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**Review Article**

# Cutaneous tuberculosis-management

**Adithyan Panchatsharam[1]*, Revathy Sundaramurthy[2]**

[1]Department of DVL, Dr B R Ambedkar Medical College, Bangalore, Karnataka, India
[2]Department of Paediatrics, Akash Institute of Medical Sciences and Research Centre, Devanahalli, Karnataka, India

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***Correspondence:**
Dr. Adithyan Panchatsharam,
E-mail: adithya2988@gmail.com



## ABSTRACT

Since antiquity, people have been plagued by tuberculosis (TB), and its effects are likely older than recorded history. For many centuries, TB was the most significant human infection due to its devastating mortality and morbidity rates and global prevalence. In developed nations, there is less evidence of *Mycobacterium tuberculosis* infections as a result of improved socioeconomic status. In developing nations like India, it is still regarded as a significant cause of morbidity and mortality. Cutaneous TB occurs worldwide and it comprises only a small proportion of all cases of TB, but considering the high prevalence of TB in many developing countries like India these numbers become significant. Cutaneous TB is a form of extra pulmonary TB and is characterized by a spectrum of multiple distinct clinical and histopathological presentations. It is imperative that the treating dermatologist is aware of the varied manifestations of cutaneous TB which would significantly prevent the morbidity and complications. This review aims to discuss the epidemiology, clinical, histopathological features, diagnosis, differential diagnosis and treatment options, especially in the Indian context.

**Keywords:** Cutaneous TB, Lupus vulgaris, Scrofuloderma, tuberculid, TB verrucosa cutis

## INTRODUCTION

The infectious disease TB is a significant cause of death from a single infectious agent (ranked above HIV/AIDS), one of the top 10 causes of death worldwide, and a major contributor to poor health. Mycobacterium TB, the bacillus that causes TB, spreads when TB patients discharge bacteria into the air, such as when they cough. The disease typically affects the lungs (pulmonary TB) but can also affect other sites (extra pulmonary TB). A quarter of the world's population has *M. tuberculosis* infection.[1]

Around 0.9% of patients who visit the dermatology OPD have cutaneous TB, an extra pulmonary TB variant. Cutaneous TB can manifest clinically in a variety of different yet distinct ways in both adults and children. This review's goal is to discuss about cutaneous TB's clinical characteristics, diagnosis, and available treatments in the context of India.

## EPIDEMIOLOGY

In 2019, there were an estimated 10.0 million (range, 8.9-11.0 million) TB cases worldwide. According to estimates, 1.2 million (1.1-1.3 million) HIV-negative persons died from TB in 2019 (down from 1.7 million in 2000), while 2,08,000 (177000-242000) HIV-positive people also died from the disease (down from 678 000 in 2000). Men (aged >15 years) made up 56% of those who contracted TB in 2019; women (32%); and children (aged <15 years) accounted for 12%. 8.2% of all those impacted by the incident were HIV positive. A total of eight nations-India (26%), Indonesia (8.5%), China

(8.4%), the Philippines (6%) Pakistan (5.7%), Nigeria (4.4%), Bangladesh (3.6%), and South Africa (3.6%)-accounted for two thirds of the global total.

Drug-resistant TB is still a danger to the public's health. Nearly 500,000 persons developed rifampicin-resistant TB (RR-TB) in the world in 2019, of which 78% had MDR-TB. India (27%), China (14%) and the Russian Federation (8%), three nations, carried the lion's share of the global burden. 3.3% of new cases of TB and 17.7% of those that had already been treated developed MDR/RR-TB globally in 2019.[1]

## CLASSIFICATION BASED ON ROUTE OF INFECTION[2]

**Table 1: Classification.**

| | |
|---|---|
| **Exogenous source** | **Tuberculous chancre** |
| | Warty TB |
| | Lupus vulgaris |
| **Endogenous source** | |
| Contiguous spread | Scrofuloderma |
| Autoinoculation | Orificial TB |
| Haematogenous | Military TB |
| | Lupus vulgaris |
| | Tuberculous gumma |
| | Tuberculids |
| | Metastatic tuberculous abscess |

