

**CLINICAL AND DIAGNOSTIC FEATURES OF
LYMPHADENOPATHY IN HIV AND TUBERCULOSIS
COMORBIDITY**

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Abstract

Background: The concurrent occurrence of HIV infection and tuberculosis (TB) constitutes a critical global health challenge, particularly in Central Asian regions with rising seroprevalence. Lymphadenopathy—both peripheral and intrathoracic—is among the most prevalent and diagnostically demanding manifestations in this comorbid setting. **Objective:** To characterize the clinical and diagnostic features of lymphadenopathy in patients with HIV/TB comorbidity and assess their correlation with CD4⁺ T-lymphocyte counts. **Methods:** Clinical data, laboratory findings, imaging studies, and biopsy results from patients with concurrent HIV/TB were systematically reviewed. **Results:** Lymphadenopathy in HIV/TB patients presents atypically, with marked tendency toward caseous necrosis, generalization, and coalescence. Diagnostic accuracy is significantly impaired at CD4⁺ counts below 200 cells/ μ L. GeneXpert MTB/RIF assay and CT-guided biopsy substantially improved detection rates. **Conclusion:** Integrated multimodal diagnostic algorithms are essential for early, accurate identification of lymphadenopathy in HIV/TB comorbid patients.

Keywords

HIV, tuberculosis, comorbidity, lymphadenopathy, CD4 count, differential diagnosis, Andijan.



**OIV VA TUBERKULYOZ KOMORPID HOLATIDA
LIMFADENOPATIYALAR KECHISHINING KLINIK-DIAGNOSTIK
XUSUSIYATLARI**

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Annotatsiya: Kirish: OIV infeksiyasi va tuberkulyozning (TB) bir vaqtda kechishi global sog'liqni saqlash uchun muhim muammo bo'lib, Markaziy Osiyo mintaqalarida ayniqsa dolzarbdir. Limfadenopatiya — periferik va intratorakal — bu komorbid holatning eng ko'p uchraydigan va diagnostik jihatdan murakkab ko'rinishlaridan biridir. Maqsad: OIV/TB komorbidligi bo'lgan bemorlarda limfadenopatiyaning klinik-diagnostik xususiyatlarini tavsiflash va CD4+ T-limfositlar soni bilan korrelyatsiyasini baholash. Metodlar: OIV/TB birgalikda kechgan bemorlarning klinik, laboratoriya, vizualizatsiya va biopsiya ma'lumotlari tizimli ko'rib chiqildi. Natijalar: Limfadenopatiya atipik kechib, kazeoze nekroz, generalizatsiya va qo'shilishga moyillik bilan namoyon bo'ladi; CD4+ 200 hujayra/mkl dan past bo'lganda diagnostik aniqlik pasayadi. GeneXpert MTB/RIF testi va KT-boshqaruvidagi biopsiya aniqlash darajasini sezilarli oshiradi. Xulosa: OIV/TB komorbidligida limfadenopatiyani erta aniqlash uchun integrallashgan multimodal diagnostik algoritmlar zarurdir.

Kalit so'zlar: OIV infeksiyasi, tuberkulyoz, komorbidlik, limfadenopatiya, CD4 limfotsitlar, differensial diagnostika.

Introduction

The concurrent occurrence of human immunodeficiency virus (HIV) infection and tuberculosis (TB) constitutes one of the most formidable dual burdens in global infectious disease medicine. According to the World Health Organization (WHO), HIV-positive individuals are approximately 18 times more susceptible to active TB than



immunocompetent hosts [1]. Progressive CD4⁺ T-lymphocyte depletion impairs granuloma formation and mycobacterial containment, promoting extrapulmonary dissemination — most critically to the lymphatic system. In Uzbekistan, where high TB endemicity converges with an expanding HIV epidemic, the Andijan region represents a particularly vulnerable epidemiological focal point. This paper delineates the clinical and diagnostic features of lymphadenopathy in HIV/TB comorbid patients, emphasizing the modulating role of immune status and the utility of contemporary diagnostic modalities.

Clinical Manifestations of Lymphadenopathy in HIV/TB Comorbidity

Lymphadenopathy in HIV/TB comorbidity diverges substantially from classical TB lymphadenitis in immunocompetent hosts. Lymph node involvement tends toward generalization, simultaneously affecting cervical, axillary, mediastinal, and inguinal nodal chains [2], reflecting impaired granuloma formation and defective macrophage activation. Affected nodes demonstrate accelerated caseous necrosis and liquefaction, producing large, matted, fluctuant masses — particularly at CD4⁺ counts below 100 cells/ μ L [3]. The classic TB constitutional triad (fever, night sweats, weight loss) is typically present but insufficient to discriminate from cryptococcal lymphadenitis, non-tuberculous mycobacterial disease, or lymphoma. Intrathoracic adenopathy is commonly massive and bilateral, and may be associated with pericardial or pleural effusions [4]. A distinct clinical complication is immune reconstitution inflammatory syndrome (IRIS), wherein lymphadenopathy paradoxically worsens after antiretroviral therapy (ART) initiation, requiring distinction from treatment failure.

