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Infective Endocarditis in Patients Receiving Immunosuppressive Therapy: A Guide to Diagnosis and Care

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Abstract

Background: Infective endocarditis (IE) remains a life-threatening condition with high mortality rates. The rising prevalence of immunosuppressive therapies-including chemotherapy, biologics, and post-transplant regimens-has created a highly vulnerable population with distinct epidemiological and clinical profiles.

Objective: To provide a comprehensive guide on the predisposing mechanisms, microbiological spectrum, diagnostic challenges, and multidisciplinary management of IE in immunosuppressed patients.

Discussion: Immunosuppression alters the classic presentation of IE, often resulting in attenuated inflammatory markers and atypical clinical signs. Diagnosis requires a high index of suspicion and the early integration of advanced imaging, such as transesophageal echocardiography (TEE) and FDG PET-CT. Microbiologically, these patients show a higher incidence of Staphylococcus aureus, fungal pathogens (e.g., Candida), and multidrug-resistant organisms. Management involves complex antimicrobial stewardship, the careful titration of immunosuppressive drugs to balance infection control against graft rejection, and early surgical intervention in nearly 50% of cases.

Conclusion: Effective care for immunosuppressed patients with IE necessitates a personalized, multidisciplinary approach. Future research must focus on prospective trials and the validation of molecular diagnostics to optimize outcomes in this complex cohort.

Keywords: Infective Endocarditis; Cardiac Pathology; Immunosuppression; IE management; Streptococci

Abbreviations: IE: Infective endocarditis; TNF-α: Tumor necrosis factor-α; JAK: Janus kinase; CIED: Cardiac implantable electronic device; IgG: Immunoglobulin G; HIV/AIDS: Human immunodeficiency virus/Acquired immunodeficiency syndrome; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; sTREM-1: Soluble triggering receptor expressed on myeloid cells-1; TTE: Transthoracic echocardiography; TEE: Transesophageal echocardiography; CT: Computed tomography; FDG-PET/CT: Fluorodeoxyglucose positron emission tomography combined with CT; MRI: Magnetic resonance imaging; PCR: Polymerase chain reaction; CVC: Central venous catheter; mRNA: Messenger ribonucleic acid

Introduction

Infective endocarditis (IE) is a cardiac pathology that continues to be constantly updated for study due to its high global prevalence rate, reported at 5-14.3 per 100,000 adults/year, with a hospital mortality rate of up to 50% and a 5-year mortality rate

of 19-82% [1-3]. This is driven by the aging population and the use of medical devices. Although uncommon, it is nonetheless serious and even fatal. It can affect any organ and system. Previously, it was mostly attributed to viridin group streptococci, which have become less prevalent, with Staphylococcus aureus taking its place as the primary cause [4].

It should be noted that this epidemiological aspect has changed in recent years, as other patients are now included, such as younger individuals, drug users, pregnant women, hemodialysis patients, cancer patients, patients with rheumatic heart valve disease, complex acquired and congenital comorbidities, and those undergoing surgical and accessory procedures. Intracardiac infections, while not affecting older adults, present a diagnostic, prognostic, and therapeutic challenge due to their microbiological variability and atypical clinical presentation, requiring multidisciplinary care and potentially surgical intervention at some point in their course [4-6].

Immunosuppressed patients due to chronic degenerative diseases or immunosuppressive therapies (specifically antineoplastic agents, high-dose corticosteroids, or biological therapies for organ transplantation) have been added to the list of risk factors for infective endocarditis (IE). Pathophysiologically speaking, immunosuppression facilitates the adhesion of pathogens to the endocardium, resulting in vegetative lesions that will modify its clinical presentation and therapeutic response [5]. Patients immunosuppressed by chronic degenerative diseases or immunosuppressive therapies (specifically antineoplastic agents, high-dose corticosteroids, or biological treatments for organ transplantation) have been added to the list of risk factors for IE. Two contemporary facts regarding infective endocarditis are worth mentioning. First, the global population is increasingly elderly, which raises the likelihood of one or more comorbidities. Second, there has been a noticeable increase in the use of immunotherapies for chronic and oncological diseases. It is understood, then, that this epidemiological growth and change have been paralleled by a proportion of patients with altered immune susceptibility and, consequently, a higher risk of opportunistic infections such as infective endocarditis [5-7].

Therefore, current research and management of infective endocarditis require a comprehensive approach that bases its therapy not only on typical risks but also on a model of individualized medicine for each patient, considering their immune status, nosocomial determinants, interactions between their immunosuppressive therapies (if any), and the type of infectious pathogen. This review article addresses the emerging aspects, emphasizing the relationship with immunosuppressive states, predisposing mechanisms, and impact on contemporary epidemiology, as well as current needs for improvement in prevention, diagnosis, and treatment.

