oxygen from getting into the blood.
- Chills and shaking (rigors): Bacteria or fungi have spilled into the bloodstream (called bacteremia or fungemia). The body's thermostat in the hypothalamus (a control center deep in the brain) swings wildly, causing violent shivering to generate heat.
- Feeling very tired, weak, or confused: Infection uses up the body's energy and oxygen. Cytokines (the alarm proteins) also slow down the brain and muscles. If the infection spreads, low blood pressure means less oxygen reaches the brain, causing confusion or fog.

- Diarrhea, belly pain, or nausea (especially with CMV or *C. difficile* infection): CMV (cytomegalovirus) can attack the lining of the intestines, causing sores that bleed and hurt. *C. difficile* (a bacterium that overgrows when antibiotics kill good gut germs) releases toxins that inflame the colon and trigger watery, sometimes bloody diarrhea.
- New or worsening fluid buildup (like swelling in the legs, belly, or around the lungs): Infection damages blood vessel walls, letting fluid leak out. If the transplanted organ (like the kidney or heart) isn't working well because of infection, the body can't pump or filter fluid properly, so it pools in the tissues.

Focused exam

- Fever without clear source in the first month after transplant: Early fever usually means germs from the hospital—bacteria like *Pseudomonas* or *E. coli*, or fungi like *Candida*—snuck in during surgery or through catheters and IV lines. Because the immune system is turned way down to protect the new organ, these germs spread fast. You need to find the source (blood, urine, wound, line) and treat it immediately before it turns into sepsis.
- Cough, shortness of breath, or oxygen saturation below 92% on room air between 1 and 6 months post-transplant: This is the classic danger window for *Pneumocystis jirovecii* pneumonia (PJP), a fungus that attacks the lungs when T-cells are crushed by immunosuppression. Low oxygen means the fungus is clogging the air sacs where oxygen enters the blood. If you see this pattern and the patient is not on TMP-SMX prevention (prophylaxis), PJP is at the top of your list. Untreated PJP can kill in days.
- Blurred vision, floaters, headache, confusion, or altered mental status: These can mean the infection has spread to the eyes or brain. Cytomegalovirus (CMV) can cause retinitis (inflammation of the retina that leads to blindness if untreated). *Toxoplasma*, *Cryptococcus*, or *Aspergillus* can invade the brain and cause encephalitis or abscess. These are late, serious findings that mean the infection is ahead of the immune system. Brain and eye infections in transplant patients are emergencies.
- New or enlarging skin nodules, mouth ulcers, or swollen lymph nodes: Epstein-Barr virus (EBV) can drive unchecked B-cell growth in transplant patients, causing post-transplant lymphoproliferative disorder (PTLD)—a cancer-like illness that behaves like lymphoma. Skin nodules, mouth sores, and swollen nodes are clues that EBV is pushing cells to multiply out of control. Early recognition lets you cut back immunosuppression or start targeted therapy before it spreads everywhere.

- Rising creatinine (kidney) or rising bilirubin and transaminases (liver) on routine labs: The transplanted organ itself can get infected—BK virus attacks transplanted kidneys, CMV attacks transplanted livers and intestines. Falling organ function (rising creatinine in kidney transplant, rising liver enzymes in liver transplant) means the infection is damaging the graft. You need to figure out if it is infection, rejection, or both, because the treatments pull in opposite directions.
- Redness, warmth, pus, or tenderness at a central line, urinary catheter, or surgical incision site: Catheters and lines are doorways for hospital bacteria and fungi to enter the bloodstream. In a transplant patient whose immune system cannot wall off infection, a small line infection can seed the blood and cause sepsis in hours. Catching it early—removing the line, culturing it, and starting antibiotics—stops the chain before it becomes life-threatening.