## SCROFULODERMA

Before the development of tuberculostatic medications, scrofuloderma, also known as TB colliquativa cutis, was the most frequently observed form of cutaneous TB. It is still the most prevalent form of cutaneous TB in developing nations like Brazil and India (especially in children).[3]It may affect anyone of any age, but children, teenagers, and the elderly are the most likely to be impacted because they are the life stages during which the majority of immunologic failures in containing infectious pathogens occur.[4,5] Scrofuloderma results from the contiguous spread to the overlying skin from adjacent structures such as lymph node, joint, bone or the epididymis. The neck, axillae, or groin are most frequently affected. It appears clinically as a firm subcutaneous lump that enlarges, confluents, ulcerates, and develops sinus tracts that drain caseous material.[6] The ulcers typically have bluish edges and are shallow. Scrofuloderma typically leaves behind scars that are cribriform, bridging, and puckered.[7]

By performing a culture, it is necessary to rule out non-tuberculous mycobacterial infections, particularly *M. avium* complex lymphadenitis and the benign *M. scrofulaceum*. Sporotrichosis, actinomycosis, syphilitic gummata, hidradenitis suppurativa, meliodosis, and bacterial abscess are additional differential diagnoses.

### *HPE*

Scrofuloderma usually appears as an ulcerated dermal abscess with an ill-defined histiocytic component. Peripheral granulomata may be present. Marked caseation necrosis in which bacilli may be numerous can be seen in deeper tissues.[8]

## LUPUS VULGARIS (TB LUPOSA)

A person with a moderate to high level of immunity may develop the chronic, progressive, post primary, paucibacillary form of cutaneous TB known as lupus vulgaris. The most prevalent type of cutaneous TB in adults in South Africa and the Indian subcontinent is lupus vulgaris.[9] Women are affected 2-3 times more frequently than men for unidentified reasons.[10] The most common routes for infection are continuity, lymphohematogenous routes, and rarely exogenous routes.[11,12] Although lesions are typically solitary, they can occur simultaneously at two or more sites. Multiple foci may form in TB patients with active disease.[13] The most distinctive clinical feature is a papulo-tuberous lesion with slow evolution, which may invade mucosae and coalesce into a plaque.[14] In India buttocks and thighs are involved. Commonly manifests as a solitary lesion, though it can also happen at the site of a primary inoculation, like after getting a tattoo or where the BCG vaccine was administered.[15] The typical appearance by diascopy is referred to as "apple jelly nodules".[14]

The clinical forms fall into five different general patterns, depending on the local tissue response to the infection: In plaque form, the lesions have irregular or serpiginous edge and large plaques show irregular areas of scarring with islands of active lupus tissue. The edge often becomes thickened and hyperkeratotic. In ulcerative and mutilating forms, scarring and ulceration predominate with crusts forming over areas of necrosis. The deep tissues and cartilage are invaded and contractures and deformities can occur. Vegetating form is characterized by marked infiltration, ulceration and necrosis with minimal scarring. Mucous membranes are invaded and cartilage is slowly destroyed. When the nasal or auricular cartilage is involved, extensive destruction and disfigurement occurs. Tumour-like forms, present either as soft-tumour-like nodules or as epithelial hyperplasia with the production of hyperkeratotic masses. In papular and nodular forms, multiple lesions occur simultaneously in disseminated lupus-true “miliary lupus” and usually occur after immunosuppression.[9]

Constitutional symptoms are generally absent or very mild and systemic involvement is less commonly seen as compared to scrofuloderma. Regional lymphadenopathy is frequently associated with LV. [16-19]

Few challenges arise from the well-established plaque with central scarring, but in the early stages, lupus vulgaris can be mistaken for lymphocytoma, Spitz

naevus, or lupus erythematosus. The deep mycoses, which may closely resemble the vegetating and crusted type of lupus vulgaris, can be distinguished by histological characteristics and culture findings. Clinically, it might be impossible to distinguish leishmaniasis in its lupoid form. It could be mistaken for rosacea or a port wine stain on the face, and for other mycobacterial infections on an extremity.

