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THE COURSE AND DIAGNOSIS OF PYODERMAS IN IMMUNOCOMPROMISED STATES: A FOCUS ON HIV INFECTION AND DIABETES MELLITUS

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ABSTRACT: This review analyzes the distinctive features of the clinical course and diagnosis of pyodermas in patients with significant immunocompromising conditions, namely Human Immunodeficiency Virus (HIV) infection and Diabetes Mellitus (DM). It explores the characteristic clinical presentations, predominant causative pathogens, and their patterns of antimicrobial resistance. The article highlights the challenges in differential diagnosis and proposes an optimized framework for managing these complex patients. The urgency of this topic is underscored by the growing global prevalence of immunocompromised individuals and the concurrent shift in the clinical presentation of dermatoses within this population, demanding advanced knowledge and a re-evaluation of traditional therapeutic approaches by dermatologists and infectious disease specialists.

Keywords: pyoderma, immunodeficiency, HIV infection, diabetes mellitus, *Staphylococcus aureus*, MRSA, atypical presentation, diagnosis, antibiotic resistance, skin infection.

INTRODUCTION

Pyodermas, a diverse group of bacterial skin infections primarily caused by *Staphylococcus aureus* and *Streptococcus pyogenes*, represent one of the most common reasons for dermatological consultation worldwide (Stevens et al., 2014). While typically presenting as self-limiting and superficial infections in immunocompetent individuals, their clinical behavior is dramatically altered in the presence of an underlying immunodeficiency.

Two of the most significant causes of acquired immunodeficiency globally are HIV infection and diabetes mellitus. The progressive depletion of CD4+ T-helper lymphocytes in HIV infection cripples the cell-mediated immune response, rendering the host highly susceptible to a wide range of opportunistic and bacterial pathogens (Kaplan et al., 2008). On the other hand, diabetes mellitus induces a complex state of immune dysregulation characterized by impaired neutrophil function (chemotaxis, phagocytosis, and bactericidal activity), microvascular and macrovascular complications, and peripheral neuropathy. This triad of pathology compromises the skin's barrier function and its ability to mount an effective inflammatory response, creating a fertile ground for severe infections (Peleg, Weerarathna, & McCarthy, 2007).

In these patient populations, pyodermas frequently deviate from their classic presentation. They are often characterized by an aggressive, extensive, recurrent, and treatment-refractory course. Clinicians are increasingly confronted with atypical morphologies, such as ulcerative, necrotic, or vegetative lesions, which can mimic other opportunistic infections or even malignancies, posing significant diagnostic challenges. Furthermore, the etiological landscape is complicated by the high prevalence of multidrug-resistant organisms,

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particularly Methicillin-resistant *Staphylococcus aureus* (MRSA), which renders empirical antibiotic therapy unreliable and increases patient morbidity and mortality (Dryden, 2010).

Given these complexities, there is a pressing need for a comprehensive understanding of the unique aspects of pyoderma in immunocompromised hosts. This article aims to systematically review and analyze the specific features of the clinical course and diagnosis of pyodermas in patients with HIV infection and diabetes mellitus to inform and optimize clinical management strategies.

MATERIALS AND METHODS

This study was conducted as a comprehensive narrative review of the existing scientific literature. A systematic search was performed using major electronic databases, including PubMed/MEDLINE, Scopus, Google Scholar, and the Cochrane Library, for articles published between January 2010 and December 2024.

The search strategy employed a combination of Medical Subject Headings (MeSH) terms and keywords, including: "pyoderma," "skin and soft tissue infections," "Staphylococcus aureus," "MRSA," "HIV infections," "AIDS," "diabetes mellitus," "immunocompromised host," "atypical presentation," "clinical course," and "diagnosis." Boolean operators (AND, OR) were used to refine the search results.

Inclusion criteria for articles were: Published in the English language. Original research (cohort studies, case-control studies, case series) and comprehensive review articles. Focus on the clinical, microbiological, or diagnostic aspects of pyodermas in adult patients with confirmed HIV infection or diabetes mellitus.

Exclusion criteria were: Case reports of a single patient (unless they highlighted a particularly novel or significant finding). Studies focused exclusively on pediatric populations. Articles where the full text was not accessible.

The initial search yielded over 800 articles. After screening titles and abstracts for relevance and applying the inclusion/exclusion criteria, 62 articles were selected for full-text review. Data extracted from these articles included information on the prevalence of different pyoderma forms, clinical characteristics, microbiological findings (including antibiotic resistance profiles), diagnostic challenges, and differential diagnoses.

