Review Article:

Revisiting Cutaneous Tuberculosis: Clinical Patterns, Diagnostic Challenges and Therapeutic Perspectives —A Narrative Review

Mohammed Atiqur Rahman[1]*, Mohammed Saiful Islam Bhuiyan[2], Kazi Rahila Ferdousi[3], Rajashish Chakrabortty[4]

1. Bangladesh Medical University, Dhaka, Bangladesh. Email: m_a_rahman88@yahoo.com; Orchid ID: https://orcid.org/0000-0002-6960-9004
2. Bangladesh Medical University, Dhaka, Bangladesh. Email: drsaifulib@bsmmu.edu.bd; Orchid ID: https://orcid.org/0000-0001-8532-4992
3. Bangladesh Medical University, Dhaka, Bangladesh. Email: rahilaferdous@gmail.com
4. Bangladesh Medical University, Dhaka, Bangladesh. Email:drrajashish@gmail.com; Orchid ID: https://orcid.org/0000-0001-9060-6185

Abstract

Cutaneous tuberculosis (CTB) is a rare yet clinically significant form of extrapulmonary tuberculosis caused primarily by Mycobacterium tuberculosis infection of the skin. Although it constitutes only a small fraction of total TB cases, CTB continues to present diagnostic and therapeutic challenges, particularly in endemic regions. This narrative review comprehensively summarizes current knowledge on the epidemiology, clinical patterns, diagnostic strategies, and management approaches of CTB, highlighting emerging perspectives and gaps in clinical practice. The disease exhibits a wide clinical spectrum, including lupus vulgaris, scrofuloderma, tuberculosis verrucosa cutis, and tuberculids, which reflect variations in host immunity and routes of infection. Diagnosis remains challenging due to its polymorphic presentation and similarity to other granulomatous dermatoses; while histopathology, Ziehl–Neelsen staining, and culture offer limited sensitivity; molecular techniques such as GeneXpert and PCR have enhanced diagnostic accuracy. Standard anti-tubercular therapy continues to be the mainstay of treatment, though drug-resistant cases necessitate individualized regimens. Early diagnosis and strict adherence to therapy are vital to prevent chronic disfigurement and relapse. Despite recent advancements, CTB remains under-recognized, underscoring the need for greater clinical awareness, improved laboratory capacity, and integrated collaboration between dermatology and infectious disease specialties to effectively address this neglected but important manifestation of tuberculosis.

Keywords: Cutaneous tuberculosis, Mycobacterium tuberculosis, diagnostic challenges, clinical spectrum, anti-tubercular therapy.

Introduction

Tuberculosis (TB) remains one of the world's most formidable infectious diseases, with an estimated 10.6 million new cases globally in 2023, according to the World Health Organization (WHO). While pulmonary tuberculosis accounts for the majority of cases, extrapulmonary TB represents approximately 15–25%, and cutaneous tuberculosis (CTB)—though rare—constitutes a clinically significant subset, comprising about 1–2% of extrapulmonary cases.[1] Recent WHO regional data indicate that South Asia and sub-Saharan Africa bear a disproportionately higher burden of TB, together contributing over 65% of global cases, with CTB prevalence correspondingly elevated in these endemic regions due to higher background infection rates, socioeconomic challenges, and limited access to diagnostic facilities.

CTB represents a dynamic interplay between Mycobacterium tuberculosis complex (MTBC) organisms and the host immune response, resulting in diverse clinical morphologies. Over recent decades, a decline in classical forms and a parallel rise in atypical and drug-resistant variants, particularly among immunocompromised individuals, have been noted. These trends highlight the need for renewed clinical

Corresponding author
Mohammed Atiqur Rahman, Professor, Department of Respiratory Medicine, Bangladesh Medical University, Dhaka, Bangladesh.
Email: m_a_rahman88@yahoo.com; Orchid ID: https://orcid.org/0000-0002-6960-9004

Cite this Article:
Rahman MA, Bhuiyan MSI, Ferdousi KR, Chakrabortty R. Revisiting Cutaneous Tuberculosis: Clinical Patterns, Diagnostic Challenges and Therapeutic Perspectives —A Narrative Review. Ban Acad Dermatol. 2025; 05 (02): 8-14

Available at: www.jbadbd.com
An official publication of Bangladesh Academy of Dermatology (B.A.D.)

vigilance and early recognition. This narrative review aims to synthesize current evidence on the epidemiology, clinical spectrum, diagnostic strategies, and therapeutic approaches of CTB, with particular emphasis on global–regional prevalence patterns and emerging molecular and dermoscopic advances that enhance diagnostic precision.[2]

Epidemiology and Pathogenesis

Cutaneous tuberculosis (CTB) prevalence varies geographically, being more common in TB-endemic regions of Asia, Africa, and Latin America. The burden correlates closely with the incidence of systemic tuberculosis and socioeconomic determinants such as overcrowding, malnutrition, and limited access to healthcare.[3]

