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A REVIEW ARTICLE

A Review of Opportunistic Infections in Modern Immunosuppressed Populations

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Abstract

Opportunistic infections are a major clinical concern in individuals with impaired immune systems. More widespread organ transplantation, chemotherapy, biologic therapies, and long-term corticosteroids have created a population at greater risk of such infections. Consequently, a variety of patients, including not only those with late-stage HIV infection, have been affected by opportunistic pathogens.The type and degree of immune deficiency affect the range of potential infections. Neutropenia, for example, is known to come with severe bacterial and invasive fungal infection; defects in T-cell immunity lead to increased susceptibility to viral reactivation and infections by intracellular pathogens. Early recognition and application of appropriate diagnostic strategies are key to maximizing patient outcomes.New diagnostic tools, including molecular tests and metagenomic sequencing, has enhanced pathogen diagnosis in the immunocompromised. Successful management requires early initiation of antimicrobial therapy, targeted adjustment of immunosuppression, and supportive clinical care. Other critical preventive strategies include vaccination, antimicrobial prophylactic measures, screening for latent infections and establishing infection-control practices.This review describes the common pathogens, risk factors, diagnostic approaches and preventive measures relevant to opportunistic infections in modern immunocompromised populations.

1. Introduction

Although modern immunosuppressed populations (i.e. those with HIV, transplant recipients, patients with hematologic malignancy/stem cell transplants, those receiving corticosteroids or biologic therapies, and critically drugs) have the benefit of antiretroviral therapy in cases of HIV infection leading to reliance on opportunistic infections (OIs) as a major cause of morbidity and mortality. Infection risk is determined by the overall state of immunosuppression, which reflects the severity of immune impairment, epidemiologic exposures, and mucosal integrity in addition to any indwelling devices and previous antimicrobial use [1–3].

Risk is dynamic in solid organ transplantation, including acquisition of community pathogens, healthcare- associated bacteria, involved viruses such as *Cytomegalovirus* and invasive environmental mold under extensive immunosuppression [1,2]. Colonization by multidrug-resistant pathogens is common and can precede invasive disease; therefore, there is a need for surveillance and stewardship [4].

Novel diagnostics, such as multiplex PCR, fungal biomarkers, viral load testing and metagenomic sequencing, are improving the detection of pathogens but require judicious interpretation [5–8]. This review highlights epidemiology, pathogen spectrum, risk factors, diagnostics, prevention, and management of OIs in contemporary immunosuppressed populations.

2. Definition of Opportunistic Infections

Infections that are more frequent, more severe or atypically disseminated in hosts with compromised immune defenses are termed opportunistic infections (OIs). Its defining feature is the interaction of host immune deficit (e.g., neutropenia, T-cell dysfunction, impaired humoral immunity, with barrier disruption), and the microbial capacity for invasion, latency, or immune evasion [3]. This principle may account for how the same microorganism can occupy a niche as a commensal flora in an immunocompetent host but emerge as a potentially fatal pathogen in individuals undergoing

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therapy with lymphocyte depletion, high-dose or long-course corticosteroids, or transplantation immunosuppression [1–3].

Examples of fungal opportunists include invasive *Candida*, which has an incidence that rises with the length of device exposure and antimicrobial selection pressure, along with a host of healthcare-associated opportunistic organisms (e.g., MDR Gram-negative bacilli [1], *Candida* bloodstream infection [2], invasive molds). Reactivation syndromes (e.g. CMV disease, TB reactivation, Toxoplasmic encephalitis) can also be considered as opportunistic infections whose incidence rises from device exposure and prior antibiotic selection pressure.

3. Importance of Studying Immunosuppressed Populations

It is critical to investigate immunosuppressed populations, as management of these patients in the clinic has been increasingly complicated. First, immunosuppression is no longer limited to classic categories; with the advent of biologics and targeted therapies, we are seeing a broadening of our patients overall who have selective immune deficiencies and atypical infection patterns. Second, mixed infections and clinical deterioration over a short time frame are common, meaning that missed diagnosis is particularly costly [5–7]. Third, the opportunities for prevention are considerable: vaccination, treatment of latent infection (especially latent TB), and prophylaxis (e.g., *Pneumocystis* prophylaxis among high-risk patients) can prevent severe sequelae when implemented through risk-based frameworks [8–12].

