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Cutaneous tuberculosis: A great imitator

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Abstract Tuberculosis (TB) is still prevalent in many developing countries and can pose a new potential threat to global health due to international migration. As an uncommon form of extrapulmonary TB, cutaneous TB is complicated in its clinical manifestation, pathogenesis, and classification. Cutaneous TB can be divided into two major categories, true cutaneous TB and tuberculid, depending on the source of infection, the route of transmission, the amount of bacteria, and the immune state of the host. Clinical manifestations may include patches and plaques (lupus vulgaris, TB verrucosa cutis), macules and papules (acute miliary TB, papulonecrotid tuberculid, lichen scrofulosorum), nodules, and abscesses (erythema induratum of Bazin, tuberculous gumma), erosions, and ulcers (tuberculous chancre, orificial TB, scrofuloderma), mimicking diverse skin diseases. Uncommon localizations such as external genitalia, unusual presentations such as nodular granulomatous phlebitis, and coexistence with other morbidities such as Behçet disease and acne inversa or hidradenitis suppurativa deserve special attention. Treatment of both true and tuberculid cutaneous TB follows the same drug regimens of the World Health Organization's recommendation for treatment of new cases of pulmonary TB. Erythema induratum of Bazin may need longer treatment duration and adjuvants such as dapsone, potassium iodide, doxycycline, and corticosteroids to tackle inflammation. Misdiagnosis and undertreatment in daily practice are likely, and contemplation of this classic great imitator in dermatology is warranted.

Introduction

Tuberculosis (TB) is an ancient disease that is caused by *Mycobacterium tuberculosis* and mainly affects human beings. With the pandemic of HIV infection and the emergence of antimicrobial resistance against *M tuberculosis,* TB has reemerged in recent decades and represents a growing threat to public health and economic growth.[1] According to the World Health Organization (WHO), TB is the ninth leading cause of death worldwide and the leading cause as a single infectious agent, ranking above HIV and AIDS.[2] The incidence rates of TB vary widely among countries. Developing countries have the greatest burden of this disease, whereas China, India, and Indonesia alone accounted for 45% of global cases in the newly issued global TB report.[2] Pulmonary TB is the most common type, accounting for more than 80% TB cases. About 20% of TB cases are extrapulmonary, including TB lymphadenitis, pleural TB, TB meningitis, osteoarticular TB, genitourinary TB, abdominal TB, cutaneous TB (CTB), ocular TB, TB pericarditis, and breast TB.[1,3] The incidence of extrapulmonary TB has a much stronger relationship with HIV infection, emergence of multidrug-resistant TB, and immunosuppressive treatment.[4] Only 1% to 2% of all extrapulmonary TB cases shows cutaneous involvement.[5,6]

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CTB is a relatively uncommon disease. The disease's prevalence increased dramatically during the 17th and 18th centuries and declined in the 19th and early 20th centuries, as Bacillus Calmette-Guerin (BCG) vaccine and improved therapeutic approaches effectively reduced the incidence of TB worldwide. TB has become a major health concern again in recent decades, with CTB being a threat, especially in areas where TB prevalence, HIV infection, and multidrug-resistant TB remain high.

Pathogenesis

CTB was first described by Theophile Laennec (1781-1826) in 1826, before *M tuberculosis* bacillus was discovered and isolated by Robert Koch (1843-1910) in 1882.[5] CTB is mainly caused by *M tuberculosis* bacillus, and rarely by *M bovis* or BCG.[7] The main components of *M tuberculosis* are proteins, polysaccharides, and lipoids. The acute skin reaction to the tuberculin is caused by polysaccharides. Proteins are the most important antigens of *M tuberculosis* and can induce the T-cell immune response and other allergic reactions, including late-onset hyperreactive cellular immune response. Lipoids induce caseous necrosis of TB lesions.[8] The bacilli of CTB are transmitted through direct inoculation or lymphatic or hematogenous dissemination.[7] After infection, macrophages in the infected tissues are first activated to eliminate the bacilli at the infected site. Monocytes, lymphocytes, neutrophils, and dendritic cells are then recruited by the chemokines and cytokines released by macrophages. With recruitment and activation of T lymphocytes, the entire immunologic cascade is terminated with the formation of granulomas.[9] The immune response of CTB is analogous to that of systemic TB. In addition to the direct immune response to the bacilli, there is also a hyperactive immune response to form tuberculid lesions without locally residing bacilli.