The synthesized information forms the basis of the Results and Discussion sections of this review. The methodology did not involve primary data collection or statistical analysis of raw data, but rather a qualitative synthesis of published findings to construct a coherent overview of the topic.

RESULTS

The literature review revealed profound differences in the clinical presentation, microbial etiology, and diagnostic approach for pyodermas in immunocompromised patients compared to the general population.

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Clinical Course in HIV-Infected Patients - In patients with HIV, the severity and morphology of pyoderma are strongly correlated with the degree of immunosuppression, specifically the CD4+ lymphocyte count.

Prevalence and Forms: While superficial infections like impetigo and folliculitis can occur at any stage, deeper and more aggressive forms become common as the CD4+ count falls below 500 cells/μL. The most frequently reported manifestations include widespread, persistent furunculosis and carbuncles (Kaplan et al., 2008; Serrano-Atero et al., 2017).

Atypical Presentations: A hallmark of pyoderma in advanced HIV (CD4+ count < 200 cells/μL) is the appearance of atypical forms. Ecthyma gangrenosum-like lesions, deep, "punched-out" ulcers with necrotic bases, are common. Another significant presentation is botryomycosis, a chronic, suppurative, and granulomatous infection that forms large, vegetating nodules and sinus tracts, often mistaken for a deep fungal infection or a tumor (Schwartz & Kapila, 2021). Widespread papular or pustular eruptions caused by *S. aureus* are also frequently seen.

Complications: There is a high propensity for local complications such as abscess and cellulitis, as well as systemic dissemination leading to bacteremia and sepsis, particularly in patients not receiving effective antiretroviral therapy (ART).

Clinical Course in Patients with Diabetes Mellitus - In patients with DM, the clinical picture is dominated by deep-seated, rapidly progressing infections, heavily influenced by underlying vasculopathy and neuropathy.

Prevalence and Forms: Recurrent furuncles and carbuncles are extremely common, classically appearing on the nape of the neck, back, and axillae. However, the most critical manifestations involve the lower extremities. Diabetic foot infections (DFIs) are a leading cause of hospitalization and amputation in this population (Lavery, Armstrong, & Wunderlich, 2006). These infections range from superficial cellulitis to deep abscesses and life-threatening necrotizing fasciitis.

Specific Features: The inflammatory response is often blunted. Classic signs like erythema and warmth may be subtle, while pain can be absent due to peripheral neuropathy, leading to a dangerous delay in diagnosis. The infections are characterized by extensive tissue necrosis and poor wound healing (Peleg et al., 2007).

Polymicrobial Nature: Unlike pyodermas in other settings, DFIs are frequently polymicrobial. While *S. aureus* is a key pathogen, infections often involve a complex mixture of aerobes (*Streptococcus* spp., Enterobacteriaceae like *E. coli* and *Klebsiella*) and anaerobes (*Bacteroides* spp., *Peptostreptococcus* spp.) (Lipsky et al., 2012).

Microbiological Profile and Antibiotic Resistance - A critical finding across numerous studies is the alarmingly high prevalence of MRSA in both HIV-infected and diabetic populations.

Staphylococcus aureus and MRSA: *S. aureus* remains the single most important pathogen. Nasal and skin colonization with *S. aureus*, including MRSA, is significantly higher in these

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groups compared to healthy individuals. In hospital and community settings, the prevalence of MRSA as the causative agent of pyoderma in HIV-positive patients can reach 40-75% in some reports (Crum-Cianflone et al., 2009). Similarly, MRSA is a major pathogen in DFIs, complicating treatment and worsening outcomes.

Resistance Patterns: Beyond methicillin resistance, these strains often exhibit resistance to multiple other antibiotic classes, including macrolides (e.g., erythromycin), clindamycin, and fluoroquinolones, leaving few oral treatment options.

Diagnostic Challenges - The atypical nature of pyodermas in these hosts creates significant diagnostic dilemmas.

Mimics and Differential Diagnosis: In HIV patients, ulcerative pyoderma must be differentiated from other opportunistic infections like cryptococcosis, histoplasmosis, mycobacterial infections (tuberculosis cutis), and viral infections (CMV ulcers), as well as from malignancies like Kaposi's sarcoma and lymphoma cutis. In diabetic patients, a rapidly progressing cellulitis must be urgently distinguished from necrotizing fasciitis, which is a surgical emergency.

Importance of Invasive Diagnostics: A definitive diagnosis often cannot be made on clinical grounds alone. Skin biopsy for histopathology and tissue culture is mandatory in most atypical or non-responsive cases. Histology helps to rule out non-bacterial causes, while tissue culture provides a more accurate microbiological diagnosis than superficial swabs, which may only reflect surface colonizers (Stevens et al., 2014).