Diagnostics · every test answers a question

- Complete blood count with differential (CBC with diff) → Is the white blood cell count dangerously low (neutropenia) or dangerously high (suggesting bacterial infection or PTLD)?
- Blood cultures (aerobic and anaerobic, two sets from separate sites) → Is there bacteria or yeast in the bloodstream?
- CMV quantitative PCR (viral load) in plasma → Is cytomegalovirus actively replicating in the blood?
- BK virus quantitative PCR in plasma (kidney transplant) and urine → Is BK virus replicating and damaging the transplanted kidney?
- Bronchoalveolar lavage (BAL) with PJP staining (Gomori methenamine silver or immunofluorescence), fungal culture, bacterial culture, and viral PCR panel → What pathogen is in the lungs causing the cough, shortness of breath, and low oxygen?
- Chest CT with contrast (if respiratory symptoms) → What pattern of lung disease is present, and does it point to a specific infection?
- Serum creatinine, BUN, liver function tests (AST, ALT, bilirubin, alkaline phosphatase), and tacrolimus or cyclosporine trough level → Is the transplanted organ failing, and is the immunosuppression level in the safe range?
- EBV quantitative PCR in plasma (if swollen lymph nodes (lymphadenopathy), constitutional symptoms, or atypical lymphocytosis) → Is Epstein-Barr virus driving uncontrolled B-cell proliferation (PTLD)?

Treatment logic

- Broad-spectrum empiric antibiotics (e.g. piperacillin-tazobactam or meropenem plus vancomycin) started immediately when fever appears in the first month post-transplant → Covers hospital-acquired bacteria—gram-negatives like *Pseudomonas* and *E. coli*, gram-positives like MRSA and coagulase-negative Staph—before they cause sepsis or seed the new organ. Early treatment (within 1 hour of fever recognition) prevents septic shock and graft loss. Once cultures identify the organism, you narrow to targeted therapy.
- TMP-SMX (trimethoprim-sulfamethoxazole) prevention (prophylaxis) started at transplant and continued for 6–12 months (single-strength or double-strength daily or three times weekly) → Prevents *Pneumocystis jirovecii* pneumonia (PJP) by killing the fungus before it colonizes the lungs. Also prevents *Toxoplasma gondii* reactivation and some urinary tract infections. PJP is one of the deadliest opportunistic infections in transplant—prevention (prophylaxis) drops the risk from 10–15% to under 1%.
- Valganciclovir prevention (prophylaxis) for 3–6 months in high-risk CMV mismatch (donor CMV-positive, recipient CMV-negative, or recipient CMV-positive on heavy immunosuppression) → Suppresses CMV replication before it causes pneumonitis (lung inflammation), colitis (bloody diarrhea), retinitis (blindness), or hepatitis. CMV is the most common opportunistic viral infection after transplant. Prevention (Prophylaxis) prevents disease in patients who have never seen CMV before (seronegative recipients) or who cannot mount memory T-cell responses.
- Antifungal prevention (prophylaxis) with fluconazole (for *Candida*) or echinocandin or posaconazole/voriconazole (for invasive mold in high-risk patients like lung or liver transplant) for 1–3 months → Prevents invasive yeast (*Candida*) and, with broader agents, invasive molds (*Aspergillus*) from seeding the bloodstream, lungs, or brain. Lung and liver transplant recipients are at highest risk because of heavy immunosuppression and surgical exposure. Prevention (Prophylaxis) cuts the risk of deadly invasive fungal infection by 50–70%.
- Reduce or temporarily hold calcineurin inhibitor (tacrolimus or cyclosporine) and antimetabolite (mycophenolate) doses if life-threatening opportunistic infection (PJP, CMV, invasive fungal infection) develops, while continuing or adjusting corticosteroid → Lets the patient's T-cells and other immune defenses wake up enough to help fight the current infection without rejecting the transplanted organ. This rebalances the immune scale: too much immunosuppression kills the patient with infection, too little kills the graft with rejection. Dose reduction is a bridge while antimicrobials work.

