**RESEARCH PAPER**

# Psoas Abscess in Patients Living with Human Immunodeficiency Virus: A Narrative Review of Epidemiology, Clinical Presentation, Diagnostic Challenges, Microbiology, and Management

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**ABSTRACT**

**Background:** Psoas abscess is an uncommon but potentially life-threatening infectious condition involving the iliopsoas compartment. The condition may occur as a primary infection through hematogenous spread or as a secondary process arising from contiguous extension of infection from adjacent structures. Individuals living with human immunodeficiency virus (HIV) are at increased risk of deep-seated infections because of immune dysregulation, opportunistic pathogens, delayed diagnosis, and coexisting comorbidities. Although psoas abscess is uncommon in the general population, it represents an important diagnostic consideration in HIV-infected individuals presenting with fever, constitutional symptoms, abdominal pain, or lower back pain.

**Objective:** To provide a comprehensive narrative review of the epidemiology, pathogenesis, microbiology, clinical manifestations, diagnostic evaluation, and management of psoas abscess in patients living with HIV.

**Methods:** A narrative review of published literature was performed using major biomedical databases. Relevant studies including observational studies, case series, case reports, reviews, and guideline documents addressing psoas abscess and HIV infection were evaluated. Particular emphasis was placed on epidemiology, microbial spectrum, diagnostic approaches, and therapeutic outcomes.

**Results:** The available literature suggests that psoas abscess in HIV-positive individuals demonstrates considerable heterogeneity in etiology and clinical presentation. Staphylococcus aureus remains the predominant pathogen in pyogenic disease, while Mycobacterium tuberculosis constitutes an important cause in regions with high tuberculosis prevalence. Clinical manifestations are frequently nonspecific and may contribute to delayed diagnosis. Computed tomography and magnetic resonance imaging remain the cornerstone imaging modalities. Management requires prompt antimicrobial therapy combined with image-guided or surgical drainage when indicated.

**Conclusion:** Psoas abscess should be considered in HIV-infected patients presenting with persistent fever, back pain, abdominal discomfort, gait disturbance, or unexplained constitutional symptoms. Early recognition, appropriate microbiological evaluation, and timely intervention are essential for reducing morbidity and mortality. Further studies are required to better characterize HIV-specific risk factors, optimal treatment strategies, and long-term outcomes.

**Keywords**: *HIV; Human Immunodeficiency Virus; Psoas Abscess; Iliopsoas Abscess; Tuberculosis; Opportunistic Infections; Deep-Seated Infection; Immunocompromised Host.*

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## INTRODUCTION

Psoas abscess is a suppurative infection involving the iliopsoas muscle compartment and represents an uncommon but clinically significant cause of fever, abdominal pain, and lower back discomfort. The condition has been increasingly recognized with the widespread availability of advanced imaging modalities, particularly computed tomography (CT) and magnetic resonance imaging (MRI). Despite improvements in diagnostic capabilities, psoas abscess continues to pose substantial diagnostic challenges because of its nonspecific clinical manifestations and variable underlying etiologies[1-3,5-7,31-36].

The iliopsoas compartment is formed by the psoas major, psoas minor, and iliacus muscles. Owing to its extensive anatomical relationships with the vertebral column, gastrointestinal tract, retroperitoneal structures, kidneys, ureters, pancreas, and hip joint, the psoas muscle can serve as a conduit for infection arising from multiple anatomical

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locations. Consequently, psoas abscess may develop either through hematogenous dissemination of microorganisms or through direct extension from adjacent infectious foci[1-3,5-7,31-36].

Historically, psoas abscess was considered a rare disease. However, increased clinical awareness and advances in radiological imaging have resulted in more frequent diagnosis. The incidence remains difficult to determine because many cases are reported as isolated case reports or small series. The epidemiology varies considerably across geographical regions, reflecting differences in tuberculosis burden, prevalence of immunosuppressive conditions, healthcare access, and microbial ecology[1-3,5-7,31-36].

Human immunodeficiency virus infection remains a major global health concern despite remarkable advances in antiretroviral therapy. HIV-associated immune dysfunction predisposes affected individuals to a broad spectrum of bacterial, mycobacterial, fungal, and parasitic infections. While opportunistic infections have declined substantially in regions with widespread antiretroviral therapy coverage, deep-seated infections continue to contribute significantly to morbidity and healthcare utilization[1-3,5-7,31-36].

Patients living with HIV possess several characteristics that may increase susceptibility to psoas abscess formation. Progressive depletion of CD4+ lymphocytes impairs cellular immunity and facilitates infection by both common bacterial pathogens and opportunistic organisms. Coexisting tuberculosis, recurrent bacteremia, intravenous drug use, malnutrition, diabetes mellitus, chronic kidney disease, and delayed healthcare presentation may further increase risk. In addition, atypical clinical presentations are common among immunocompromised individuals, potentially delaying diagnosis and treatment[1-3,5-7,31-36].