DISCUSSION

The findings clearly demonstrate that immunodeficiency profoundly reshapes the host-pathogen interaction in pyoderma. The underlying pathophysiology explains the distinct clinical patterns observed. In HIV infection, the defect in T-cell-mediated immunity, particularly the Th1 and Th17 pathways, impairs the recruitment and activation of neutrophils and macrophages required to contain staphylococcal infections. This leads to a failure to form well-organized abscesses, resulting in more diffuse, ulcerative, and chronic disease courses (Kaplan, 2010). The restoration of immune function with ART often leads to a dramatic improvement in the control of recurrent pyodermas.

In diabetes mellitus, the pathogenic triad of hyperglycemia, vascular insufficiency, and neuropathy is central. Hyperglycemia directly impairs neutrophil function and promotes bacterial growth. Diabetic microangiopathy compromises blood flow, limiting the delivery of immune cells and antibiotics to the site of infection and creating a hypoxic environment that favors the growth of anaerobic bacteria. Peripheral neuropathy reduces sensory perception, meaning that minor trauma can go unnoticed, allowing infections to establish and progress to a severe stage before being detected (Joshi, Caputo, & Weitekamp, 1999).

The high prevalence of MRSA in both groups is a major public health concern. It is driven by multiple factors, including frequent healthcare contact, recurrent hospitalizations, and repeated courses of antibiotics. This underscores the inadequacy of standard empirical antibiotic choices (e.g., cephalexin or dicloxacillin) in these patients. The choice of therapy

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must be guided by local resistance data and, ideally, by culture and sensitivity results from the specific patient.

The diagnostic challenge presented by these atypical infections necessitates a paradigm shift from simple visual diagnosis to a more aggressive, investigation-driven approach. The clinician's threshold for performing a skin biopsy for culture and histopathology should be very low. A multidisciplinary approach involving dermatologists, infectious disease specialists, endocrinologists (for DM), and surgeons is often crucial for optimal patient outcomes, particularly in cases of deep-seated or necrotizing infections.

CONCLUSION

Pyodermas in patients with HIV infection and diabetes mellitus are clinically distinct from those in the immunocompetent population, characterized by a more severe, extensive, atypical, and recurrent course.

The clinical presentation is directly influenced by the nature of the immunodeficiency: ulcerative and vegetative forms are more characteristic of advanced HIV, while deep, necrotic, and polymicrobial infections are hallmarks of poorly controlled diabetes.

The microbiological landscape is dominated by *Staphylococcus aureus*, with an alarmingly high prevalence of Methicillin-resistant *S. aureus* (MRSA) in both patient groups, posing a significant therapeutic challenge.

Accurate diagnosis is often difficult due to atypical presentations that mimic other infectious and malignant conditions, making invasive diagnostic procedures like skin biopsy for histology and tissue culture essential.

Effective management of pyoderma in these patients is impossible without simultaneous and rigorous management of the underlying immunodeficiency, requiring a collaborative, multidisciplinary approach.

RECOMMENDATIONS

Mandatory Microbiological Diagnosis: Bacterial culture with antibiotic susceptibility testing should be considered a standard of care for any significant pyoderma (deep, extensive, or non-responsive) in HIV-positive or diabetic patients before initiating or modifying systemic antibiotic therapy.

Informed Empirical Therapy: When empirical therapy is necessary for severe infections, it should be guided by local institutional and community MRSA prevalence data. Agents with reliable MRSA coverage (e.g., trimethoprim-sulfamethoxazole, doxycycline, clindamycin for outpatient; vancomycin, linezolid, or daptomycin for inpatient) should be considered.

Aggressive Management of Underlying Conditions: Dermatological treatment must be integrated with optimal management of the primary disease. This includes adherence to antiretroviral therapy (ART) to achieve immune reconstitution in HIV patients and stringent glycemic control (targeting HbA1c < 7.0%) in diabetic patients.

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Low Threshold for Surgical Consultation: In cases of deep abscesses, carbuncles, or any suspicion of necrotizing fasciitis (e.g., pain out of proportion to clinical signs, dusky skin, crepitus), urgent surgical consultation for incision and drainage or debridement is paramount.

Patient Education: Comprehensive patient education on preventative measures is critical. This includes instruction on daily skin inspection (especially foot checks for diabetics), proper hygiene, prompt care of minor wounds, and understanding the signs of a worsening infection that require immediate medical attention.

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