The two most common causes of diagnostic difficulty are leprosy and sarcoidosis. Leprosy nodules are firmer, and there are additional symptoms. Sarcoidosis nodules feel more like grains of sand than "apple jelly"; this is true of the feel during probing rather than the colour, which is frequently greyish.

Lupus vulgaris may resemble psoriasis, but is more infiltrated and usually solitary. Lupus vulgaris of the perianal area mimics lichen simplex chronicus and Crohn. Warty TB usually affects the hand and is more scaly and verrucous than lupus and the histological features are different. [15]

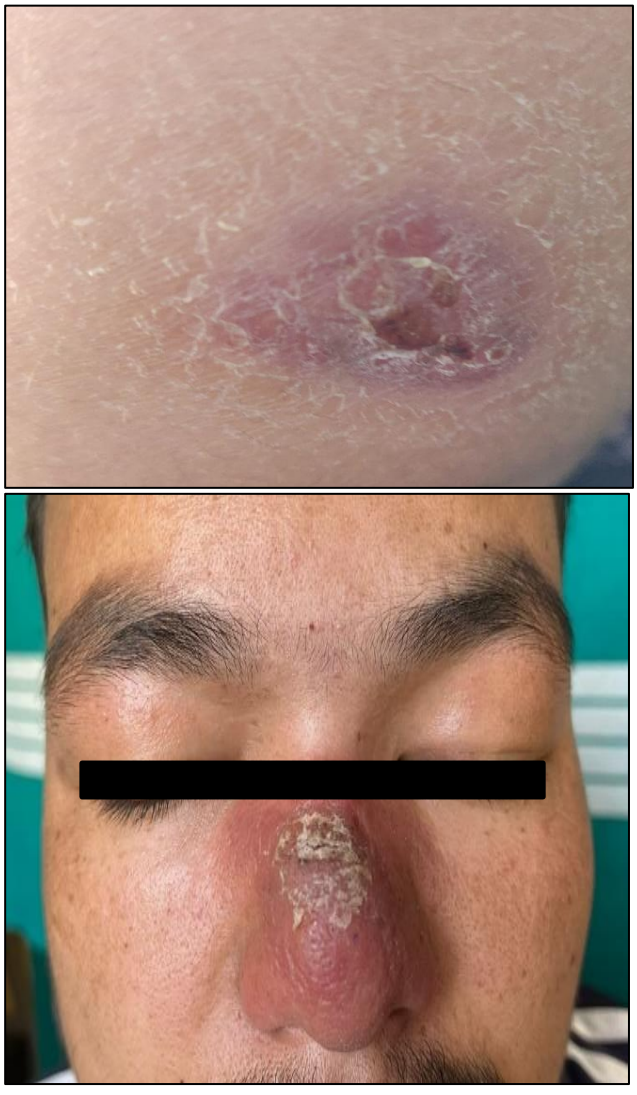

**Figure 1: Lupus vulgaris.**

***HPE***

The typical histopathology consists of tuberculoid granulomas composed of Epitheloid granulomas and giant cells in superficial dermis. Caseation necrosis within the tubercles is slight or absent. Giant cells are of the Langhans type or foreign body type.[20] Peripheral lymphocytes and plasma cells are also prominent. The overlying epidermis may be ulcerated, atrophic or acanthotic. [8] In areas of healing extensive fibrosis may be seen. [20]

## TB VERRUCOSA CUTIS

TBVC is also referred to as post-mortem wart, prosector's wart, butcher's wart, and warty TB. TBVC is probably the third most prevalent type of lupus in India, behind scrofuloderma and lupus vulgaris.[21] Exogenous reinfection (inoculation) in previously sensitized people with moderate to high immunity results in TVC, a paucibacillary disorder.[13] In adults, TBVC typically affects the hands, while in kids, it typically affects the lower extremities. Frequent barefoot walking raises the risk of developing TBVC on the sole.[22]

It manifests as painless, single or multiple, slowly evolving, spontaneously involuting verrucous and tuberous papules without adenopathy. Due to the possibility of self-inoculation, it can be classified as an occupational disease. For example, a dentist treating the mouth of a patient with pulmonary TB or a butcher handling contaminated meat (the latter is typically the result of *M. bovis* infection) may both contract it.[14] The main TBVC variants include psoriasiform, keloidal, crusted, exudative, sporotrichoid, destructive, tumour-like, and exuberant granulomatous forms.[23]