Immunosuppressive States and Mechanisms Predisposing to Infective Endocarditis

Solid Organ Transplant Recipients

Solid organ transplant recipients carry a substantially increased risk of infective endocarditis, largely attributable to chronic exposure to potent immunosuppressive regimens and frequent healthcare contact. Standard maintenance therapy typically combines calcineurin inhibitors such as tacrolimus or cyclosporine, antimetabolites including mycophenolate mofetil or azathioprine, and long-term corticosteroids [8]. These agents exert complementary immunosuppressive effects across both innate and adaptive immune pathways. Calcineurin inhibitors primarily impair T-cell activation and cytokine signaling, antimetabolites limit lymphocyte proliferation by disrupting nucleotide synthesis, and corticosteroids suppress macrophage and neutrophil function while blunting inflammatory responses [9,10].

Collectively, these mechanisms reduce the host's ability to effectively clear transient bacteremia, facilitating microbial persistence and adherence to the endocardium. Beyond pharmacologic immunosuppression, transplant recipients demonstrate broader immune dysfunction, including altered neutrophil activity and impaired humoral responses, which further compromise pathogen clearance [11]. This vulnerability is compounded by recurrent hospitalizations, invasive procedures, indwelling vascular devices, and cumulative healthcare exposure, all of which increase the frequency of bacteremic episodes and thereby heighten the risk of endocardial infection [12].

Hematologic Malignancies and Stem Cell Transplantation

Patients with hematologic malignancies and those undergoing hematopoietic stem cell transplantation experience profound immune compromise, placing them among the highest-risk populations for infective endocarditis. Neutropenia, whether disease-related or treatment-induced, significantly impairs early bacterial clearance from the bloodstream, allowing prolonged bacteremia and increasing the likelihood of valvular seeding [13,14]. The duration and severity of neutropenia correlate closely with infection risk.

Disruption of mucosal barriers further contributes to susceptibility. Chemotherapy-associated mucositis damages the integrity of the oral and gastrointestinal epithelium, promoting microbial translocation into circulation [15]. In parallel, the near-universal need for central venous access introduces an additional risk. Central venous catheters provide a portal of entry for microorganisms and a surface for biofilm formation, increasing the incidence of catheter-related bloodstream infections that may secondarily involve the cardiac valves [16,17]. Recurrent or persistent bacteremia related to vascular access remains a critical mechanism

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linking this population to increased rates of infective endocarditis.

Autoimmune and Inflammatory Diseases

The expanding use of targeted immunomodulatory therapies in autoimmune and inflammatory diseases has reshaped infection risk profiles, including susceptibility to infective endocarditis. Biologic therapies such as tumor necrosis factor-α inhibitors, interleukin pathway inhibitors, and Janus kinase inhibitors disrupt key cytokine signaling pathways essential for antimicrobial defense [18,19]. TNF-α inhibition interferes with macrophage activation and innate immune coordination and has been consistently associated with an increased incidence of serious bacterial infections [20]. JAK inhibitors exert broader immunosuppressive effects by interfering with multiple cytokines signaling cascades, which may further compromise immune surveillance and pathogen clearance [21].

Chronic corticosteroid therapy remains an independent and clinically significant contributor to infection risk. Prolonged glucocorticoid exposure impairs neutrophil function, suppresses lymphocyte activity, and diminishes antigen presentation, thereby weakening both innate and adaptive immune responses [19]. Importantly, infection risk appears dose-dependent, with higher cumulative steroid exposure conferring progressively greater vulnerability [20]. In many patients, the combination of biologics and corticosteroids produces additive immunosuppression, further increasing susceptibility to sustained bacteremia and potential endocardial involvement.

Oncologic Immunosuppression

Patients with malignancy are exposed to multifactorial immune impairment arising from the underlying disease, anticancer therapies, and repeated healthcare interactions. Cytotoxic chemotherapy frequently induces myelosuppression, leading to neutropenia, lymphopenia, and impaired humoral immunity, all of which reduce the capacity to contain bloodstream infections [20,21]. In addition, chemotherapy-related mucosal injury facilitates bacterial translocation from epithelial surfaces into the circulation, further increasing the risk of bacteremia [15]. These mechanisms together create a permissive environment for microbial persistence and potential cardiac involvement.

Immune checkpoint inhibitors introduce a more complex and evolving dimension of risk. Although these agents are designed to enhance antitumor immunity, they frequently precipitate immune-related adverse events that require treatment with high-dose corticosteroids or other immunosuppressive agents, thereby indirectly increasing infection susceptibility [19,20]. Emerging data also suggest that immune dysregulation associated with checkpoint inhibition may influence endothelial inflammation and cardiac immune homeostasis in ways that remain incompletely characterized [21]. As clinical experience with these agents grows, their broader implications for cardiovascular infections warrant continued investigation.