Finally, antimicrobial resistance disproportionately affects patients who are immunocompromised, resulting in prolonged hospitalization, toxic combination therapy, and mortality [4]. A better mechanistic understanding of risk enables clinicians to provide more targeted prophylaxis, tailored diagnostics, and early therapy to patients—decreasing preventable morbidity and burden upon the healthcare system.

4. Normal Immune Defense Mechanisms

In normal individuals, the immune system provides protection against infection by various means: physical and mucosal barriers, innate immune responses mediated by neutrophils and macrophages, and adaptive immunity by T lymphocytes and B lymphocytes. Inhibition of these protective systems leads to greater vulnerability to opportunistic pathogens. In an unperturbed host, the immune system is non-self-responsive and comprises multiple lines of defense, including physical barriers like the skin and mucosal surfaces, innate humoral and cellular responses triggered by neutrophils and macrophages, as well as adaptive responses through T lymphocytes and B lymphocytes. Interference with these defense systems predisposes the person to opportunistic pathogens.

5. Immune Defects and Infection Susceptibility

Modern immunosuppression can be seen as quantitative (to what degree immune cell numbers decline) and qualitative (which immune pathways are dysfunctional). In practice, the following immune patterns can be clinically helpful for predicting OIs:

The relationship between the type of immune dysfunction and the anticipated spectrum of opportunistic pathogens is clinically relevant, as it directs risk stratification, diagnostic prioritization, and preventive strategies Table 1. [1-4,10,12-14].

Table note: Risk of opportunistic infection is an interplay between the type, severity, and duration of immune dysfunction in addition to concomitant therapies used, integrity of barriers, prior exposure to antimicrobials, as well as healthcare or environmental exposures.

The immune-defect stratification facilitates genomic selection of diagnostic tests (e.g., BAL for PJP/mold disease; CSF evaluation for cryptococcosis or PML) and informs prevention (vaccines, prophylaxis, latent infection screening) [9–12,15].

Table 1. Examples of major immune defects and their associated opportunistic infections in immunocompromised populations

Immune defect	Main associated opportunistic infections / pathogens
Neutropenia / impaired phagocyte function	Severe bacterial infections, candidemia, invasive aspergillosis, and other filamentous fungal infections
T-cell dysfunction / lymphopenia	CMV, HSV, VZV, *Pneumocystis jirovecii*, mycobacterial infections, *Cryptococcus neoformans*, and *Toxoplasma gondii*
Humoral deficiency / splenic dysfunction	Encapsulated bacterial infections, especially *Streptococcus pneumoniae*
Barrier disruption	Bloodstream infections, invasive candidiasis, catheter-related infections, and multidrug-resistant bacterial infections
Iatrogenic immunosuppression	Broad susceptibility to bacterial, viral, fungal, and parasitic opportunistic infections, including reactivation syndromes
Critical illness–associated immune dysregulation	Healthcare-associated pneumonia, bloodstream infections, multidrug-resistant Gram-negative infections, and selected invasive fungal infections

6. Causes of Immunosuppression

The combination of pre-existing diseases, therapeutic agents and other factors leads to immunosuppression.

• **Intrinsic disease-related immune dysfunction**: HIV, hematologic malignancy, advanced organ failure, and systemic inflammatory states with immune paralysis in critical illness [3,4].

• **Medication-related immunosuppression:** Cytotoxic chemotherapy, prolonged corticosteroids; calcineurin inhibitors and antiproliferatives in transplantation, anti-CD20 therapies and other immune-modulating agents [1–3]

• **Metabolic and chronic disease contributors:** Diabetes and malnutrition disturb immune function and barrier integrity, raising risk for severe infection as well as certain opportunistic fungal syndromes (e.g., the rising risk of mucormycosis in vulnerable metabolic contexts) [15].

• **Healthcare exposure and antimicrobial pressure:** Multiple hospitalization, devices, and broad-spectrum antibiotic exposure favor colonization/infection with MDR organisms, which poses a challenge for OI management [4].