The relationship between HIV infection and psoas abscess is particularly relevant in low- and middle-income countries where tuberculosis remains endemic. Tuberculous spondylodiscitis with extension into the psoas compartment represents a well-recognized mechanism of abscess formation. HIV-associated immunosuppression substantially increases the risk of both pulmonary and extrapulmonary tuberculosis, thereby creating a unique clinical environment in which psoas abscess may emerge as a manifestation of disseminated disease[1-3,5-7,31-36].

Despite the clinical importance of this condition, the available literature remains fragmented and largely consists of case reports, retrospective studies, and small observational cohorts. Consequently, clinicians frequently encounter uncertainty regarding epidemiology, microbiological evaluation, optimal diagnostic pathways, antimicrobial selection, and procedural management in HIV-infected individuals[1-3,5-7,31-36].

This narrative review aims to summarize current knowledge regarding psoas abscess in patients living with HIV, with particular emphasis on epidemiology, pathogenesis, microbiology, clinical presentation, diagnosis, treatment, and future research priorities[1-3,5-7,31-36].

## EPIDEMIOLOGY

Psoas abscess is generally classified into primary and secondary forms. Primary psoas abscess develops through hematogenous or lymphatic dissemination of microorganisms from distant sites in the absence of an identifiable local source. Secondary psoas abscess results from contiguous spread of infection from adjacent anatomical structures. This distinction remains clinically relevant because microbial profiles, risk factors, and therapeutic considerations differ between the two categories[5-7,10,16,24].

Primary psoas abscess accounts for a greater proportion of cases in developing regions and among immunocompromised individuals. Staphylococcus aureus is responsible for most cases of primary disease. HIV infection represents an important predisposing condition because impaired immune defenses facilitate bloodstream dissemination and establishment of infection within deep muscular compartments[5-7,10,16,24].

Secondary psoas abscess is commonly associated with gastrointestinal disorders such as Crohn's disease, appendicitis, diverticulitis, colorectal malignancy, and perforated viscus. Additional causes include vertebral osteomyelitis, spondylodiscitis, renal infections, infected vascular grafts, and postoperative complications. In HIV-positive individuals, secondary abscesses may arise from both conventional pathogens and opportunistic infections[5-7,10,16,24].

The epidemiological profile of psoas abscess in HIV differs according to geographical location. In high-income countries with widespread antiretroviral therapy availability, pyogenic bacterial infections predominate. Conversely, in regions with high tuberculosis prevalence, tuberculous psoas abscess remains a major clinical entity. Numerous reports from Asia and sub-Saharan Africa have highlighted the significant contribution of spinal tuberculosis and disseminated mycobacterial disease to psoas abscess formation among HIV-infected patients[5-7,10,16,24].

Although robust incidence data are lacking, several studies have demonstrated that immunosuppression is consistently associated with increased risk of deep-seated soft tissue infections. Lower CD4 cell counts have been associated with greater susceptibility to invasive bacterial disease, recurrent bacteremia, and extrapulmonary tuberculosis, all of which may contribute to abscess formation[5-7,10,16,24].

The widespread implementation of antiretroviral therapy has altered the epidemiology of HIV-associated infections. Improved immune restoration has led to reductions in many opportunistic infections; however, psoas abscess continues to occur because of delayed HIV diagnosis, treatment interruptions, antimicrobial resistance, and persistent tuberculosis burden in endemic regions. As HIV-positive individuals live longer, age-related

comorbidities such as diabetes mellitus, malignancy, and chronic kidney disease may further influence susceptibility to psoas abscess[5-7,10,16,24].

## ANATOMY AND PATHOPHYSIOLOGY

The psoas major muscle originates from the transverse processes and vertebral bodies of the twelfth thoracic and lumbar vertebrae and descends through the retroperitoneum to insert on the lesser trochanter of the femur. Together with the iliacus muscle, it forms the iliopsoas complex, which functions as the principal flexor of the hip[24,30-32,38,40].

Several anatomical characteristics contribute to the vulnerability of the psoas compartment to infectious processes. The muscle possesses a rich vascular supply, facilitating hematogenous seeding during episodes of bacteremia. Furthermore, its proximity to the vertebral column, gastrointestinal tract, kidneys, ureters, pancreas, and pelvic structures permits direct extension of infection from contiguous sites[24,30-32,38,40].

In HIV-infected individuals, multiple mechanisms may contribute to psoas abscess development. Defects in cell-mediated immunity impair macrophage activation and intracellular pathogen clearance. HIV-associated immune activation and chronic inflammation may further compromise host defense mechanisms. Reduced CD4 counts are associated with increased susceptibility to invasive bacterial infections, mycobacterial disease, and opportunistic pathogens[24,30-32,38,40].