Epidemiology and Risk Factors

Incidence and Prevalence of IE in Immunosuppressed Patients

The epidemiology of infective endocarditis has undergone substantial transformation in recent decades, with immunosuppression emerging as a principal risk factor alongside prosthetic valve replacement, hemodialysis, venous catheters, and intravenous drug use [22]. While the overall incidence of community-acquired native-valve endocarditis in developed countries ranges from 1.7 to 6.2 cases per 100,000 person-years, specific populations receiving immunosuppressive therapy face considerably elevated risk [23]. Among solid organ transplant recipients, infective endocarditis occurs in approximately 1.8% of cases, with most episodes being healthcare-related or nosocomial in origin [24].

The median time from transplantation to endocarditis diagnosis is approximately 1,017 days, indicating that the risk persists well beyond the immediate post-transplant period [25]. Importantly, immunosuppression has been identified as an independent predictor of infective endocarditis development in patients with bacteremia, particularly in those with Enterococcus faecalis infection, where immunosuppression significantly increases the likelihood of endocardial involvement even after adjusting for other risk factors.

Patient-Specific Risk Factors

Patient-specific risk factors substantially amplify the risk of infective endocarditis in immunosuppressed individuals. Indwelling catheters represent a critical portal of entry for bloodstream infections and subsequent endocardial seeding. Colonized intravascular catheters account for one- to two-thirds of nosocomial endocarditis cases, with staphylococci being the predominant etiologic agents [26]. Among solid organ transplant recipients with infective endocarditis, 50% possess indwelling central venous catheters within the 30 days prior to diagnosis, compared to 27% of non-transplant patients with endocarditis [27].

The pathogenesis involves both direct endothelial damage from the foreign body and indirect effects through interference with normal valve function, creating sites of nonbacterial thrombotic endocarditis that serve as excellent nidi for bacterial colonization during episodes of bacteremia [28]. Prosthetic valves confer particularly high risk in immunosuppressed patients, with the incidence of prosthetic valve endocarditis ranging from 0.3 to 1.2 per 100 person-years and accounting for 20-30% of all infective endocarditis cases [29]. Patients with prosthetic valves have a 19-fold increased risk of infective endocarditis compared to matched controls, and when combined with immunosuppression, the risk is further magnified [26,30].

Cardiac implantable electronic devices (CIEDs) represent another major risk factor, with infection incidence increasing from

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1.45% in 2000 to 3.41% in 2012 [29]. The overall incidence of CIED infection ranges from 1 to 10 per 1,000 device-years, with complex devices such as implantable cardioverter-defibrillators carrying a higher risk (8-9 per 1,000 device-years) than pacemakers [22,30]. Prior valvular disease, including degenerative valvular disease, congenital abnormalities such as a bicuspid aortic valve, and rheumatic heart disease, creates structural abnormalities and turbulent blood flow that damages endothelium and promotes platelet-fibrin deposition, establishing the substrate for bacterial adherence and vegetation formation [31-35].

Therapy-Related Risk Factors

Therapy-related risk factors, particularly the intensity and duration of immunosuppression, play crucial roles in determining infective endocarditis risk. While specific data quantifying the relationship between immunosuppression intensity and endocarditis incidence remains limited, immunosuppressant therapy has been identified as an independent risk factor for endocarditis development in multivariate analyses [26]. Among solid organ transplant recipients, the spectrum of causative organisms differs markedly from the general population, with fungal infections predominating early (accounting for six of 10 cases within 30 days of transplantation) when immunosuppression is most intense, while bacterial infections cause most cases (80%) after this period [32]. The overall mortality rate in transplant recipients with endocarditis is 57%, with 58% of fatal cases not being suspected during life, underscoring the diagnostic challenges posed by immunosuppression [32].

Combination of immunosuppressive regimens, which are standard in solid organ transplantation and many autoimmune conditions, create a state of compromised immunity that increases susceptibility to both typical and atypical pathogens. In immunosuppressed patients, Staphylococcus aureus and fungal species (particularly Candida) are more prevalent than in the general population, with fungal endocarditis accounting for 1-2% of cases overall but representing a higher proportion in immunocompromised hosts [28,29]. The healthcare-associated nature of most endocarditis cases in immunosuppressed patients (70.5% in solid organ transplant recipients versus 36.3% in non-transplant patients) reflects both the frequency of healthcare contact and the cumulative burden of invasive procedures, indwelling devices, and nosocomial pathogen exposure that accompanies chronic immunosuppression [33,37-39].

Microbiological Spectrum and Pathophysiology

Typical pathogens

Staphylococcus aureus is a Gram-positive, beta-hemolytic bacterium that appears as cocci arranged in clusters. It is catalase-positive and coagulase-positive, characteristics that help distinguish it from other staphylococcal species. One of its most important virulence factors is Protein A, which binds to the Fc region of IgG antibodies, reducing effective immune recognition by interfering with complement activation and phagocytosis. S. aureus commonly colonizes the anterior nares and is also found on the skin, particularly in areas such as the ears, axillae, and groin. While it may exist harmlessly as part of the normal flora, it is frequently associated with inflammatory and invasive infections, including skin and soft tissue infections, pneumonia, septic arthritis, and bloodstream infections.