The host characteristics, the load and pathogenicity of the bacillus strain, and the method of infection determine the clinical outcome of CTB. Malnutrition, poor health state, use of immunosuppressive drugs, and HIV infection are risks for active disease.[10] Age and sex are also host factors contributing to the varied manifestations of different subtypes of CTB; whereas scrofuloderma is mainly seen in children, TB verrucosa cutis is male predominant and erythema induratum of Bazin (EI) is female predominant.[11–13] According to the bacterial load in the lesion, CTB can be multibacillary or paucibacillary.[14] Considering the bacterial source and infection route, CTB can be endogenous or exogenous.[15] The endogenous mechanism is secondary to an already existing infection of the body, and transmission occurs via contiguous, lymphatic, or hematogenous dissemination. Exogenous mechanism refers to direct inoculation of bacilli into the skin of susceptible individuals.

Classification and manifestations

Classification of CTB is complex and inconsistent. Two primary categories are most widely accepted: true CTB and tuberculids (Table 1).[6,15–17] If *M tuberculosis* can be found at the lesional sites with common testing tools such as smear, culture, or polymerase chain reaction (PCR) examination, it is defined as true CTB. True CTB can be further divided into:

- exogenous TB caused by inoculation (tuberculous chancre and TB verrucosa cutis)
- endogenous TB due to direct spread or auto-inoculation (scrofuloderma, orificial TB, and some cases of lupus vulgaris) and hematogenous transmission (lupus vulgaris, tuberculous gumma, and acute military TB [MTB])

Tuberculids are defined as not being caused by *M tuberculosis* directly but rather induced by a hypersensitivity reaction to the bacterial antigens, which include mainly papulonecrotic tuberculid (PNT), lichen scrofulosorum (LS), and EI.[11,13,14]

The clinical manifestations of CTB are varied and complicated, which can mimic diverse skin conditions and make the diagnosis a great challenge to dermatologists in daily practice. In a study from China, the misdiagnosis rate has been reported

Table 1 Classification of cutaneous tuberculosis (CTB)

Classification	Infection source and pathogenesis	Subtypes
True CTB	Exogenous	Tuberculous chancre/primary inoculation tuberculosis
		Tuberculosis verrucosa cutis
	Endogenous	Scrofuloderma
		Orificial tuberculosis
		Lupus vulgaris
		Tuberculous gumma
		Acute miliary tuberculosis
Tuberculids	Hypersensitivity reaction to the bacterial antigens	Papulonecrotic tuberculid
		Lichen scrofulosorum
		Erythema induratum of Bazin

to range from 33% to 50%,[18] caused by the infrequent identification of the bacillus by common testing tools and the atypical morphology of the varying lesions. We shall present the diverse clinical manifestations of CTB and their differential diagnoses.

Patches and plaques in CTB

Patches and plaques in CTB are mainly observed in lupus vulgaris and TB verrucosa cutis.[19] Differential diagnoses may include psoriasis, nummular dermatitis, lichen simplex chronicus, dermatophytosis, erythema annulare centrifugum (EAC), cutaneous discoid lupus erythematosus (CDLE), pityriasis rosea, and pityriasis lichenoides chronica.

Psoriasiform CTB was first described in the early 1970s as psoriasiform lupus vulgaris, which is not uncommon (Figure 1).[20,21] Psoriasiform CTB has also been observed in TB verrucosa cutis.[22] An LS mimicking a psoriasiform plaque has been reported in a 16-year-old girl with systemic TB.[23]

Eczematous CTB, usually paucibacillary, can occasionally mimic lichen simplex chronicus with extensive and chronic pruritus.[24] In our experience, eczematous CTB is not uncommon in China (Figure 2).

Annular CTB may mimic tinea or EAC with centrifugal annular lesions (Figure 3).[25] Diagnosis of CTB should be considered in patients with long-standing tinea-like lesions without response to antifungal treatments. The association between systemic TB and EAC-like CTB has been reported.[26,27]

CDLE is the most important differential diagnosis of lupus vulgaris (Figure 4).[28,29] They both have prolonged courses and are most commonly seen on the face. In our own study,[12] lupus vulgaris and CDLE were more commonly seen in women. As treatment is totally different, histopathology is usually required for an early diagnosis, in particular in the endemic areas of TB infection.

Fig. 1 A 14-year-old girl with lupus vulgaris on the buttock for 3 years mimicking a psoriasis plaque.

Fig. 2 A 38-year-old man with eczematous lupus vulgaris on the sternum, present for more than 5 years.

Pityriasis-rosealike CTB has been reported as a tuberculid reaction in a 12-year-old boy after BCG vaccination.[30] CTB mimicking pityriasis rotunda has also been described as round, pigmented patches on the buttocks and back of a 63-year-old man.[31]

Macules and papules in CTB

Three variants of CTB typically manifest as maculopapules, namely, LS, PNT, and MTB.[14] LS and PNT are tuberculids, but MTB is the skin manifestation of severe active systemic TB. PNT is the most common of these three variants and is the fourth most common CTB in China.[12] LS and MTB are rare variants of CTB.