Tuberculosis deserves special consideration in this population. Vertebral tuberculosis may cause destruction of vertebral bodies and intervertebral discs, allowing extension of caseous material through fascial planes into the psoas compartment. This process may result in large abscesses containing substantial volumes of purulent or necrotic material before clinical symptoms become apparent[24,30-32,38,40].

Pyogenic psoas abscesses typically develop following bacteremia or local spread from adjacent infections. Once microorganisms gain access to the muscle compartment, inflammatory cell recruitment, tissue necrosis, and abscess cavity formation occur. Delayed diagnosis may permit progressive enlargement of the abscess, leading to compression of adjacent structures, septicemia, and metastatic infectious complications[24,30-32,38,40].

## MICROBIOLOGICAL SPECTRUM

The microbiological profile of psoas abscess in patients living with HIV is diverse and is influenced by the degree of immunosuppression, geographical location, exposure history, healthcare-associated factors, and local endemic infections. Although Staphylococcus aureus remains the predominant pathogen worldwide, HIV-positive patients demonstrate a broader microbial spectrum that includes mycobacteria, Gram-negative bacilli, anaerobes, and occasionally fungal organisms[6,13,25,30,37-39].

### Staphylococcus Aureus

Staphylococcus aureus is the most frequently isolated organism in primary psoas abscess and continues to account for a substantial proportion of cases in HIV-infected individuals. The organism possesses several virulence factors that facilitate hematogenous dissemination and abscess formation, including adhesins, toxins, and biofilm-producing capabilities. Both methicillin-sensitive Staphylococcus aureus (MSSA) and methicillin-resistant Staphylococcus aureus (MRSA) have been implicated[6,13,25,30,37-39].

The increased incidence of recurrent skin and soft tissue infections, bacteremia, and healthcare exposure among HIV-positive individuals may contribute to the predominance of S. aureus. Persistent bacteremia can result in seeding of the richly vascularized psoas muscle and subsequent abscess formation[6,13,25,30,37-39].

### Gram-Negative Organisms

Gram-negative bacilli are particularly important in secondary psoas abscesses arising from gastrointestinal or genitourinary sources. Common organisms include:[6,13,25,30,37-39]

- Escherichia coli
- Klebsiella pneumonia
- Proteus species
- Enterobacter species
- Pseudomonas aeruginosa

The prevalence of these organisms is influenced by underlying pathology. For example, urinary tract infections, renal abscesses, and gastrointestinal perforations may lead to direct extension of Gram-negative pathogens into the psoas compartment[6,13,25,30,37-39].

Among HIV-positive individuals with advanced disease, recurrent hospitalizations and exposure to broad-spectrum antibiotics may increase the likelihood of multidrug-resistant Gram-negative infections[6,13,25,30,37-39].

### Anaerobic Bacteria

Anaerobic organisms are commonly encountered in secondary psoas abscesses associated with gastrointestinal pathology. Frequently isolated anaerobes include:[6,13,25,30,37-39]

- Bacteroides fragilis
- Peptostreptococcus species
- Fusobacterium species
- Clostridium species

Polymicrobial infections involving anaerobes and facultative bacteria are not uncommon when abscesses arise from bowel-related pathology. Failure to provide adequate anaerobic coverage may contribute to treatment failure[6,13,25,30,37-39].

### Mycobacterium Tuberculosis

Tuberculosis remains one of the most important causes of psoas abscess in HIV-endemic regions. Tuberculous psoas abscess most commonly develops secondary to spinal tuberculosis (Pott disease), although hematogenous dissemination may also occur[6,13,25,30,37-39].

The burden of tuberculosis-associated psoas abscess is particularly significant in low- and middle-income countries. HIV infection substantially increases susceptibility to both pulmonary and extrapulmonary tuberculosis. In many reported cases, psoas abscess may represent the first manifestation leading to HIV diagnosis[6,13,25,30,37-39].

Tuberculous abscesses often demonstrate a more indolent course than pyogenic abscesses. Symptoms may evolve gradually over weeks or months, contributing to diagnostic delay. Constitutional symptoms such as weight loss, anorexia, night sweats, and chronic fever are frequently observed[6,13,25,30,37-39].

**Non-Tuberculous Mycobacteria**

Although uncommon, non-tuberculous mycobacteria (NTM) should be considered in severely immunocompromised individuals. Organisms reported in HIV-positive patients include:[6,13,25,30,37-39]

- Mycobacterium avium complex
- Mycobacterium kansasii
- Mycobacterium fortuitum

NTM-associated psoas abscesses are usually encountered in patients with advanced immunosuppression and low CD4 counts. Diagnosis often requires culture, molecular testing, and histopathological examination[6,13,25,30,37-39].