Importantly, S. aureus accounts for approximately 28% of all cases of infective endocarditis, highlighting its significant clinical impact [40]. Another staphylococcal species of major clinical relevance is Staphylococcus epidermidis. This organism is Gram-positive, catalase-positive, and coagulase-negative, and is a common and typically harmless resident of human skin. Despite its low intrinsic virulence, S. epidermidis has become an important cause of healthcare-associated infections. Its ability to produce adherent biofilms allows it to attach firmly to artificial surfaces, making it a frequent cause of infections involving prosthetic devices such as heart valves and intravascular catheters. These biofilms protect the bacteria from both host immune responses and antimicrobial therapy, contributing to persistent and difficult-to-treat infections [40].

Enterococci are Gram-positive bacteria that form part of the normal microbial community of the gastrointestinal tract. Under certain conditions, however, they can behave as opportunistic pathogens. Enterococci are commonly implicated in urinary tract infections, biliary tract infections, and infective endocarditis, particularly in hospitalized patients or those with underlying medical conditions. Their clinical importance has increased due to their ability to survive in harsh environments and their growing resistance to multiple antimicrobial agents [40]. The HACEK group comprising Haemophilus influenzae, Aggregatibacter species, Cardiobacterium species, Eikenella corrodens, and Kingella species-represents a collection of Gram-negative bacteria that are normal inhabitants of the human oropharyngeal and upper respiratory tract microbiota. Although taxonomically diverse, these organisms are grouped together because they share important clinical and microbiological characteristics, most notably their ability to cause infective endocarditis. Collectively, HACEK organisms are responsible for approximately 3% of all cases of infective endocarditis [41].

HACEK endocarditis typically affects patients with underlying structural heart disease, including those with damaged native valves or prosthetic cardiac valves, and is also more frequently observed in individuals with coronary artery disease. The disease is characteristically insidious in onset, often progressing slowly and remaining clinically silent for prolonged periods before diagnosis. The pathogenesis of HACEK endocarditis is believed to involve colonization of the oropharynx, followed by transient bacteremia. This bacteremia most commonly occurs in the setting of dental procedures, periodontal disease, or minor mucosal trauma, allowing organisms to gain access to the vascular system and subsequently adhere to susceptible cardiac valves. Several studies have

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reported that a significant proportion of affected patients had recent dental work or poor oral health prior to the development of infection [41]. HACEK organisms are generally considered to be of low virulence, preferentially infecting structurally abnormal or prosthetic valves rather than healthy cardiac tissue. Their fastidious and slow-growing nature contributes to diagnostic challenges, as they may require prolonged incubation periods and often demonstrate low or absent growth in routine blood cultures. Consequently, HACEK organisms should be considered in cases of culture-negative infective endocarditis [41].

Opportunistic and Atypical Pathogens

Candida albicans is a dimorphic fungus capable of existing in multiple morphological forms, including budding yeast and pseudo hyphae at lower temperatures (around 20 °C), and forming germ tubes at body temperature (37 °C). This ability to shift between forms plays an important role in its pathogenicity. C. albicans is an opportunistic organism that primarily causes disease in immunocompromised individuals, including neonates, patients receiving prolonged corticosteroid therapy, individuals with diabetes mellitus, and those with advanced HIV/AIDS. Clinically, Candida albicans is most associated with vulvovaginal candidiasis, but it can also cause mucocutaneous candidiasis, affecting the oral cavity, skin, and mucosal surfaces. In more severe cases, particularly in patients with neutropenia, the organism may invade the bloodstream and internal organs, leading to disseminated candidiasis, a serious condition associated with significant morbidity and mortality [42].

Aspergillus species are filamentous fungi characterized by septate hyphae that branch at acute angles, typically around 45 degrees, a feature that is helpful for microscopic identification. These organisms are catalase-positive and are important causes of disease in immuno-compromised individuals. Invasive aspergillosis most commonly occurs in patients with impaired neutrophil function, such as those with chronic granulomatous disease, as well as in individuals receiving chemotherapy or prolonged immunosuppressive therapy. In addition to invasive disease, Aspergillus can form aspergillomas, within preexisting lung cavities. These cavities are often the result of prior pulmonary conditions, most notably tuberculosis. Certain species of Aspergillus also produce aflatoxins, potent mycotoxins that have been strongly associated with the development of hepatocellular carcinoma, underscoring the broader public health relevance of these fungi beyond invasive infection [42].