Subungual and digital lesions must be distinguished from warts, and those on the hands from keratoses. Blastomycosis, chromoblastomycosis and actinomycosis may simulate exuberant forms and crusted lesions may resemble leishmaniasis. When the central scarring is surrounded by a serpiginous edge, secondary syphilis may be mistaken for it. Hypertrophic lichen planus and lichenification occasionally cause difficulty, but lesions of these disorders are multiple or itchy. In most cases, lupus vulgaris does not exhibit hyperkeratosis and diascopy reveals "apple jelly" nodules. Microbiological culture is typically the only method available to distinguish lesions brought on by NTM. *M. marinum* presents unique challenges.[15]

***HPE***

TVC is characterized by acanthotic papillomatosis with marked hyperkeratosis. Pseudoepitheliomatous hyperplasia may be seen. The dermal infiltrate consists of mainly neutrophils and lymphocytes, abscesses may sometimes be present. Granulomata may be present in the

deeper dermis and caseation is occasionally a feature. Bacilli may be seen. [8]

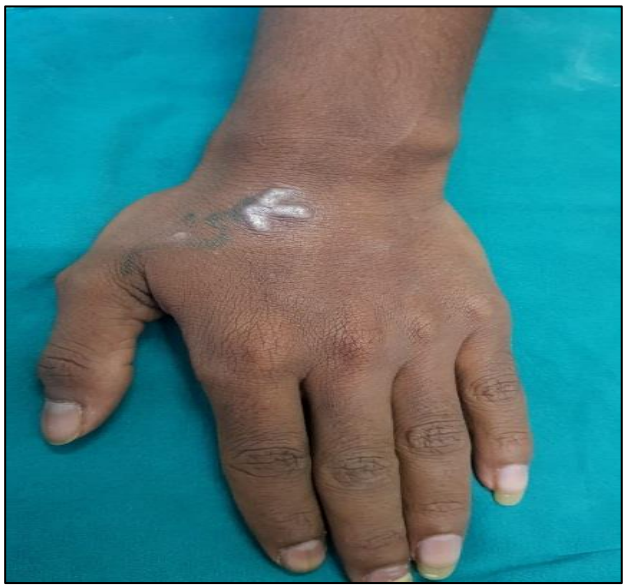

**Figure 1: TB verrucosa cutis.**

## TUBERCULOUS GUMMA

The multibacillary form of skin TB known as tuberculous gumma, also known as metastatic tuberculous abscess, is brought on by the hematogenous spread of mycobacteria from a primary focus.[24] It is typically described in severely immunosuppressed adults and malnourished children, but it can also occur in immunocompetent people.[25]A single or multiple soft, dermal, or subcutaneous nodules that soften to form fluctuant, non-tender abscesses and later degenerate to form ulcers or sinuses with frayed edges are considered the typical lesion. It might resemble scrofuloderma clinically.[7]

They can be difficult to differentiate from pyogenic bacterial infections, syphilis, non tuberculous mycobacterial infections, pyoderma gangrenosum and some fungal infections.[15]

### *HPE*

Most of the lesion of tuberculousgumma consists of caseation necrosis with a rim of epitheloid cells and giant cells. Bacilli are scanty but can be demonstrated in histologic sections.[20]

## TB CUTIS ORIFICALIS

Patients with advanced internal TB may develop the extremely rare cutaneous TB known as TB cutis-orificialis (TCO), which is brought on by mycobacterial auto-inoculation. TCO develops in adjacent skin as well as oral, perianal, or genital mucosa. The oral mucosa, particularly the tongue, is where TCO is most frequently found.[26] Haematogenous spread, lymphatic spread, direct spread from adjacent organs, and ingestion of bacilli in sputum from active pulmonary TB are proposed mechanisms leading to TCO. The exact mechanism causing TCO is still unknown.[27]