Risk factors include poor socioeconomic conditions, HIV co-infection, diabetes mellitus, and use of immunosuppressive therapies (e.g., corticosteroids, anti-TNF agents). HIV infection markedly increases susceptibility by impairing cell-mediated immunity, leading to atypical, multifocal, and disseminated CTB presentations. Diabetes mellitus compromises macrophage and neutrophil function, prolonging disease course and delaying wound healing. Similarly, immunosuppressive therapy—used in autoimmune diseases or post-transplantation—reduces granuloma formation and promotes reactivation of latent foci, thereby modifying both clinical pattern and treatment response.[4]

The skin may be infected exogenously (through direct inoculation following trauma or minor abrasions) or endogenously (via contiguous spread from underlying structures, hematogenous dissemination, or lymphatic extension). The host's immune status plays a pivotal role in determining disease expression—ranging from localized nodular lesions in individuals with robust immunity to disseminated ulcerative or verrucous lesions in immunocompromised hosts.

Mycobacterium tuberculosis remains the predominant pathogen. However, M. bovis—usually transmitted through unpasteurized dairy products or direct contact with infected cattle—can produce similar cutaneous lesions but may show resistance to pyrazinamide, a key anti-TB drug. This distinction has important diagnostic and therapeutic implications, necessitating culture-based species identification and drug susceptibility testing. Atypical mycobacteria (e.g., M. marinum, M. ulcerans) can mimic CTB but are typically associated with water exposure and require different antibiotic regimens.[5]

Histopathologically, CTB is characterized by granulomatous inflammation with central caseation necrosis, reflecting a delayed-type (Type IV) hypersensitivity reaction mediated by Th1 lymphocytes and macrophage activation.

Figure 1. Pathogenesis and Routes of Infection in Cutaneous Tuberculosis[6,20]

Route of infection	Exogenous inoculation	Contiguous spread	Lymphatic spread	Hematogenous dissemination
Source	Contaminated material or lesion	Underlying organ or lymph node	Regional lymph nodes	Systemic TB focus
Mechanism of spread	Direct entry through trauma, injection, or minor abrasions	Extension through dermis or subcutaneous tissue	Retrograde lymphatic extension	Bloodstream spread during active or latent infection
Typical clinical form	Localized lesion at inoculation site	Ulcerative or scrofuloderma-type lesion	Linear or nodular lesions along lymphatics	Multiple papulonecrotic or disseminated lesions
Example	*Tuberculous chancre, warty tuberculosis*	*Scrofuloderma*	*Tuberculosis cutis lymphangitica*	*Miliary CTB, lupus vulgaris*

Classification & Clinical Spectrum[6,7]

CTB demonstrates a wide clinical spectrum. It is traditionally classified based on the route of infection and immune response:

1. Exogenous Infection Forms

Tuberculosis chancre: Primary inoculation TB in previously unexposed individuals; begins as a painless papule progressing to an ulcer with regional lymphadenopathy.

Warty tuberculosis (tuberculosis verrucosa cutis): Results from reinfection in previously sensitized individuals, typically over extremities; lesions are verrucous, indurated plaques with central clearing.

2. Endogenous Infection Forms

Lupus vulgaris: The most common form; arises by hematogenous or lymphatic spread. Lesions are soft reddish-brown papules ("apple jelly" nodules on diascopy) that progress slowly, leading to atrophic scarring or carcinoma transformation.[20]

Scrofuloderma: Occurs by contiguous spread from underlying lymph nodes, bones, or joints. Presents as subcutaneous nodules that ulcerate and form sinuses with caseous discharge.

3. Haematogenous forms

Tuberculous gumma (metastatic tuberculous abscess): Results from hematogenous dissemination, often in immunocompromised patients; manifests as cold abscesses with undermined edges.

Miliary tuberculosis of the skin: A rare disseminated form, usually in severe systemic disease or immunodeficiency.

4. Tuberculids (Hypersensitivity Reactions)

Lichen scrofulosorum, papulonecrotictuberculid, and erythema induratum of Bazin represent hypersensitivity responses to distant foci of TB without demonstrable bacilli in lesions.

Diagnostic Challenges

Diagnosing CTB remains difficult due to its rarity, polymorphism, and resemblance to other granulomatous dermatoses such as leprosy, deep fungal infections, and sarcoidosis.[8]

1. Clinical Diagnosis

A detailed history (TB exposure, BCG vaccination, systemic symptoms) and lesion morphology guide suspicion. Chronicity, scarring, and association with lymphadenitis are clues.

2. Histopathology

Biopsy typically reveals tuberculoid granulomas with Langhans giant cells and caseous necrosis. However, histological overlap with other conditions is common.