Mycobacterial endocarditis is a rare clinical entity and occurs far less frequently than endocarditis caused by more common bacterial pathogens. Reported cases demonstrate a marked predominance of non-tuberculous mycobacteria over Mycobacterium tuberculosis as causative agents of infective endocarditis. Owing to their intrinsic resistance to many antimicrobial agents, mycobacteria are often difficult to eradicate, and infections are frequently refractory to standard antimicrobial therapy. As a result, mycobacterial endocarditis is associated with high morbidity and mortality. Among the non-tuberculous species, rapidly growing mycobacteria represents most reported isolates. Species such as Mycobacterium chelonae, Mycobacterium abscessus, and Mycobacterium fortuitum account for approximately 68% of identified cases, making them the predominant mycobacterial pathogens implicated in this condition. These organisms pose significant diagnostic and therapeutic challenges, further contributing to poor clinical outcomes [43].

Clinical Presentation and Diagnostic Challenges

Infective endocarditis in patients receiving immunosuppressive therapy presents a major diagnostic and clinical challenge because the host immune response is altered by medications such as corticosteroids, biologics, chemotherapy, calcineurin inhibitors, or post-transplant immunosuppression. These patients often have atypical presentations, muted inflammatory responses, and delayed or misleading laboratory findings, which can result in missed or late diagnoses [44,45]. Additionally, standard diagnostic tools such as the Modified Duke Criteria may be less reliable in this population, necessitating greater reliance on advanced imaging modalities and adjunctive biomarkers [48]. As a result, clinicians must maintain a high index of suspicion and adopt a more proactive and multimodal diagnostic approach when evaluating suspected IE in immunosuppressed individuals [46,47].

Atypical and Blunted Clinical Manifestations

In patients receiving immunosuppressive therapy, the classical clinical features of infective endocarditis are often attenuated or atypical, making early recognition difficult [45]. One of the most significant differences is the frequent absence of fever, which is traditionally considered a hallmark of Immunosuppressive medications, particularly corticosteroids and biologic agents targeting inflammatory pathways, that blunt the host's febrile response, leading to normothermia or only low-grade temperature elevations despite ongoing infection [44]. As a result, clinicians may underestimate the severity of illness or delay further investigation. Instead of classic signs such as fever, chills, and night sweats, these patients may present with nonspecific symptoms such as fatigue, malaise, anorexia, weight loss, or subtle neurological changes [45].

Similarly, peripheral stigmata of endocarditis, such as Osler nodes, Janewaylesions, or splinter hemorrhages, are less commonly observed in immunosuppressed patients due to altered immune and inflammatory responses [47]. Cardiac murmurs may be absent or preexisting, especially in patients with prior valvular disease, prosthetic valves, or structural heart abnormalities. Additionally, immunosuppressed patients are more likely to develop subacute or indolent infections caused by atypical organisms, including fungal pathogens, culture-negative bacteria, or opportunistic microbes, further complicating clinical recognition [44,45]. Overall, the blunted and atypical presentation necessitates a heightened clinical suspicion and a lower threshold for diagnostic evaluation in this population.

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Laboratory Findings

Traditional laboratory markers of inflammation, such as C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and leukocyte count, have significant limitations in immunosuppressed patients with infective endocarditis [45,48]. Because these individuals have impaired immune activation, inflammatory biomarkers may be only mildly elevated or even within normal ranges despite active infection. This can falsely reassure clinicians and delay diagnosis. Leukopenia, rather than leukocytosis, may be observed in patients receiving chemotherapy or certain immunosuppressive agents, further obscuring the typical inflammatory pattern associated with IE [44]. Blood cultures, which remain a cornerstone of diagnosis, may also be affected if patients are already receiving broad-spectrum antibiotics or antifungal therapy, leading to culture-negative endocarditis [45].

Procalcitonin has emerged as a potentially useful adjunctive biomarker in this setting, as it may better distinguish bacterial infection from noninfectious inflammatory conditions, even in immunosuppressed hosts [47]. Some studies suggest that elevated procalcitonin levels correlate with more severe infection and can aid in identifying IE when traditional markers are inconclusive. Novel biomarkers, including interleukin-6, soluble triggering receptor expressed on myeloid cells-1 (sTREM-1), and circulating cell-free DNA, are being investigated for their potential role in earlier detection of infection in immunocompromised patients [48]. However, these markers are not yet widely validated for routine clinical use in IE and should be interpreted in conjunction with clinical and imaging findings rather than as standalone diagnostic tools.

Imaging Modalities

Echocardiography remains the primary imaging modality for diagnosing infective endocarditis, but its interpretation in immunosuppressed patients can be challenging [44]. Transthoracic echocardiography (TTE) is often used as an initial screening tool due to its noninvasive nature, but it has limited sensitivity, particularly in detecting small vegetations, prosthetic valve infections, or early disease [44,45]. In immunosuppressed patients, where clinical suspicion may be lower and vegetations may be smaller or less inflammatory, TTE alone is frequently insufficient. Transesophageal echocardiography (TEE), on the other hand, provides superior visualization of cardiac structures and is significantly more sensitive for detecting vegetations, abscesses, and prosthetic valve involvement [44]. As a result, TEE should be strongly considered early in the diagnostic process when IE is suspected in immunosuppressed individuals.