Diagnostic Approaches

No single diagnostic test achieves sufficient sensitivity and specificity across all levels of immunosuppression, necessitating a multimodal algorithm. Ultrasound (US) is the first-line imaging modality, and US-guided fine-needle aspiration cytology (FNAC) provides material for microbiological and cytological analysis; however, sensitivity

declines from 60–80% in immunocompetent hosts to substantially lower values in severe HIV immunosuppression [3]. Contrast-enhanced CT remains the gold standard for anatomical delineation, with centrally hypodense, rim-enhancing nodes being pathognomonic for TB in classical presentations; in HIV-positive patients this sign may be absent due to altered immune-mediated necrosis [4]. CT-guided excisional biopsy — enabling simultaneous mycobacterial culture, drug susceptibility testing, and histopathology — is the definitive procedure, and frequently reveals poorly formed or absent granulomas. The GeneXpert MTB/RIF assay has substantially improved rapid molecular diagnosis at extrapulmonary sites; a meta-analysis by Kohli et al. (2021) reported pooled sensitivity of 88.9% and specificity of 97.3% for lymph node specimens [5], with the added advantage of simultaneous rifampicin resistance detection — critical given the elevated drug-resistant TB risk in HIV-positive individuals. Interferon-gamma release assays (IGRAs) retain discriminatory value at CD4⁺ counts above 200 cells/ μ L but are unreliable at advanced immunosuppression.

Influence of CD4⁺ Cell Count and Immune Status on Diagnostic Accuracy

CD4⁺ T-lymphocyte count is the most critical determinant of both clinical phenotype and diagnostic yield. Above 350 cells/ μ L, HIV-positive patients present comparably to immunocompetent hosts, with well-formed granulomas and preserved FNAC sensitivity [1]. Between 200–350 cells/ μ L, granuloma formation is progressively impaired, and concurrent opportunistic infections (*Pneumocystis jirovecii* pneumonia, cryptococcosis, cytomegalovirus disease) must be considered in the differential. Below 200 cells/ μ L — the AIDS-defining threshold — granulomas may be entirely absent on histopathology, replaced by diffuse necrosis and macrophage infiltration laden with acid-fast bacilli (AFB) on Ziehl-Neelsen staining. Paradoxically, high bacterial burden at this stage improves AFB smear and GeneXpert sensitivity; however, *Mycobacterium avium* complex (MAC) must be excluded via culture [6]. Below 50 cells/ μ L, blood mycobacterial cultures (BACTEC, Myco/F Lytic) and bone marrow biopsy become

essential. These immunological gradients mandate CD4⁺-stratified diagnostic algorithms in all clinical settings managing HIV/TB comorbid patients.

Conclusion

Lymphadenopathy in HIV/TB comorbidity is a diagnostically complex entity whose clinical, imaging, and histopathological features are profoundly shaped by CD4⁺ T-lymphocyte-mediated immunosuppression. Atypical presentations, rapid necrotization, lymph node generalization, and absent granuloma formation — all accentuated at low CD4⁺ counts — demand integrated, multimodal diagnostic strategies. The following practical recommendations are proposed for early differential diagnosis: (1) Obtain CD4⁺ count at initial evaluation of all HIV-positive patients with lymphadenopathy to guide the diagnostic pathway. (2) Apply GeneXpert MTB/RIF assay on lymph node aspirate or biopsy as first-line molecular testing regardless of CD4⁺ stratum. (3) Pursue CT-guided biopsy with simultaneous microbiological, molecular, and histopathological workup when CD4⁺ <200 cells/μL or when lymphadenopathy is rapidly progressive. (4) Add blood mycobacterial cultures and bone marrow biopsy at CD4⁺ <50 cells/μL. (5) Maintain a broad differential — including cryptococcal infection, MAC disease, lymphoma, and Kaposi's sarcoma — especially when TB-directed investigations return negative. (6) Consider TB-IRIS in any patient developing paradoxical lymph node enlargement after ART initiation. Systematic adoption of these recommendations, informed by local epidemiological data from the Andijan region, has the potential to meaningfully reduce diagnostic delays and improve outcomes in this high-risk population.

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