TCO typically manifests clinically as erythematous, oedematous plaques or nodules. This is followed by painful central ulceration covered by necrotic, pseudomembranous materials with an irregular border constitutional symptoms like fever, malaise, weight loss, and night sweats are also present in the majority of patients.[25]

Oral lesions need to be differentiated from Crohn disease, mucocutaneous leishmaniasis, oral paracoccidiomycosis and rhinoscleroma. Anogenital lesions must be differentiated from malignancy, nicorandil induced ulceration, Crohn disease, cutaneous amoebiasis, anal paracoccidiomycosis, chronic herpes simplex infection and syphilis.[15]

### *HPE*

Orificial lesions show extensive necrosis and numerous bacilli. The inflammatory infiltrate consists of lymphocytes, neutrophils with few histiocytes. [8]

## ACUTE MILIARY TB

It is a severe and uncommon form of TB that mostly affects young children, infants, and immunocompromised adults. Fever, anorexia, asthenia, and weight loss are signs of systemic compromise in the affected people.[28] Clinically, discrete pin head sized bluish red papules capped by tiny vesicles with cloudy and purulent content is seen. Vesicles later burst or dry out and form a crust. Since the seriously ill patient is unable to respond to the antigens, the tuberculin test is negative. Commonly, lesions exhibit a neutrophilic infiltrate, macrophages with numerous AFB, and later formation of granulomas.[2]

### *HPE*

In military TB center of the papule shows a micro abscess containing neutrophils, cellular debris and numerous tubercle bacilli. This is surrounded by a zone of macrophages and occasional giant cells.[20]

## TUBERCULIDS

Tuberculids can be considered to be cutaneous hypersensitivity reactions to haematogenous dissemination of *M. tuberculosis* or its antigens from a primary source in an individual with strong antituberculous cell-mediated immunity. The diagnostic criteria include tuberculoid histology on skin biopsy, a strongly positive Mantoux reaction, the absence of *M. tuberculosis* in the smear and negative culture and resolution of the skin lesions with antituberculous therapy. [15]

## LICHEN SCROFULOSORUM

In children and adolescents with TB, lichen scrofulosorum, also known as " TB cutis lichenoides," manifests as a lichenoid eruption of small papules.[29] It is usually associated with chronic TB of the lymph nodes, bones, or pleura, but it has also been seen in connection with *M. avium* intracellulare infections and after BCG vaccination.[13]

The skin-coloured to reddish-brown papules, which are typically asymptomatic, closely clustered, and often perifollicular, are most frequently found on the abdomen, chest, back, and proximal parts of the limbs. The eruption is typically accompanied by a strong tuberculin reaction.[30] Lesions persists for months, but spontaneous involution eventually occurs. Within weeks of starting anti TB treatment, the condition is fully resolved.[13]

Differential diagnosis includes all asymptomatic follicular lesions where the lesions demonstrate a tendency to group together. These include: lichen nitidus, in which the lesions are more shiny and tend to be peripheral; keratosis spinulosa, in which the lesions have spiny projections over lichenoid papules; keratosis pilaris, where the lesions are non inflammatory and usually on the upper thighs and arms; and papular or lichenoid sarcoidosis, secondary syphilis and drug eruptions. Annular lesions may closely resemble pityriasis rosea.[15]

### *HPE*

In Lichen scrofulosorum superficial dermal granulomas are usually seen in vicinity of hair follicles or sweat ducts. The granulomas are composed of epitheloid cells with some Langhan giant cells and a narrow margin of lymphoid cells at the periphery. Caseation necrosis is absent. Granulomas replacing hair follicles is a characteristic feature of lichen scrofulosorum.[20]

## PAPULONECROTIC TUBERCULID

Even in nations where TB is highly prevalent, papulonecrotic tuberculid is the least frequent type of cutaneous TB. It is deemed to be a type III hypersensitivity reaction to the presence of a focus of infection elsewhere in the body. [31] It manifests as symmetrical, painless erythematous or violaceous papulonodular lesions that develop in bouts and leave depressed scars (varioliform or punched-out), most commonly on the dorsal surfaces of children's and young adults' hands, buttocks, and extensor surfaces of their legs and forearms.[14] Skin symptoms may be preceded by constitutional symptoms like fever and asthenia.[32]