Advanced imaging techniques, such as cardiac computed tomography (CT) and fluorodeoxyglucose positron emission tomography combined with CT (FDG-PET/CT), have become increasingly important in complex or equivocal cases [46]. Cardiac CT is particularly useful for evaluating perivalvular complications, such as abscesses, pseudoaneurysms, or fistulas, which may be difficult to detect on echocardiography [46]. FDG-PET/CT can help identify metabolic activity associated with infection, especially in prosthetic valve endocarditis or device-related infections, where echocardiographic findings may be inconclusive [46]. Additionally, whole-body imaging plays a crucial role in detecting extracardiac complications of IE, such as septic emboli to the brain, spleen, kidneys, or lungs. MRI of the brain may reveal silent embolic lesions, while CT or ultrasound can identify splenic abscesses or infarcts, which may otherwise go unnoticed in immunosuppressed patients with minimal symptoms [45,46].

Diagnostic Criteria Limitations

The Modified Duke Criteria, which are widely used to diagnose infective endocarditis, have important limitations in immunosuppressed patients [48]. These criteria rely heavily on major clinical features such as persistent fever, positive blood cultures with typical organisms, and echocardiographic evidence of vegetations. However, in immunosuppressed individuals, fever may be absent, blood cultures may be negative due to prior antimicrobial exposure, and vegetations may be small or difficult to visualize, leading to misclassification as “possible” rather than “definite” IE [45,48]. As a result, the sensitivity of the Duke Criteria is reduced in this population, increasing the risk of delayed or missed diagnosis.

To address these limitations, several adaptations and adjunctive tools have been proposed. Some experts recommend placing greater emphasis on imaging findings, particularly TEE, cardiac CT, and FDG-PET/CT, when evaluating immunosuppressed patients [46,48]. The inclusion of PET-CT findings as a major criterion in prosthetic valve or device-related infections has been suggested to improve diagnostic accuracy [46]. Additionally, incorporating novel biomarkers such as procalcitonin or molecular diagnostic techniques, including polymerase chain reaction (PCR)–based pathogen detection, may enhance sensitivity in culture-negative cases [47,48]. Ultimately, a multidisciplinary approach that integrates clinical judgment, advanced imaging, and laboratory adjuncts is essential for accurate diagnosis in immunosuppressed hosts.

Therapeutic Management

Antimicrobial Therapy

Empiric Therapy Considerations

Vancomycin or daptomycin in combination with an aminoglycoside and anti-pseudomonal beta-lactam, such as cefepime or piperacillin-tazobactam, must provide broad-spectrum coverage in immunosuppressed patients in order to account for an expanded spectrum of potential pathogens beyond typical IE organisms [45]. Important factors include the increased risk of nephrotoxicity when aminoglycosides and calcineurin inhibitors are combined, as well as possible drug interactions between antimicro-

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bials and immunosuppressive agents, especially azole antifungals and macrolides, which dramatically change calcineurin inhibitor levels through cytochrome P450 3A4 interactions [49].

Pathogen-Directed Therapy

While beta-lactam antibiotics have shown better results for methicillin-susceptible Staphylococcus aureus even in immunocompromised patients, enterococcal IE necessitates combination therapy with ampicillin plus an aminoglycoside or dual beta-lactam therapy to avoid nephrotoxicity concerns, which is especially crucial in transplant recipients. Once organisms have been identified, therapy should be narrowed while maintaining bactericidal activity. Gram-negative IE requires long-term treatment with anti-pseudomonal beta-lactams, frequently in combination with aminoglycosides or fluoroquinolones based on susceptibility testing. It is more common in immunocompromised individuals with catheters or healthcare exposures [45].

Prolonged and Combination Regimens

Immunocompetent recommendations for the duration of treatment for IE in immunosuppressed patients are usually 4-6 weeks; however, extensions to 6-8 weeks or longer may be necessary due to delayed source control, persistent bacteremia, or significant perivalvular involvement. Combination therapy has several uses, such as preventing resistance and killing staphylococci in prosthetic valve endocarditis through improved biofilm penetration. However, rifampin's strong hepatic enzyme induction necessitates careful monitoring of immunosuppressant dosages, with tacrolimus and cyclosporine frequently requiring three to five-fold increases [49].

Antifungal Therapy Challenges

Fungal endocarditis, which is primarily caused by Candida species, is much more common in immunosuppressed patients (5–10% of transplant recipient IE cases versus <2% in the general population). Medical therapy alone is rarely curative, and surgical valve replacement is necessary for the best results [52]. While Aspergillus endocarditis necessitates aggressive voriconazole-based therapy, which is frequently combined with an echinocandin and complicated by significant drug interactions with azole antifungals, reducing tacrolimus dose requirements by 70–80% in most patients and requiring intensive therapeutic drug monitoring, susceptible isolates require initial echinocandin therapy followed by fluconazole step-down for treatment [52].