- Pre-transplant vaccination of recipient (and living donor) with inactivated vaccines: influenza, pneumococcal (PCV13 followed by PPSV23), hepatitis B series, and MMR/varicella if seronegative and at least 4 weeks before transplant; avoid all live vaccines post-transplant → Builds memory immunity while the immune system still works at full strength. Prevents vaccine-preventable infections (flu, pneumococcal pneumonia, hepatitis B, measles, chickenpox) that can be severe or deadly in immunosuppressed patients. Post-transplant, inactivated vaccines (flu, pneumococcal boosters) can still be given but response is weaker.
- IV immunoglobulin (IVIG) or CMV hyperimmune globulin in selected high-risk patients (e.g. severe hypogammaglobulinemia, refractory CMV despite antivirals) → Provides passive antibodies that help neutralize viruses (especially CMV) or bacteria when the patient's own B-cells cannot make enough antibody. Used as rescue therapy when antivirals fail or in patients with very low IgG (below 400 mg/dL). Does not replace antivirals or prevention (prophylaxis) but can tip the balance in desperate cases.

Medications

Trimethoprim-sulfamethoxazole (TMP-SMX) · Antibiotic combination (folate synthesis blocker)

TMP-SMX stops a tiny germ called *Pneumocystis jirovecii* (the PJP germ) from building the parts it needs to grow. This germ loves lungs and can cause deadly pneumonia in transplant patients whose immune system is turned down. The drug blocks two steps the germ uses to make folate (a vitamin-like molecule the germ must have to copy its DNA and multiply).

Without folate, the germ cannot spread.

1. Trimethoprim blocks an enzyme called dihydrofolate reductase (the last step in making active folate inside the germ).
2. Sulfamethoxazole blocks an earlier enzyme called dihydropteroate synthase (an earlier step in the same folate assembly line).
3. Together they hit two steps in one pathway, making it nearly impossible for the germ to build folate.
4. Without folate, the germ cannot make DNA or RNA, so it cannot reproduce or spread.
5. The drug is given daily or three times a week for 6–12 months after transplant, when the immune system is weakest.

SAFETY WATCH

Can cause low blood counts (fewer white cells, red cells, or platelets) because human cells also use folate—watch complete blood counts monthly. · May hurt the kidneys, especially if the patient is dehydrated or taking other kidney-toxic drugs (calcineurin inhibitors like tacrolimus)—check creatinine regularly. · Can raise potassium levels (hyperkalemia), especially in kidney transplant patients—monitor potassium. · Allergic rash (including severe Stevens-Johnson syndrome) can happen; stop the drug immediately if a painful rash or mouth sores appear. · Nausea and headache are common but usually mild.

Valganciclovir · Antiviral (DNA polymerase inhibitor)

Valganciclovir stops cytomegalovirus (CMV, a herpes-family virus) from copying its DNA. CMV hides in most people's bodies but explodes when the immune system is suppressed after transplant, attacking the gut, lungs, eyes, and the new organ itself. The drug is a fake building block that gets inserted into the virus's growing DNA chain, jamming the copy machine so the virus cannot multiply.

1. Valganciclovir is swallowed as a pill and is absorbed in the gut.
2. Inside the body it is converted to ganciclovir (the active form).
3. Ganciclovir enters CMV-infected cells and is activated by a viral enzyme called UL97 kinase (the virus itself turns on the drug).
4. Once activated, it gets mistaken for a normal DNA building block and is added to the growing viral DNA strand.
5. Because it is a fake piece, the DNA chain stops growing—the virus cannot finish copying its genome.
6. The virus cannot make new virus particles, so the infection is held in check.
7. Given daily for 3–6 months in high-risk patients (donor CMV-positive and recipient CMV-negative, or any recipient getting strong T-cell-depleting induction therapy).