Differential diagnosis includes: pityriasis lichenoides, where the lesions may be more widespread and affect the palms and soles, leukocytoclastic vasculitis, whose lesions are more pleomorphic and nodular prurigo.[15]

### *HPE*

Fully developed lesion of papulonecrotictuberculid shows cutaneous infarction comprising a necrotic epidermis with ulceration and an underlying V shaped zone of coagulativenecrosis accompanied by a dense coagulative necrosis with scattered giant cells. Well-formed granulomata can be present in older lesions but bacilli cannot be identified.[8]

## ERYTHEMA INDURATUM OF BAZIN

Young and middle-aged women who have TB are susceptible to the granulomatous lobular panniculitis known as erythema induratum of Bazin (EIB), which affects the lower limbs.[33] The disease typically affects the back of the lower legs and manifests as an indolent eruption of poorly defined nodules or subcutaneous plaques. The plaques have an indurated surface that is scaly.[13] Even without treatment, lesions may go away after a few weeks to months, leaving behind depressed post-inflammatory hyperpigmentation and scarring. Recurrences, however, are possible in flare-ups every three to four months.[28]

The differential diagnosis includes other conditions presenting as nodules on the lower extremities such as erythema nodosum, pancreatic panniculitis, polyarteritis nodosa, lupus profundus, subcutaneous sarcoid, cutaneous T cell lymphoma, perniosis (chilblains).[15]

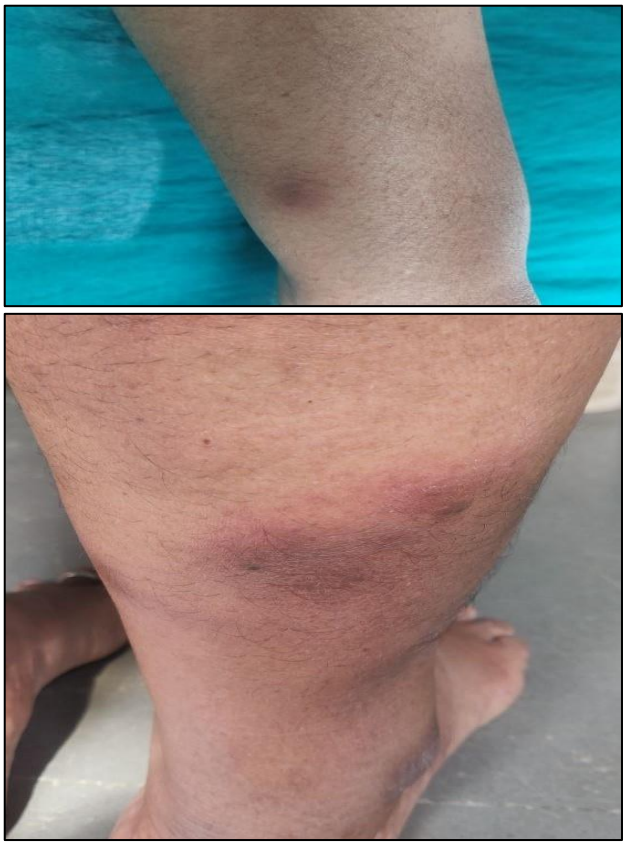

**Figure 3: EIB.**

***HPE***

In erythema Induratum there is predominantly lobular panniculitis in association with tuberculoid granulomas, areas of caseation necrosis and variable vascular involvement of mainly venules and small to medium calibre arteries including vasculitis [8]

## CUTANEOUS TB SECONDARY TO BCG VACCINATION

BCG vaccine is a live virus vaccine derived from attenuated strains of *M. bovis*. It has been widely used to prevent serious TB infections, such as meningoencephalitis and acute miliary TB. However, it may cause skin complications like tuberculids, lupus vulgaris, scrofuloderma and even outbreaks of TB in other organs, and nonspecific reactions, such as fever, local inflammation, abscesses, and lymphadenitis.[34,35]

## DIAGNOSIS

Evaluation of suspected cutaneous TB requires history and examination along with a full workup. The workup may include the demonstration of AFB on Ziehl–Neelsen (ZN) stained smears or biopsies, TB skin test (TST) or Mantoux test, serum Quantiferon-TB Gold (QFT-G) levels, isolation by culture or detection by PCR, and skin biopsy. Differential diagnoses usually include atypical mycobacterium infections, deep fungal infections such as chromoblastomycosis and sporotrichosis, sarcoidosis, cutaneous leishmaniasis and leprosy.