In low- and middle-income settings, delayed diagnosis, limited access to prophylaxis for at-risk groups, and constrained laboratory diagnostics can accentuate OIs burden and delay initiation of effective therapy [9–11].

7. Types of Immunosuppressed Populations

Modern immunosuppressed populations are groups of patients who today develop clinically significant immune dysfunction due to modern-day diseases and, more importantly, modern medical therapies. In contrast to earlier frameworks, which were mainly tailored toward advanced HIV, the contemporary concept includes both solid organ transplantation recipients as well as patients with hematologic malignancy or those having endured healthcare-associated infections following HSCT (hemopoietic stem cell transplantation), prolonged courses of corticosteroids or biologic/targeted immunomodulators, and critically ill patients with immune dysregulation [1–4]. Thus, the spectrum of opportunistic infections has broadened and moved towards healthcare-associated pathogens, viral reactivation syndromes, and invasive fungal disease outside of HIV settings. [4,12,16].

8. Infection Prevention and Control in Immunocompromised Care Settings

Infection Prevention and Control (IPC) is essential in the care of immunocompromised patients, because exposure to healthcare workers or visitors may quickly result in infection. Multidrug-resistant (MDR) colonization is common in ICUs and in oncology/transplant units, potentially evolving to invasive disease, so core measures have a high impact (hand hygiene, catheter bundles, environmental cleaning; isolation; antimicrobial stewardship) [4].

9. Common Opportunistic Infections

9.1. Bacterial Infections

9.1.1. Gram-negative bacilli with multiple drug resistance and hospital-associated bacteria

In patients with impaired immune defenses, severe bacterial infections are associated with exposure to healthcare and past use of antibiotics. The importance of MDR Enterobacterales lies in the fact that colonization may lead to invasive infection in the setting of neutropenia, mucositis, or during device use [4]. Fever may serve as the sole warning sign of bacteremia for patients with neutropenia where delayed empiric antibiotics delay mortality [12].

9.1.2. Mycobacterial disease (TB and MAC)

Tuberculosis is a significant opportunistic threat, especially in cases of impaired cellular immunity. Prevention relies on diagnosing and treating latent TB infection (LTBI). CDC updated recommendations stresses that LTBI treatment prevents progression to active TB and recommends short-course rifamycin-based regimens preferentially in many settings for overall effectiveness (and improvement of completion rates) [17–18].

For example, disseminated *Mycobacterium avium* complex (MAC) has been a classic OI in the setting of advanced HIV, and NIH guidance covers MAC [10]; management is part of ART status and immune recovery.

9.1.3. Nocardia

Nocardia species cause pulmonary disease with increased risk of CNS dissemination in transplant recipients, patients with hematologic malignancies and chronic steroid users. Recognition of disease may be delayed since radiology can resemble fungal disease or malignancy and microbiologic identification may require prolonged incubation and specific methods [19].

9.2. Viral Infections

9.2.1. *Cytomegalovirus* (CMV)

Cytomegalovirus (CMV) is the most important opportunistic virus in organ transplant recipients and people with advanced HIV. The recent international consensus guidance (2025) suggested a risk-based approach that integrates specific diagnostic definitions and either "universal prophylaxis" or preemptive therapy guided by routine viral load monitoring, with a focus on resistant or refractory CMV infection [16]. Clinically CMV is of importance not only because of its tissue-invasive disease but also due to its ability to modify host immune function, enhancing the risk for secondary infections and promoting poorer graft outcome [16,17].

9.2.2. Herpes Simplex Virus (HSV) & Varicella-Zoster Virus (VZV)

Immunocompromised patients can experience more aggressive, chronic and disseminated diseases caused by HSV or VZV. NIH has issued preventive and therapeutic guidelines for severe mucocutaneous disease, esophagitis and complicated VZV presentations in advanced HIV and other contexts of immunosuppression [20-21].

9.2.3. JC polyomavirus

Immune reconstitution inflammatory syndrome (IRIS) related to the initial recovery of neuro-immunocompetence may also occur, while progressive multifocal leukoencephalopathy (PML), a CNS opportunistic disease due to JC virus reactivation in cases of fulminant immunosuppression, represents one of the most serious adverse events associated with treatment and remains devastating. NIH guidance emphasizes immune restoration as the primary therapeutic approach demonstrated to date, underscoring the limited role of direct antiviral therapy in many situations [22].