**Fungal Pathogens**

Fungal psoas abscesses are rare but may occur in patients with profound immunosuppression. Reported organisms include:[6,13,25,30,37-39]

- Candida species
- Histoplasma capsulatum
- Cryptococcus neoformans

These infections are associated with significant morbidity and frequently require prolonged antifungal therapy in addition to drainage procedures[6,13,25,30,37-39].

**Polymicrobial Infection**

Polymicrobial infections are more commonly encountered in secondary psoas abscesses. Simultaneous isolation of aerobic and anaerobic organisms may occur, particularly in cases related to gastrointestinal perforation, inflammatory bowel disease, or postoperative infections[6,13,25,30,37-39].

The possibility of polymicrobial disease should be considered when selecting empirical antimicrobial therapy, especially in HIV-positive patients with severe sepsis or intra-abdominal pathology[6,13,25,30,37-39].

## CLINICAL PRESENTATION

The clinical manifestations of psoas abscess are often nonspecific and may overlap with numerous infectious, neurological, musculoskeletal, gastrointestinal, and genitourinary disorders. Consequently, diagnosis is frequently delayed[2,3,7,10,12,14].

**Classical Triad**

Traditionally, psoas abscess has been described by a clinical triad consisting of:

- Fever
- Back or flank pain
- Limping or gait disturbance

However, the complete triad is present in only a minority of patients. Reliance on these classical findings may therefore result in missed diagnoses[2,3,7,10,12,14].

**Constitutional Symptoms**

Many HIV-positive patients present with constitutional symptoms including:

- Fever
- Weight loss
- Fatigue
- Malaise
- Anorexia
- Night sweats

These manifestations may be especially prominent in tuberculous psoas abscesses.

**Pain Syndromes**

Pain represents the most common presenting symptom. Patients may report:

- Lower back pain
- Flank pain
- Abdominal pain
- Groin pain
- Hip pain

Pain frequently worsens with hip extension because stretching of the inflamed psoas muscle provokes discomfort. Some patients assume a flexed hip posture to reduce symptoms[2,3,7,10,12,14].

**Gait Abnormalities**

Inflammation of the iliopsoas complex may impair hip flexion and ambulation. Patients may present with:[2,3,7,10,12,14]

- Limping
- Difficulty walking
- Restricted hip movement
- Antalgic gait

These symptoms can mimic orthopedic disorders such as septic arthritis, osteomyelitis, or lumbar radiculopathy[2,3,7,10,12,14].

**Abdominal Manifestations**
Abdominal symptoms are common and may include:

- Lower abdominal pain
- Palpable abdominal mass
- Abdominal tenderness
- Nausea
- Vomiting

Large abscesses may occasionally present as palpable flank or lower abdominal swellings[2,3,7,10,12,14].

**Neurological Symptoms**
Compression of adjacent neural structures may result in:

- Femoral neuropathy
- Sensory disturbances
- Muscle weakness
- Reduced knee reflexes

Although uncommon, these findings may indicate extensive disease.

**HIV-Specific Considerations**
In HIV-positive individuals, clinical presentation may be atypical. Patients with advanced immunosuppression may have muted inflammatory responses and fewer localizing symptoms. Concurrent opportunistic infections may further obscure the diagnosis[2,3,7,10,12,14].

A high index of suspicion is therefore required in HIV-infected patients presenting with prolonged fever, unexplained constitutional symptoms, persistent back pain, or evidence of disseminated infection[2,3,7,10,12,14].

**DIAGNOSTIC EVALUATION**
Prompt diagnosis is critical because delayed recognition may result in sepsis, extension of infection, and increased mortality[2,11,18,19,27].

**Laboratory Investigations**
Routine laboratory studies often reveal nonspecific inflammatory changes.

**Common Abnormalities Include:**
- Leukocytosis
- Elevated erythrocyte sedimentation rate (ESR)
- Elevated C-reactive protein (CRP)
- Anemia of chronic disease
- Hypoalbuminemia

Among HIV-positive patients, CD4 count and HIV viral load provide important information regarding immune status and may influence differential diagnosis[2,11,18,19,27].

**Microbiological Studies**
Blood cultures should be obtained before initiation of antimicrobial therapy whenever possible. Positive blood cultures may identify causative organisms in a significant proportion of pyogenic abscesses[2,11,18,19,27].

Image-guided aspiration remains the most valuable microbiological diagnostic procedure because it permits:[2,11,18,19,27]

- Gram staining
- Aerobic culture
- Anaerobic culture
- Mycobacterial culture
- Fungal culture
- Molecular diagnostic testing

Failure to obtain microbiological confirmation may result in inappropriate antimicrobial selection and poorer outcomes[2,11,18,19,27].

**Ultrasonography**
Ultrasonography is frequently used as an initial imaging modality because it is inexpensive, widely available, and free from ionizing radiation[2,11,18,19,27].