## MICROSCOPY

Conventional microscopy using Ziehl-Neelsen staining is a rapid and cost-effective way of detecting tubercular bacilli but lacks sensitivity. Lower sensitivity is encountered in pediatric TB, extra pulmonary TB and in HIV-infected TB patients. Conventional fluorescence microscopy is more sensitive than Ziehl-Neelsen staining and takes less time. Light-emitting diodes (LED) have been developed to offerthe benefits of fluorescent microscopy without the associated costs. LED microscopy is more sensitive than conventional light microscopy and has a qualitative, operational and cost advantages over both conventional fluorescence and light microscopy.[36]

## CULTURE

Currently, liquid cultures like Mycobacterium growth indicator tube (MGIT) cultures are used and advised as the gold standard for diagnosis. Traditional solid cultures like LJ culture have been replaced by these relatively quick cultures. [37]

The BACTEC MGIT 960 culture system is a fluorescent signalling system for earlier detection of growth. MGIT has several advantages over the BACTEC 460 TB system (radiometric system). It provides an early recovery of *Mycobacterium*, i.e., within 10 days as compared to 24 to 28 days by conventional culture methods and drug susceptibility can be checked in shorter time span.

## ANTIGEN DETECTION METHODS

Tests that detect *M. tuberculosis* antigens in clinical specimens provide rapid direct evidence of infection. Lipoarabinomannan (LAM) is the antigen that is most frequently targeted.[36]

## IMMUNOLOGICAL METHODS

Immunological techniques like the QuantiFERON-TB gold (QFT) and Tuberculin Skin Test (TST) are primarily used for screening and excluding purposes.[38]

## MANTOUX TEST (TUBERCULIN SKIN TESTING)

Tuberculin skin test is an intra-dermal injection of PPD.It is an immunological test which elicits delayed type hypersensitivity. A positive test indicates present or past infection with mycobacterium TB but cannot distinguish infection from disease.

Current recommendation is to use 2TU PPD RT23 for all diagnostic purposes.Mantoux`s test or PPD skin test is considered positive if the induration is 10mm or more. In HIV co infected cases 5mm may be taken as cut off. [37]

***QuantiFERON-TB Gold***

A popular substitute for TST in developed nations is the interferon gamma (IFN-) release assay known as QuantiFERON-TB Gold (QFT) or IGRA, which measures cell-mediated immune response. The use of IGRA is justified in patients who are unlikely to undergo a TST reading, who have a history of exposure to MTB or HIV, or who already have compromised immune systems as a result of other medical conditions. MTB antigen coated tubes are used to collect blood samples, and the enzyme immunoassay (EIA) technique is used to measure IFN-y release.[38]

## MOLECULAR DIAGNOSIS AND DETECTION OF DRUG-RESISTANT MTB

Polymerase chain reactions (PCR), strand displacement amplification (SDA), transcription mediated amplification (TMA), reporter mycobacteriophage system, oligonucleotide ligation amplification, and Q-beta replicase amplification are just a few of the molecular techniques currently used for the diagnosis of mycobacterial diseases using nucleic acid probes and gene amplification techniques. A useful and affordable alternative method for quickly identifying active TB in various clinical specimens is polymerase chain reaction (PCR).[38]

## LOOP MEDIATED ISOTHERMAL AMPLIFICATION

The target region can be amplified quickly and effectively using the loop mediated isothermal amplification (LAMP) technique. It uses an auto-cycling strand displacement DNA synthesis process powered by a Bst DNA polymerase and can be done in a simple water bath. By adding 0.1% SYBR Green to the tube and analysing the colour of the solution under UV light, it is possible to see with the unaided eye the significant amount of DNA generated in less than an hour and the positive LAMP reaction. In the presence of a LAMP amplicon, the solution turns green; in the absence of amplification, it remains orange. LAMP has a number of benefits, including not requiring a thermocycler and being a quick, easy, and affordable method of the diagnosis in the environments with the limited resources.[36]