SAFETY WATCH

Bone marrow suppression: can drop white blood cells (neutropenia, raising infection risk) and platelets (raising bleeding risk)—check complete blood count weekly at first, then every 2 weeks. · Kidney toxicity: must dose-adjust based on creatinine clearance; too much drug in kidney transplant patients can worsen kidney function. · Diarrhea and nausea are common. · Rare but serious: seizures, confusion, or low blood sugar. · Do not use during pregnancy (teratogenic—harms the developing baby); reliable birth control is required.

Fluconazole or other azole antifungal (e.g., voriconazole, posaconazole) · Azole antifungal (ergosterol synthesis inhibitor)

Azole antifungals stop fungi (like *Candida* and *Aspergillus*) from building their cell membrane. Fungi use a fat-like molecule called ergosterol to keep their membrane strong—like the plastic wrap around the cell. Azoles block the enzyme that makes ergosterol. Without it, the fungal membrane leaks and the fungus dies or stops growing.

1. The azole drug enters fungal cells.
2. It binds to and blocks lanosterol 14-alpha-demethylase, an enzyme fungi need to turn lanosterol into ergosterol.
3. Without ergosterol, the fungal membrane becomes weak and full of holes.
4. The fungus cannot grow or divide properly, and the immune system (even a weakened one) can finish the job.
5. Fluconazole is used for *Candida* prevention (prophylaxis) (preventing yeast infections of the mouth, throat, and bloodstream).
6. Voriconazole or posaconazole are used if there is high risk of mold infections like *Aspergillus* (lung transplant, prolonged dangerously low infection-fighting cells (neutropenia), or prior fungal infection).

SAFETY WATCH

Liver toxicity: azoles can raise liver enzymes (AST, ALT)—check liver function tests regularly. · Drug interactions are huge: azoles strongly inhibit cytochrome P450 enzymes, raising blood levels of calcineurin inhibitors (tacrolimus, cyclosporine) and mTOR inhibitors (sirolimus), which can cause kidney damage and over-immunosuppression. Dose of immunosuppressants must be cut, often by 50% or more, and levels monitored closely. · QT prolongation (heart rhythm risk)—avoid in patients with long QT or those on other QT-prolonging drugs. · Nausea, rash, and headache are common. · Voriconazole can cause visual changes (blurry vision, light sensitivity) and, with long-term use, skin cancer and bone pain.

Immunosuppressive agents (tacrolimus, mycophenolate, prednisone) · Immunosuppressants (calcineurin inhibitor, purine synthesis blocker, corticosteroid)

These drugs are not treating infection—they are preventing the transplanted organ from being rejected. But they are listed here because managing infection in transplant patients means carefully balancing these drugs. Too much suppression invites infection; too little risks losing the organ. Tacrolimus blocks T-cell activation (the immune cells that would attack the new organ). Mycophenolate stops lymphocytes from multiplying. Prednisone broadly dampens

inflammation and immune response. During serious infection, doses are often reduced temporarily to let the immune system fight back, then raised again once infection is controlled.

1. Tacrolimus enters T-cells, binds a protein called FKBP12, and together they block calcineurin. Without calcineurin, the T-cell cannot activate genes (like IL-2) needed to attack the transplant.
2. Mycophenolate is converted to mycophenolic acid, which blocks the enzyme that makes guanosine, a DNA building block. Lymphocytes depend heavily on this pathway, so they cannot multiply.
3. Prednisone is converted to prednisolone and binds glucocorticoid receptors, turning off genes for cytokines (inflammatory signals) and immune-cell movement.
4. Together these three drugs keep the immune system quiet enough not to reject the organ—but also quiet enough to let infections take hold.

SAFETY WATCH

Tacrolimus: kidney damage (nephrotoxicity), high blood sugar (new-onset diabetes), tremor, high blood pressure, and raised infection risk. Requires regular blood levels (trough monitoring) and dose adjustment. · Mycophenolate: diarrhea, nausea, low white blood cell count (leukopenia), and higher risk of infection and some cancers (lymphoma). · Prednisone: weight gain, high blood sugar, bone thinning (osteoporosis), mood changes, high blood pressure, and increased infection risk, especially fungal and viral. · All three together profoundly raise the risk of opportunistic infections (CMV, PJP, fungi, BK virus, tuberculosis reactivation) and certain cancers (post-transplant lymphoproliferative disorder, skin cancer). Preventive (Prophylactic) antimicrobials (TMP-SMX, valganciclovir, azoles) are given precisely because these drugs make infection so likely.