Management of Immunosuppressive Therapy

Temporary Reduction or Discontinuation

To manage immunosuppression during active IE, it is necessary to weigh the potential advantages of increased immune responses against the risks of rejection. In transplant recipients, total discontinuation is rarely appropriate, and careful dose reduction with close monitoring is typically preferred [50]. While corticosteroids should be kept at physiological doses to prevent adrenal insufficiency, and calcineurin inhibitors are usually continued at therapeutic levels because they are still necessary for rejection prevention, antimetabolites like mycophenolate mofetil and azathioprine are usually reduced or temporarily stopped first because they contribute most significantly to infection risk and are less critical for rejection prevention in stable recipients [49].

Risk of Graft Rejection or Disease Flare

The risk of acute rejection after immunosuppression varies depending on the type of organ. Heart transplant recipients are at a particularly high risk (15–30% after significant reduction), kidney recipients are better able to tolerate temporary reduction, especially after a year post-transplant (5–10% rejection rates), and liver recipients are at an intermediate risk with relative immunologic privilege. Disease flare is the main concern for non-transplant patients receiving immunosuppression for autoimmune conditions. Patients with active diseases at IE diagnosis and those receiving biological agents are most at risk, so careful disease monitoring and coordination with rheumatology or other pertinent specialists are necessary [50].

Multidisciplinary Decision-Making

To manage immunosuppression as effectively as possible, infectious disease specialists, transplant doctors or rheumatologists, cardiologists, and cardiac surgeons must work together to make decisions that take into consideration the severity of the infection, the immunosuppressive regimen, the length of time since transplantation, rejection history, and personal risk factors [50]. The virulence of causative organism, the degree of cardiac involvement, the antimicrobial response, and complications like heart failure or embolic events are important factors. Ongoing management is guided by serial monitoring of graft function using suitable biomarkers and surveillance biopsies, when necessary, while keeping immunosuppressive trough levels at the lower end of therapeutic ranges frequently represents a reasonable compromise between infection control and rejection prevention [49].

Surgical Intervention

Indications for Early Surgery

About 40-50% of IE patients need surgery. The main reasons for this are heart failure brought on by severe valvular dysfunction (the strongest indication for urgent surgery), uncontrolled infection despite the use of the right antibiotics, and prevention of embolic events, especially when vegetations are larger than 10 mm on the anterior mitral leaflet [1]. Additional surgical indications include fungal endocarditis, which nearly always necessitates surgery, perivalvular abscess formation, infection with highly resistant organisms, persistent bacteremia after five to seven days of appropriate antibiotics, and prosthetic valve endocarditis, especially if it develops early after implantation or is brought on by staphylococci [1].

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Surgical Risk in Immunosuppressed Patients

Immunosuppressed patients face elevated perioperative risks with increased rates of postoperative infections, impaired wound healing, sternal dehiscence, and prolonged mechanical ventilation, with perioperative mortality rates of 15-25% in solid organ transplant recipients compared to 5-10% in the general IE population [51]. Contributing factors include immunosuppressive medication effects on wound healing through reduced collagen synthesis and impaired cellular migration, corticosteroid-associated sternal wound complications particularly at doses exceeding prednisone 20 mg daily, renal dysfunction common with calcineurin inhibitor use amplifying surgical risk through acute kidney injury susceptibility, and thrombocytopenia or coagulopathy complicating perioperative bleeding management, though withholding necessary surgery based solely on immunosuppression status remains inappropriate as untreated IE carries even higher mortality [51].

Outcomes of Valve Replacement or Repair

According to Otto et al. [51], the long-term results of valve surgery for IE in immunosuppressed patients show 5-year survival rates of 60-70% for transplant recipients and 75-80% for non-immunosuppressed patients, with recurrent endocarditis occurring in roughly 10-15% and 5-8% of cases, respectively [51]. When choosing between mechanical and bioprosthetic valves, one must carefully weigh the durability of mechanical valves, which require lifelong warfarin anticoagulation with possible immunosuppressive interactions and bleeding risk, against bioprosthetic valves, which do not require anticoagulation but may deteriorate more quickly. Current research indicates that there are no appreciable differences in infection rates, so patient age, bleeding risk, and preferences can guide the decision, while valve repair, when technically possible, offers benefits like native tissue preservation and may reduce the risk of recurrent infections [1].

Prevention and Prophylactic Strategies

Antibiotic Prophylaxis: Current Guidelines and Controversies

According to current American Heart Association guidelines, antibiotic prophylaxis before dental procedures is only advised for the highest-risk patients, such as those with prosthetic valves, prior IE, certain congenital heart diseases, and cardiac transplant recipients who develop valvulopathy. This recommendation is based on concerns about antimicrobial resistance, a very low absolute risk of IE after dental procedures (less than 1 in 10,000), and a higher cumulative risk of bacteremia from routine activities [54]. However, there is still debate about whether immunosuppressed patients can benefit from broader prophylaxis due to their three to five times higher incidence of IE. When appropriate, recommended regimens include amoxicillin 2 grams orally 30 to 60 minutes before the procedure, or ampicillin 2 grams intravenously/intramuscularly for those unable to take oral medications. Patients who are allergic to penicillin are given clindamycin 600 mg, azithromycin 500 mg, or cephalexin 2 grams [45].