9.3. Fungal Infections

9.3.1. *Candida* (mucosal and invasive disease)

Candida causes mucocutaneous disease (most notably oral/esophageal candidiasis in advanced HIV) and invasive disease/candidemia found in hospitalized and neutropenic patients. The guidelines for candidiasis published by the Infectious Diseases Society of America (IDSA) address

source control (catheter removal if appropriate), antifungal choice, and the need to personalize treatment duration according to syndrome and host characteristics. [23].

9.3.2. *Aspergillus* (invasive aspergillosis)

Invasive aspergillosis is closely linked with prolonged neutropenia, hematopoietic stem cell transplantation, graft-versus-host disease, and high-dose corticosteroids. The Infectious Diseases Society of America suggests that imaging is performed in conjunction with microbiologic testing and biomarkers (e.g., galactomannan) and mold-active therapy is initiated early when suspicion is elevated as a delay to treatment increases mortality. [24].

9.3.3. Pneumocystis jirovecii pneumonia (PJP/PCP)

Pneumocystis jirovecii pneumonia (PJP) has long been recognized as a key opportunistic fungal infection in patients with HIV and non-HIV- related immunosuppression [1]. Non-HIV cases tend to result in more rapid progression with a lower burden of organisms, thus also making microscopy less sensitive and reliance on PCR and biomarkers more dominant [9] Using HIV guidance should be reasonably robust; however, we train on data all the way through October 2023. Recent evidence supports the risk-based extension of prophylaxis [25] and the use of a non-HIV–specific diagnostic algorithms for expediting early detection [26].

9.3.4. Cryptococcus and endemic mycoses

Cryptococcal meningitis remains a serious opportunistic infection in advanced HIV, where treatment must be stepped and the timing of antiretroviral therapy judiciously planned to limit immune reconstitution inflammatory syndrome [27]. Additionally, endemic fungi like *Histoplasma* and Coccidioides can cause disseminated disease in incompetent T-cell patients and the approach to management should be per appropriate NIH specific guidance for that relevant disease. [28-29].

9.3.5. Mucormycosis

Mucormycosis is less common overall but may be more rapidly progressive and life-threatening, particularly in profound immunosuppression and specific metabolic situations. International guidelines highlight the importance of early recognition, always surgical debridement when possible and rapid antifungal therapy (usually liposomal amphotericin B-based regimens) [15].

9.4. Parasitic Infections

9.4.1. Toxoplasma gondii

Toxoplasma encephalitis is a classical CNS OI associated with advanced HIV, commonly presenting with focal neurologic deficits and brain lesions. Clear prevention (prophylaxis in persons at risk) and treatment/maintenance strategies are provided by NIH guidance ([30] updated 2025).

9.4.2. Cryptosporidium and Microsporidia

Such organisms are major pathogens of chronic, watery diarrhea, dehydration, and wasting among immunosuppressed individuals. Guidelines issued by the National Institutes of Health focus on diagnostic modalities and underscore the critical role of immune recovery in long-lasting control of such infections [31,32].

9.4.3. Strongyloides stercoralis

Strongyloides is particularly important because chronic infection can persist silently for decades and then convert into hyperinfection syndrome or disseminated disease after corticosteroids or other immunosuppressive therapy. CDC clinical guidance emphasizes that *Strongyloides* infection may range from subclinical to fatal and highlights severe illness in patients using high-dose corticosteroids [33]. CDC treatment guidance further notes that in severe cases, immunosuppressive therapy should be reduced or stopped, if possible, alongside antiparasitic therapy such as ivermectin [34].

Different organ systems in the host can be infected by opportunistic pathogens depending on their immune status. Common opportunistic pathogens associated with their target organs are indicated in Figure 1.