**Advantages Include:**
- Bedside availability
- Guidance for aspiration
- Rapid assessment

However, ultrasonography is operator dependent and may miss small or deeply located abscesses[2,11,18,19,27].

**Computed Tomography**
Contrast-enhanced computed tomography is considered the diagnostic modality of choice in most patients[2,11,18,19,27].

**CT Provides:**
- Precise anatomical localization
- Determination of abscess size
- Identification of loculations
- Detection of adjacent pathology
- Guidance for percutaneous drainage

Reported diagnostic sensitivity approaches 90–100% in many studies.

**Magnetic Resonance Imaging**
MRI offers excellent soft tissue characterization and is particularly useful when:[2,11,18,19,27]

- Spinal tuberculosis is suspected
- Vertebral osteomyelitis is present
- Epidural extension is considered
- Neurological complications are suspected

MRI may outperform CT in detecting early spinal and soft tissue involvement.

**Histopathology and Molecular Diagnostics**

In selected cases, tissue biopsy may be necessary to establish diagnosis. Histopathological examination is particularly valuable in suspected tuberculosis, fungal infections, or malignancy-associated lesions[2,11,18,19,27].

Modern molecular techniques including nucleic acid amplification tests can improve diagnostic accuracy and facilitate earlier pathogen identification[2,11,18,19,27].

## TUBERCULOUS PSOAS ABSCESS IN HIV

Tuberculosis remains one of the most important causes of psoas abscess in regions with a high burden of HIV infection. The association between HIV and tuberculosis has significantly altered the epidemiology of extrapulmonary tuberculosis, leading to increased incidence of skeletal, spinal, and soft-tissue involvement. Among these manifestations, tuberculous psoas abscess represents a clinically important but frequently underrecognized entity[4,9,17,20-22,31-36].

The majority of tuberculous psoas abscesses arise secondary to vertebral tuberculosis (Pott disease). Destruction of vertebral bodies and intervertebral discs facilitates the spread of caseous material through fascial planes into the psoas compartment. Less commonly, abscess formation may occur through hematogenous dissemination or direct extension from intra-abdominal tuberculous lesions[4,9,17,20-22,31-36].

HIV-induced impairment of cell-mediated immunity increases susceptibility to both primary infection and reactivation of latent tuberculosis. Patients with advanced immunosuppression are more likely to develop disseminated and extrapulmonary disease. In some cases, psoas abscess may be the initial manifestation leading to the diagnosis of previously unrecognized HIV infection[4,9,17,20-22,31-36].

Clinically, tuberculous psoas abscess often follows a more indolent course than pyogenic abscesses. Symptoms may evolve over several weeks or months and include:[4,9,17,20-22,31-36]

- Chronic low-grade fever
- Progressive weight loss
- Night sweats
- Lower back pain
- Limping
- Constitutional symptoms

The absence of acute inflammatory manifestations frequently contributes to delayed diagnosis. Large abscesses may be present before medical attention is sought[4,9,17,20-22,31-36].

Magnetic resonance imaging is particularly valuable in evaluating tuberculous psoas abscess because it can simultaneously assess vertebral involvement, paraspinal collections, epidural extension, and neural compression. Computed tomography remains useful for defining abscess anatomy and facilitating image-guided aspiration[4,9,17,20-22,31-36].

**Microbiological confirmation should be pursued whenever feasible through:**

- Acid-fast staining
- Mycobacterial culture
- Nucleic acid amplification tests
- Histopathological examination

The management of tuberculous psoas abscess involves standard antitubercular therapy in accordance with national or international guidelines. Percutaneous drainage is frequently employed for large collections, while surgical intervention is reserved for selected patients with neurological compromise, spinal instability, extensive vertebral destruction, or failure of conservative treatment[4,9,17,20-22,31-36].

## MANAGEMENT STRATEGIES

The management of psoas abscess requires a multidisciplinary approach involving infectious disease specialists, internists, radiologists, microbiologists, surgeons, and HIV care providers. Successful treatment depends upon prompt diagnosis, source control, appropriate antimicrobial therapy, and optimization of immune status[23,27-29,34-36].

**Initial Stabilization**

Patients presenting with systemic inflammatory response syndrome or sepsis require immediate stabilization. Key components include:[23,27-29,34-36]

- Hemodynamic assessment
- Intravenous fluid resuscitation
- Blood culture collection
- Baseline laboratory investigations
- Early antimicrobial therapy

Severe sepsis and septic shock warrant intensive monitoring and supportive care.