Catheter and Device Management

With central venous catheter-related bloodstream infection rates of 2-5 per 1,000 catheter-days in transplant recipients, intravascular catheters and cardiac devices are significant IE risk factors. Preventive measures include strict insertion bundle adherence with maximal barrier precautions, chlorhexidine antisepsis, optimal site selection, and prompt removal when unnecessary. For long-term access, tunnelled catheters or implantable ports are preferred over non-tunneled CVCs [17]. Cardiac implantable electronic devices, which are becoming more common in immunocompromised patients, have a 1-4% infection rate that could result in device-related endocarditis. This calls for careful sterile technique, perioperative prophylaxis with cefazolin or vancomycin, and consideration of antibacterial envelopes in high-risk patients. When infection occurs, complete system removal is typically required for cure, and reimplantation is postponed until blood culture clearance and appropriate antibiotics are finished [45].

Infection Surveillance in High-Risk Populations

Early IE detection is made possible by systematic infection surveillance through structured protocols such as routine clinical evaluations, monitoring for unexplained fever or bacteremia, and pathogen screening based on epidemiological risk. Patient education regarding warning signs, such as persistent fever, chills, new murmur, unexplained fatigue, or embolic phenomena, is essential for timely medical evaluation [50]. Echocardiography strategies use liberal transthoracic echocardiography for unexplained fever or positive cultures, with transesophageal echocardiography when initial studies are non-diagnostic and clinical suspicion remains moderate to high. Blood culture protocols should adhere to standard IE diagnostic criteria, obtaining at least three sets from separate sites before antibiotics, when possible, with extended incubation beyond standard 5 days warranted for fastidious organisms and molecular diagnostics, such as PCR and next-generation sequencing [45].

Vaccination and Preventive Care

Transplant candidates should ideally receive all recommended vaccinations before transplantation for stronger immune responses. Post-transplant vaccination should avoid live-attenuated vaccines, with special attention paid to pneumococcal vaccination (PCV13 followed by PPSV23), annual influenza vaccination (possibly with high-dose or adjuvanted formulations), and COVID-19 mRNA vaccines with additional doses to enhance responses given significantly elevated severe disease risks [53]. While antimicrobial stewardship programs optimize antibiotic use to prevent resistance development and preserve the microbiome by avoiding unnecessary or prolonged courses, choosing narrow-spectrum agents based on culture data, and switching from intravenous to oral therapy when feasible, complementary preventive strategies include careful dental hygiene and routine dental care. Poor oral

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health increases bacteremia frequency from oral flora, elevating IE risk. Some experts recommend more frequent professional cleaning every three to four months in high-risk immunosuppressed patients [54].

Future Directions and Research Gaps

To address the existing knowledge gaps, future research must prioritize methodologically rigorous, multicenter prospective studies and randomized clinical trials specifically designed for the immunocompromised population. Key efforts should focus on defining distinct clinical phenotypes and long-term prognoses across various immunosuppressive states, such as solid organ transplantation, hematologic malignancies, and the use of targeted immunomodulatory therapies. Furthermore, there is a critical need to validate advanced diagnostic technologies, including FDG PET-CT and next-generation metagenomic sequencing, to establish their sensitivity and predictive value in detecting early-stage or culture-negative endocarditis in these high-risk individuals [1,3,5,10,45].

Additionally, the research schedule must optimize personalized antimicrobial management through therapeutic drug monitoring and the integration of artificial intelligence to combat increasing rates of multidrug resistance. Investigation into innovative prevention strategies, such as adjusted vaccination protocols and monoclonal antibody prophylaxis, remains essential given the limited evidence for immunoprophylaxis in this cohort [2,4]. Finally, establishing international collaborative registries that incorporate patient-centered outcomes, including health-related quality of life and long-term functional recovery-will be vital for evolving current guidelines into more comprehensive, evidence-based standards of care [1,45].

Conclusion

Infective endocarditis in the immunosuppressed patient represents one of the most complex intersections in modern medicine. The traditional “classic” signs of IE are frequently absent, replaced by an indolent clinical course that demands a proactive diagnostic strategy. As the population of patients living with altered immunity grows, clinicians must move beyond standard protocols and adopt individualized models of care. Success in these cases relies on the cardiology team’s approach synergy between infectious disease specialists, surgeons, and transplant or oncology teams. By integrating advanced metabolic imaging with precision antimicrobial therapy and timely surgical intervention, we can mitigate the historically high mortality rates seen in this vulnerable population.

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