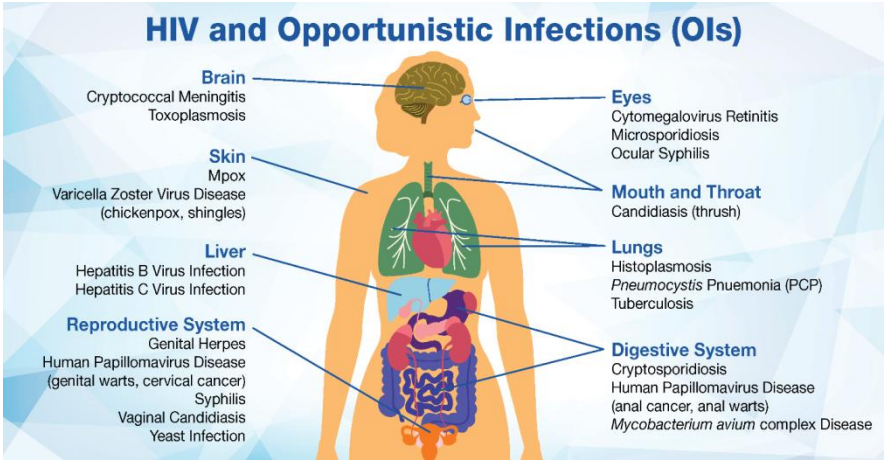

Figure 1. Opportunistic pathogens and the organ systems typically impact immunocompromised hosts.[36]

10. Risk Factors for Opportunistic Infections:

Risk factors include healthcare exposures, environmental/geographic exposures, and socioeconomic/health-system barriers, which together increase acquisition and progression of opportunistic pathogens [4,35].

They may promote transition from colonization to invasive infection, especially in hospitalized high-risk patients [4,27].

11. Diagnosis and Management

11.1.Diagnostic Methods

In the immunocompromised patient, a pragmatic diagnostic strategy integrates risk- based assessment with syndrome-directed evaluation and selective laboratory testing. The gold-standard diagnostic test for most pathogens is microbiological culture; tissue pathology should be done if invasive disease is suspected, and we also have rapid molecular assays in the forms of antigen detection tests and PCR panels. Quantitative viral load monitoring is particularly important for cytomegalovirus management in transplant recipients [16].

For severe or progressive pneumonia, early use of lower respiratory tract specimens—most commonly bronchoalveolar lavage—can significantly improve diagnostic yield when noninvasive testing is inconclusive [5]. Metagenomic next-generation sequencing (mNGS) of bronchoalveolar lavage fluid may further enhance pathogen detection in selected immunocompromised patients, although results should be interpreted cautiously and used as an adjunct to conventional microbiology [5–7].

11.2.Treatment Strategies

Treatment in immunocompromised patients should start promptly when clinical risk is high, since delays in severe presentations (such as neutropenic sepsis, invasive mold disease, severe *Pneumocystis jirovecii* pneumonia, or *Cytomegalovirus* end-organ disease) are associated with worse outcomes [12,16,28]. Once microbiologic data become available, therapy should be narrowed whenever possible to reduce toxicity, limit selection of resistance, and minimize drug–drug interactions [4,16,27]. Attention to interactions and adverse effects is essential, particularly with azole antifungals, calcineurin inhibitors, rifamycin-based regimens, ganciclovir-related marrow suppression, and amphotericin-associated nephrotoxicity [15,16,28].

Because azole antifungals are potent inhibitors of the cytochrome P450 (CYP3A4) enzyme system, clinically significant drug–drug interactions may result when they are administered together with immunosuppressive agents such as calcineurin inhibitors. Selected clinically significant interactions are summarized in Table 2[37].

Table 2. Important interaction of azole antifungals and immunosuppressants

Azole antifungal	Immunosuppressant	Interaction effect	Clinical risk
Voriconazole	Tacrolimus	↑tacrolimus concentration	Nephrotoxicity / neurotoxicity
Voriconazole	Cyclosporine	Increases cyclosporine concentration	Renal toxicity
Posaconazole	Tacrolimus	Increase in tacrolimus concentration	Drug toxicity
Fluconazole	Cyclosporine	↑cyclosporine concentration	Renal toxicity

Such interactions are of clinical significance as greater systemic exposure to these immunosuppressants can lead to severe adverse events including nephrotoxicity/neurotoxicity. Consequently, when azole antifungals are contraindicated along with immunosuppressive therapy, dosage adjustment and therapeutic drug monitoring should be considered.