**Empirical Antimicrobial Therapy**

Empirical therapy should be guided by:

- Clinical presentation
- Probable source of infection
- Local resistance patterns
- HIV disease status
- Presence of healthcare-associated risk factors

**For Suspected Pyogenic Abscesses, Antimicrobial Regimens Should Provide Coverage Against:[23,27-29,34-36]**

- Staphylococcus aureus

- Streptococci
- Enterobacterales
- Anaerobes when indicated

**Examples of Empirical Regimens May Include:**
- Piperacillin–tazobactam
- Ceftriaxone plus metronidazole
- Carbapenem-based therapy in selected patients
- Vancomycin when MRSA is suspected

Subsequent therapy should be modified according to culture results and susceptibility testing[23,27-29,34-36].

**Image-Guided Percutaneous Drainage**
Percutaneous drainage has become the preferred intervention for many patients because it is minimally invasive and associated with favorable outcomes[23,27-29,34-36].

**Advantages Include:**
- Reduced morbidity
- Shorter hospitalization
- Diagnostic specimen acquisition
- Avoidance of major surgery

CT-guided drainage remains the most commonly employed technique. Ultrasound-guided drainage may be appropriate in selected cases[23,27-29,34-36].

**Surgical Management**
Although less frequently required in the modern era, surgical drainage remains important in selected situations including:[23,27-29,34-36]

- Multiloculated abscesses
- Failure of percutaneous drainage
- Associated intra-abdominal pathology
- Extensive necrotic tissue
- Recurrent abscess formation
- Spinal instability

Surgical intervention permits direct visualization, debridement, and definitive management of underlying pathology[23,27-29,34-36].

**Antiretroviral Therapy Considerations**
Optimal HIV management represents an essential component of treatment. Assessment should include:[23,27-29,34-36]

- CD4 cell count
- HIV viral load
- Current antiretroviral regimen
- Drug interactions
- Adherence status

Patients not receiving antiretroviral therapy should be evaluated for treatment initiation according to current HIV guidelines. In those with active tuberculosis, timing of antiretroviral therapy should be individualized based on immune status and risk of immune reconstitution inflammatory syndrome[23,27-29,34-36].

**Monitoring Response to Treatment**
Clinical improvement is typically assessed through:

- Resolution of fever
- Reduction in pain
- Improved mobility
- Decreasing inflammatory markers
- Radiological improvement

Repeat imaging may be necessary when clinical response is suboptimal or recurrence is suspected[23,27-29,34-36].

## OUTCOMES AND PROGNOSIS

The prognosis of psoas abscess has improved substantially because of advances in imaging, antimicrobial therapy, and minimally invasive drainage techniques. Nevertheless, morbidity remains considerable, particularly among immunocompromised individuals[5,10,13,26,28,29].

**Several Factors Influence Outcomes:**
- Delay in diagnosis
- Advanced HIV disease
- Low CD4 count
- Disseminated tuberculosis
- Septic shock
- Multidrug-resistant organisms
- Failure to achieve source control

Early diagnosis and timely intervention are consistently associated with improved outcomes. Most patients experience substantial clinical improvement following appropriate antimicrobial therapy and drainage procedures[5,10,13,26,28,29].

**Recurrence May Occur In Cases Involving:**
- Inadequate drainage
- Persistent underlying source
- Drug-resistant pathogens
- Poor adherence to treatment
- Severe immunosuppression

Mortality is generally higher among patients with delayed presentation, extensive disease burden, or severe systemic infection. HIV-associated comorbidities may further contribute to adverse outcomes[5,10,13,26,28,29].

## CURRENT GAPS IN EVIDENCE AND FUTURE DIRECTIONS

Despite increasing recognition of psoas abscess in HIV-positive individuals, significant knowledge gaps remain[2,5,35,36].

First, the majority of available data are derived from case reports and small retrospective studies. Consequently, robust epidemiological estimates remain limited[2,5,35,36].

Second, there is a lack of prospective studies evaluating HIV-specific predictors of outcomes. The relationship between CD4 count, viral suppression, and treatment response requires further investigation[2,5,35,36].

Third, optimal antimicrobial duration remains uncertain in many clinical scenarios, particularly for polymicrobial infections and unusual pathogens[2,5,35,36].

Fourth, emerging molecular diagnostic techniques may facilitate earlier pathogen identification and improve therapeutic precision. Future research should explore their role in routine clinical practice[2,5,35,36].

Finally, multi-center registries and prospective observational studies are needed to better characterize clinical presentations, microbiological patterns, treatment strategies, and long-term outcomes among patients living with HIV[2,5,35,36].

## CONCLUSION

Psoas abscess is an uncommon but clinically significant infection that should be considered in HIV-positive patients presenting with fever, back pain, abdominal discomfort, gait abnormalities, or unexplained constitutional symptoms. HIV-associated immune dysfunction predisposes affected individuals to a broad spectrum of bacterial, mycobacterial, and opportunistic pathogens. Tuberculosis remains an especially important cause in regions with a high burden of HIV and endemic tuberculosis[1,2,23,34-36].