### Xpert MTB/RIF

A fully automated molecular test for detecting TB cases and determining drug resistance is called Xpert MTB/RIF (Xpert® MTB/RIF). Targeted nucleic acid sequences in the TB genome are purified, concentrated, amplified (by real-time PCR), and identified using the Xpert® MTB/RIF. The Xpert MTB/RIF uses a hemi-nested real-time polymerase-chain reaction (PCR) assay to detect *M. tuberculosis* (MTB) and rifampin resistance (RIF) by amplifying MTB-specific sequence of the rpoB gene, which is then probed with molecular beacons for mutations within the rifampin-resistance-determining region.[36]

## LINE PROBE ASSAY

The line probe assay (LPA) uses PCR amplification to detect the presence of *M. tuberculosis* and drug resistance by determining mutations, followed by hybridization on a strip immobilized with particular oligonucleotide probes. The commercial molecular line probe assays that have been most thoroughly studied are the (1) INNO-LiPA Rif.TB kit from Innogenetics in Zwijndrecht, Belgium, and (2) genotype MTBDR and genotype MTBDRplus assays from Hain Life science in Germany.[38]

## GENOTYPING

Genotyping, the recent advance in the diagnosis of cutaneous TB, has a tendency to separate atypical mycobacterium from Mtb-and detect mutant if it persists inducing drug resistance in the pathogen. The major molecular typing methods—Spoligotyping, MIRU-VNTR (*Mycobacterium* Interspersed repetitive unit-variable number tandem repeats), and RFLP-detect *Mycobacterium tuberculosis*, DNA, or RNA in clinical specimens by *in vitro* nucleic acid amplifications.[39]

## UREASE BREATH TEST FOR RAPID DIAGNOSIS OF TB

Metabolic pathway detection may offer quick and efficient new TB diagnostic tools that will benefit patients with HIV and children. The fact that metabolic breath tests are a quick and safe way to assess a drug's efficacy gives them advantages. For both primary diagnosis and treatment monitoring, the signal correlated with bacterial load. A useful diagnostic and biomarker assay for TB and treatment response may be provided by urea breath testing.[36]

## TREATMENT

All patients with the following results should be treated for cutaneous TB, Patients with histology diagnostic of Cutaneous TB, Patients with positive culture of *Mycobacterium* TB or microscopy for AFBs from skin biopsy. Patients with equivocal histology findings and negative microscopy and culture, but strongly positive Mantoux test.[40]

Cutaneous TB is treated as extra pulmonary TB. Intensive phase consists of HRZE (isoniazid, rifampicin, pyrazinamide, and ethambutol) given daily for 2 months followed by maintenance phase of 4 months of HRE (isoniazid, rifampicin, and ethambutol) daily.[7]

Response to treatment should be assessed at 4-6 weeks. Most patients will show significant improvement by this time. Failure to improve or deterioration may be due to misdiagnosis or drug résistance. Subsequent follow up of patients responding to treatment can continue at 8 weekly intervals until treatment is completed.[40]

WHO defines MDR-TB as resistance to isoniazid and rifampicin and XDR-TB as resistance to isoniazid, rifampicin along with any fluoroquinolone and one of the 3 second-line injectable drugs (amikacin, capreomycin, and kanamycin). Treatment of MDR-TB requires the use of second-line drugs. The treatment regimen usually consists of five or six drugs which include a fluoroquinolone and an injectable administered for 6 months followed by administration of four oral drugs for the next 18 months.[7]

Scarring caused by the initial infection and then healing of the skin can cause disfigurement. There is an increased risk of squamous cell carcinoma in patients with long standing untreated disease.[40]

## CONCLUSION

TB is a major health problem due to its high prevalence with high morbidity and mortality. It should be considered in patients presenting with atypical skin lesions suggestive of an infectious etiology. It is important that the dermatologist has a high index of

suspicion in order to quickly and effectively diagnose and treat these morbid skin lesions.

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