Finally, optimal management also includes host-directed measures, such as immune restoration with antiretroviral therapy in HIV and cautious adjustment of immunosuppression when feasible, alongside supportive care for organ dysfunction [9,16].

12. Preventive Measures:

Reducing opportunistic infections in immunocompromised patients relies on prevention. Vaccination should be guided by recommendations for changed immunocompetence including appropriate timing and type (i.e., avoiding live vaccines when indicated) [34]. Strategic chemoprophylaxis and surveillance—like *Pneumocystis* prophylaxis, *cytomegalovirus* prevention strategies in transplantation, and antifungal prophylaxis in particular high-risk settings—are required [16,28–30]. Screening for latent tuberculosis infection (LTBI) and selected latent parasitic infections, especially *Strongyloides* in at-risk individuals, prior to immunosuppression is proposed as a means of lowering reactivation-associated morbidity [17,18,32,33,35]; infection control and antimicrobial stewardship are intended to minimize acquisition of multidrug-resistant pathogens and invasive disease [4].

13. Conclusion

Opportunistic infections are still a major cause of morbidity and mortality in modern-day immunocompromised people, not only among advanced HIV–infected patients but also those receiving transplants, patients with hematologic malignancies and stem cell transplant recipients, patients on steroids or biologic therapies, and critically ill individuals. The broadening spectrum of pathogens—from bacteria and fungi to viruses and parasites—calls for an organized, microbiology-driven strategy that merges immune-defect profiles with syndrome-based diagnostics and early treatment. Prevention remains at the forefront, complemented by vaccination and other forms of prophylaxis, management of latent infection, infection control, and antimicrobial stewardship. Improving outcomes in this vulnerable population will require intensified access to high-yield diagnostics and a stepwise approach with sustained risk-based preventive efforts.

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مراجعة للعدوى الانتهازية لدى الأفراد ذوي المناعة الضعيفة

الخلاصة

تُعدّ العدوى الانتهازية مصدر قلق سريري كبير لدى الأفراد الذين يعانون من ضعف في جهاز المناعة. وقد أدّى الانتشار الواسع لعمليات زراعة الأعضاء، والعلاج الكيميائي، والعلاجات البيولوجية، واستخدام الكورتيكوستيرويدات لفترات طويلة، إلى زيادة خطر الإصابة بهذه العدوى لدى هذه الفئة من السكان. ونتيجةً لذلك، تأثرت فئات متنوعة من المرضى، بمن فيهم المصابون بفيروس نقص المناعة البشرية في مراحله المتأخرة، بمسببات الأمراض الانتهازية. ويؤثر نوع ودرجة نقص المناعة على نطاق العدوى المحتملة. فعلى سبيل المثال، من المعروف أن قلة العدلات تترافق مع عدوى بكتيرية حادة وعدوى فطرية غازية؛ كما أن عيوب المناعة الخلوية التائية تؤدي إلى زيادة القابلية لإعادة تنشيط الفيروسات والإصابة بمسببات الأمراض داخل الخلايا. ويُعدّ التشخيص المبكر وتطبيق استراتيجيات التشخيص المناسبة أمرًا أساسيًا لتحقيق أفضل النتائج للمرضى. وقد ساهمت أدوات التشخيص الحديثة، بما في ذلك الاختبارات الجزيئية وتسلسل الميتاجينوم، في تحسين تشخيص مسببات الأمراض لدى الأفراد ذوي المناعة الضعيفة. تتطلب الإدارة الناجحة البدء المبكر للعلاج بالمضادات الحيوية، والتعديل الدقيق لجرعات كبت المناعة، والرعاية السريرية الداعمة. تشمل الاستراتيجيات الوقائية الهامة الأخرى التطعيم، والتدابير الوقائية بالمضادات الحيوية، وفحص العدوى الكامنة، وتطبيق ممارسات مكافحة العدوى. تستعرض هذه الدراسة مسببات الأمراض الشائعة، وعوامل الخطر، والأساليب التشخيصية، والتدابير الوقائية المتعلقة بالعدوى الانتهازية لدى المرضى الذين يعانون من نقص المناعة في العصر الحديث.

الكلمات المفتاحية: العدوى الانتهازية؛ كبت المناعة؛ المرضى الذين يعانون من نقص المناعة