The diagnosis is frequently challenging because clinical manifestations are often nonspecific. Computed tomography and magnetic resonance imaging remain the cornerstone diagnostic modalities, while microbiological confirmation through aspiration and culture is critical for targeted therapy[1,2,23,34-36].

Successful management requires a combination of appropriate antimicrobial treatment, effective drainage procedures, optimization of antiretroviral therapy, and close clinical monitoring. Early recognition and multidisciplinary care are essential for minimizing morbidity and mortality[1,2,23,34-36].

Given the limited HIV-specific literature currently available, further research is required to establish evidence-based recommendations regarding diagnosis, antimicrobial therapy, procedural interventions, and long-term outcomes in this vulnerable population[1,2,23,34-36].

**Table 1:** Common Etiological Organisms Associated with Psoas Abscess in HIV Patients

| Category | Common Organisms |
|---|---|
| Gram-positive bacteria | Staphylococcus aureus, Streptococcus spp. |
| Gram-negative bacteria | Escherichia coli, Klebsiella spp., Pseudomonas spp. |
| Anaerobes | Bacteroides spp., Peptostreptococcus spp. |
| Mycobacteria | Mycobacterium tuberculosis, NTM |
| Fungi | Candida spp., Histoplasma spp., Cryptococcus spp. |

**Table 2:** Diagnostic Modalities in Psoas Abscess

| Modality | Advantages | Limitations |
|---|---|---|
| **Ultrasonography** | Bedside, inexpensive | Operator dependent |
| **CT Scan** | Excellent anatomical detail, drainage guidance | Radiation exposure |
| **MRI** | Superior soft tissue characterization | Higher cost |
| **Aspiration and Culture** | Definitive microbiological diagnosis | Invasive procedure |

**Table 3:** Prognostic Factors Associated with Poor Outcome

| Factor | Potential Impact |
|---|---|
| **Delayed diagnosis** | Increased morbidity |
| **Low CD4 count** | Higher risk of dissemination |
| **Septic shock** | Increased mortality |
| **Drug-resistant pathogens** | Treatment failure |
| **Inadequate drainage** | Recurrence |
| **Disseminated tuberculosis** | Prolonged treatment |

## REFERENCES

1. Shields D, Robinson P, Crowley TP. Iliopsoas abscess – a review and update on the literature. Int J Surg. 2012;10(9):466-469(1,2,23,34-36).
2. Xu C, Zhou Z, Wang S, Ren W, Yang X. Psoas abscess: an uncommon disorder. Postgrad Med J. 2024;100(1185):482-489(1,2,23,34-36).
3. Mallick IH, Thoufeeq MH, Rajendran TP. Iliopsoas abscesses. Postgrad Med J. 2004;80(946):459-462(1,2,23,34-36).

