

Transplant infection

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.

THE ONE-LINE TRUTH

Transplant drugs that block rejection also block the body's germ fighters, so infections come in waves based on time since surgery.

The chain — how it unfolds

1. Transplant surgery and induction immunosuppression (monoclonal antibodies, high-dose steroids) wipe out most T-cells and slow neutrophils and macrophages right away. — why: 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. — why: 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. — why: 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). — why: 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. — why: 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, PTLN, or disseminated fungal infection. — why: 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. — why: 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. — why: 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.

Spot it — what you see, 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.

Red flags — act now

- 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

Treat it — in this order

1. 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.
2. 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%.
3. 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.

4. 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%.
5. 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.

Your top priorities at the bedside

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 — why: 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 — why: 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 — why: 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