Red flags

- Sudden drop in blood pressure or fast heart rate (signs the infection may be in the bloodstream, called sepsis, which can quickly damage organs)
- Confusion, extreme sleepiness, or trouble waking up (the brain isn't getting enough oxygen or is being poisoned by infection toxins)
- Trouble breathing, very fast breathing, or oxygen levels dropping below 90% (the lungs or blood can't deliver enough oxygen to keep the body alive)
- Lab results show the transplanted organ is failing fast (like creatinine jumping in a kidney transplant or liver enzymes spiking in a liver transplant)
- Signs of rejection happening at the same time as infection (fever plus biopsy changes), because the two can look alike and treating one wrong can make the other worse

Nursing priorities

1. Obtain blood cultures, urine culture, and chest X-ray immediately when fever reaches 100.4°F (38°C) or higher, before giving any antibiotics — Cultures identify the exact germ causing infection so doctors can switch from broad antibiotics to targeted treatment; drawing cultures after antibiotics start often gives false-negative results and leaves the team guessing
2. Start broad-spectrum IV antibiotics within one hour of fever detection as ordered, covering gram-positive bacteria like Staph, gram-negative bacteria like E. coli and Pseudomonas, and atypical bacteria — Transplant patients cannot contain infections while waiting for culture results; sepsis (life-threatening bloodstream infection) develops in hours because their white blood cells are suppressed and cannot fight back
3. Measure and document vital signs every 2 hours, watching specifically for rising heart rate above 100, dropping blood pressure below 90/60, or oxygen saturation below 92 percent — These vital sign changes signal septic shock (when infection causes dangerously low blood pressure) developing; early recognition allows IV fluid resuscitation and ICU transfer before organs shut down from poor blood flow
4. Check immunosuppression drug levels (tacrolimus trough or cyclosporine trough) and hold the next dose if levels are supratherapeutic until the transplant team gives instructions — High immunosuppression drug levels worsen infection by further weakening white blood cells; temporary dose reduction helps the immune system fight while maintaining enough medicine to prevent rejection
5. Enforce strict hand hygiene, mask use by anyone with respiratory symptoms, and no fresh flowers or plants in the patient room — Transplant patients catch infections from normal environmental germs and sick visitors; hand washing removes 99 percent of bacteria, masks stop respiratory droplets, and soil in plants contains Aspergillus fungus that causes deadly lung infection
6. Monitor urine output hourly if the patient has a kidney transplant, and report output dropping below 30 mL per hour immediately — Decreasing urine means the transplanted kidney is failing from infection, rejection, or sepsis reducing blood flow; catching this early allows interventions like IV fluids or biopsy before permanent kidney damage occurs
7. Administer prevention (prophylaxis) medications (TMP-SMX and valganciclovir) on schedule even during acute illness unless the doctor specifically orders them held — Stopping prevention (prophylaxis) during infection treatment leaves patients vulnerable to additional opportunistic infections on top of the current infection; continuing protection prevents double or triple infections that overwhelm the weakened immune system

8. Assess and document pain level, mental status changes like confusion or drowsiness, and any new skin lesions or rashes every 4 hours — New confusion signals brain infection or sepsis affecting the brain; skin lesions show disseminated fungal or viral infection spreading through blood; recognizing these patterns early changes treatment to add antifungal or antiviral medicines

Sources

- American Society of Transplantation (AST) Infectious Diseases Guidelines
- CDC guidelines on transplant-associated infections
- Infectious Diseases Society of America (IDSA)
- American Journal of Transplantation consensus statements

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