4. Vasigh M, Manteghi MJ, Alinejad S. Isolated psoas abscess caused by Mycobacterium tuberculosis. Clin Case Rep. 2022;10:e05882(1,2,23,34-36).
5. Sato T, Yasuda K, Furukawa K, et al. Epidemiological features and outcomes of patients with psoas abscess. Int J Infect Dis. 2021;105:293-298(1,2,23,34-36).
6. Ricci MA, Rose FB, Meyer KK. Pyogenic psoas abscess: worldwide variations in etiology. World J Surg. 1986;10(5):834-843(1,2,23,34-36).
7. van den Berge M, de Marie S, Kuipers T, Jansz AR, Bravenboer B. Psoas abscess: report of a series and review of the literature. Neth J Med. 2005;63(10):413-416(1,2,23,34-36).
8. Aboobakar R, Maharaj J, Singh B. Surgical management of psoas abscess in the human immunodeficiency virus era. Int J Surg Case Rep. 2018;42:95-99(1,2,23,34-36).
9. Olasinde AA, Olasinde OA, Oginni LM. Bilateral tuberculosis psoas abscess in a human immunodeficiency virus-positive patient. J Med Case Rep. 2024;18:310(1,2,23,34-36).
10. Thakral A, Gupta S, Sharma V, et al. Characteristics and outcomes of psoas abscess. Cureus. 2022;14:e21854(1,2,23,34-36).
11. He W, Zhang Y, Li J, et al. A case report of iliopsoas abscess and literature review. Medicine (Baltimore). 2024;103:e39288(1,2,23,34-36).
12. Xu BY, Shi J, Liu X, et al. Atypical presentation of pyogenic iliopsoas abscess. BMC Infect Dis. 2019;19:58(1,2,23,34-36).
13. Navarro López V, Ramos JM, Meseguer V, et al. Microbiology and outcome of iliopsoas abscess. Eur J Clin Microbiol Infect Dis. 2009;28(6):621-626(1,2,23,34-36).
14. Gruenwald I, Abrahamson J, Cohen O. Psoas abscess: case report and review of literature. J Urol. 1992;147(6):1624-1626(1,2,23,34-36).
15. Bartolo DCC, Ebbs SR, Cooper MJ. Psoas abscess in Bristol: a 10-year review. Int Surg. 1987;72:250-252(1,2,23,34-36).
16. Garner JP, Meiring PD, Ravi K, Gupta R. Psoas abscess—not as rare as we think? Colorectal Dis. 2007;9(3):269-274(1,2,23,34-36).
17. Goni V, Thapa BR, Vyas S, et al. Bilateral psoas abscess: atypical presentation of spinal tuberculosis. Arch Iran Med. 2012;15(4):253-256(1,2,23,34-36).
18. John Hopkins ABX Guide. Psoas abscess. Baltimore: Johns Hopkins University; 2010(1,2,23,34-36).
19. De U, Pal DK. Retroperitoneal abscess: diagnosis and treatment. Int Urol Nephrol. 2005;37(4):733-736(1,2,23,34-36).
20. Rakesh TP, Nair SP, Krishnan S. Large primary tuberculous psoas abscess in HIV patient. SAARC J Tuberc Lung Dis HIV/AIDS. 2014;11(2):56-60(1,2,23,34-36).
21. Ayen AA, Mohammed A, Tesfaye D. Pott's disease presenting with bilateral psoas abscesses. Int J Surg Case Rep. 2025;128:111001(1,2,23,34-36).
22. Laatiris H, Elouazzani H, et al. Bilateral tuberculous psoas abscesses: case report and literature review. Access Microbiol. 2025;7:1003(1,2,23,34-36).
23. Tabrizian P, Nguyen SQ, Greenstein A, Rajhbeharrysingh U, Divino CM. Management and treatment of iliopsoas abscess. Arch Surg. 2009;144(10):946-949(1,2,23,34-36).
24. Santaella RO, Fishman EK, Lipsett PA. Primary vs secondary iliopsoas abscess. Medicine (Baltimore). 1995;74(2):81-89(1,2,23,34-36).
25. Lin MF, Lau YJ, Hu BS, et al. Pyogenic psoas abscess: analysis of 27 cases. J Microbiol Immunol Infect. 1999;32(4):261-268(1,2,23,34-36).
26. López VN, Ramos JM, Meseguer V, et al. Clinical significance and prognosis of iliopsoas abscess. Infection. 2010;38(2):120-124(1,2,23,34-36).
27. Cantasdemir M, Kara B, Cebi D, et al. Percutaneous drainage of iliopsoas abscess. Cardiovasc Intervent Radiol. 2003;26(5):482-487(1,2,23,34-36).
28. Yacoub WN, Sohn HJ, Chan S, et al. Psoas abscess rarely requires surgical intervention. Am J Surg. 2008;196(2):223-227(1,2,23,34-36).
29. Dietrich A, Vaccarezza H, Vaccaro CA. Iliopsoas abscess: presentation, management and outcomes. Surg Laparosc Endosc Percutan Tech. 2013;23(1):45-48(1,2,23,34-36).
30. Crum-Cianflone NF. Bacterial, fungal, parasitic and viral myositis. Clin Microbiol Rev. 2008;21(3):473-494(1,2,23,34-36).
31. Golden MP, Vikram HR. Extrapulmonary tuberculosis: an overview. Am Fam Physician. 2005;72(9):1761-1768(1,2,23,34-36).
32. Sharma SK, Mohan A. Extrapulmonary tuberculosis. Indian J Med Res. 2004;120:316-353(1,2,23,34-36).
33. Lawn SD, Bekker LG, Miller RF. Immune reconstitution disease associated with mycobacterial infections. Lancet Infect Dis. 2005;5(6):361-373(1,2,23,34-36).
34. WHO. Consolidated Guidelines on Tuberculosis. Geneva: World Health Organization; 2023(1,2,23,34-36).

35. WHO. Consolidated Guidelines on HIV Prevention, Testing, Treatment, Service Delivery and Monitoring. Geneva: World Health Organization; 2024(1,2,23,34-36).

36. CDC. Guidelines for prevention and treatment of opportunistic infections in adults and adolescents with HIV. Atlanta: Centers for Disease Control and Prevention; 2023(1,2,23,34-36).

37. Brook I. Microbiology and management of abdominal infections. Dig Dis Sci. 2008;53(10):2585-2591(1,2,23,34-36).

38. Crum NF. Update on bacterial infections in HIV-positive patients. Curr Infect Dis Rep. 2005;7(5):377-384(1,2,23,34-36).

39. Habous M, Alghamdi M, et al. Psoas abscess caused by non-typhoid Salmonella in a man with HIV. IDCases. 2025;42:e02398(1,2,23,34-36).

40. Meyers BR, Sherman E. Infectious diseases involving the retroperitoneum. Infect Dis Clin North Am. 1988;2(4):963-976(1,2,23,34-36).