3. Microbiological

Ziehl-Neelsen staining and culture (Lowenstein–Jensen medium or MGIT system) confirm diagnosis but have limited sensitivity (20–60%).[9]

PCR-based assays (GeneXpert MTB/RIF, multiplex PCR) have greatly improved detection rates and allow identification of rifampicin resistance.

4. Molecular Techniques

Interferon-gamma release assays (IGRA) and tuberculin skin tests (TST) support diagnosis but cannot differentiate active from latent infection.[10]

5. Imaging

Useful in scrofuloderma or deep lesions to assess contiguous spread.

Differential Diagnosis

Includes atypical mycobacteriosis, deep fungal infections (sporotrichosis, chromoblastomycosis), sarcoidosis, syphilis, and leishmaniasis. Correlation of clinical, histological, and molecular findings is essential.[11]

Therapeutic Perspectives

1. Antitubercular Therapy (ATT)

The cornerstone of Cutaneous tuberculosis (CTB) management is standard anti-TB therapy as per WHO and national guidelines. Treatment typically follows the same regimen used for pulmonary tuberculosis.

Table 1: Standard Antitubercular Regimens for CTB[6,7,12]

Type of TB	Drug-susceptible CTB	Extended regimen (for bone, joint, or disseminated CTB)	Drug-resistant / MDR CTB
Drugs Used	Isoniazid (H), Rifampicin (R), Pyrazinamide (Z), Ethambutol (E)	Same as above	Fluoroquinolone (e.g., levofloxacin or moxifloxacin), Linezolid, Clofazimine, Cycloserine, Bedaquiline (as per DST results)
Phases	**Intensive phase:** HRZE **Continuation phase:** HR	HRZE → HR	Individualized
Duration	2 months 4 months	2 + 7 months	9–12 months (may extend up to 18 months in refractory cases)
Notes	All four drugs daily	Total 9 months	Requires specialist supervision

2. Monitoring for side effect[13,17]

Isoniazid: Hepatotoxicity, peripheral neuropathy → give pyridoxine (vitamin B6) 10–25 mg/day.

Rifampicin: Hepatotoxicity, orange discoloration of urine, drug interactions.

Pyrazinamide: Hepatotoxicity, hyperuricemia → monitor liver enzymes and uric acid.

Ethambutol: Optic neuritis → perform baseline and periodic visual acuity/color vision tests.

Fluoroquinolones/Linezolid: Tendinopathy, myelosuppression, neuropathy in MDR regimens.

Monitoring Protocols:

Baseline: Liver function tests (LFT), renal profile, CBC, and vision screening.

Monthly follow-up: Clinical review for side effects and adherence; repeat LFTs if symptoms or risk factors are present.

3. Role of DOTS (Directly Observed Treatment, Short-course):

Ensures treatment adherence and completion, reducing risk of relapse or drug resistance.

Facilitates early detection of adverse reactions and prompt management.

Strengthens community-based TB control through patient support and counseling.

4. Role of Surgical Intervention[1]

Surgical management in CTB is adjunctive, not curative. It complements medical therapy when indicated for:

- Diagnostic confirmation (biopsy or excision for histopathology).
- Drainage of cold abscesses or sinus tracts.
- Debridement or excision of fibrotic, ulcerative, or non-healing lesions unresponsive to adequate ATT.
- Reconstructive procedures to manage cosmetic or functional deformities after disease resolution.

5. Adjunctive Therapies[14]

Topical corticosteroids and immunomodulators may aid in tuberculid management.

Nutritional support and management of comorbidities (especially HIV and diabetes) improve outcomes.

Recent Advances and Future Directions

The advent of molecular diagnostics and whole-genome sequencing has refined the understanding of M. tuberculosis strain diversity and drug resistance. Dermoscopic features of lupus vulgaris and scrofuloderma are being increasingly characterized toaid early noninvasive diagnosis. Novel biomarkers such as lipoarabinomannan (LAM) antigen detection and host gene expression profiles may soon complement traditional tests.[15,16]

The integration of dermatology, infectious disease, and public health approaches remains vital. Community awareness and early referral, particularly in resource-limited settings, are key to preventing chronic scarring and deformity.[18,19,20]

Conclusion

Cutaneous tuberculosis, though rare, remains a clinically significant and diagnostically challenging manifestation of Mycobacterium tuberculosis infection. Awareness of its polymorphic presentations, judicious use of histopathology and molecular diagnostics, and strict adherence to treatment regimens are essential for optimal patient outcomes. Renewed attention to this neglected entity is necessary, especially in TB-endemic regions where under diagnosis persists. "Strengthening integrated TB–dermatology networks and surveillance can enhance early recognition and reduce disease burden in endemic countries."

Declarations

Ethics Approval: Not applicable (narrative review).
Consent to Participate: Not applicable.
Consent for Publication: Not applicable.
Funding: No external funding received.
Conflict of Interest: The author declares no conflict of interest.
Clinical Trial Number: Not applicable.

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