LS usually presents as asymptomatic, closely grouped, skin-colored to reddish-brown papules, often perifollicular, and are mainly found on the abdomen, chest, back, and proximal parts of the arms and legs (Figure 5).[32] LS mostly appears

Fig. 3 A 34-year-old woman with lupus vulgaris on the thigh for 1 year mimicking erythema anulare centrifugum.

Fig. 4 Lupus vulgaris mimicking chronic discoid lupus erythematosus on the face of an 11-year-old boy.

in children and adolescents with strong immune reactivity to *M tuberculosis*. This rare variety of tuberculid can mimic many other maculopapular dermatides, such as lichen nitidus, lichen planus, and lichen spinulosus.[33,34] With an annular distribution of papules, misdiagnosis as granuloma anulare is likely.[35,36] Generalized lesions, resembling granuloma anulare disseminatum after BCG vaccination, is known.[37]

The typical PNT consists of multiple symmetric papules, 1 to 5 mm in diameter, with an umbilicated, necrotic center. The lesions are usually found on the extensor sides of the extremities, sometimes on the trunk, buttocks, lower aspect of the abdomen, and even earlobes (Figure 6).[38] Skin disorders displaying excoriated or necrotic papules can be part of the

Fig. 5 A 12-year-old boy with lichen scrofulosorum on the anterior aspect of the forearm.

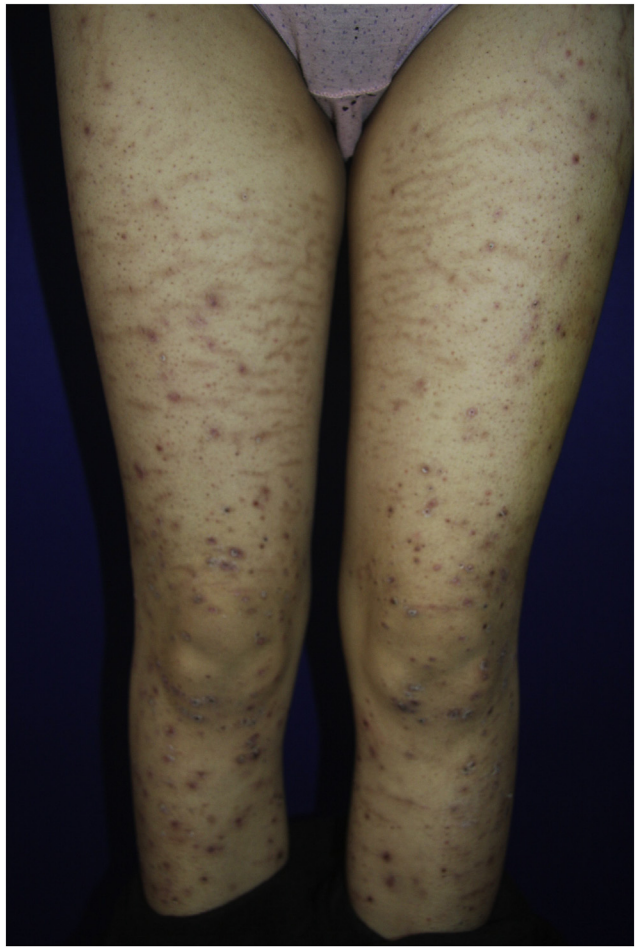

Fig. 6 An 18-year-old woman with papulonecrotic tuberculid mimicking pityriasis lichenoides chronica.

differential diagnoses of PNT in endemic areas, such as lymphomatoid papulosis, pityriasis lichenoides et varioliformis acuta (PLEVA), varicella, secondary syphilis, and prurigo. Rare presentations may even imitate molluscum contagiosum,[39] pityriasis lichenoides chronica,[40] or perforating granuloma annulare.[41]

MTB is caused by hematogenous dissemination of tubercle bacilli. It is more common in infants and immunocompromised patients with HIV infection, undergoing chemotherapy, or having immunosuppression after organ transplantation or under treatment of autoimmune diseases. Cutaneous involvement in MTB is rare, with nonspecific maculopapules, pustules, or erythematous subcutaneous nodules.[42] The skin lesions are commonly observed on the buttocks, thighs, genitalia, and extremities, particularly the extensor surfaces, but rarely on the face, which are generally asymmetric in distribution and several dozens in number.[43] Unusual cutaneous manifestations have been reported, such as whitlows of the thumb,[44] subcutaneous abscesses,[45] and infiltrated dermal subcutaneous and ulcerative lesions.[46]

A very rare form of disseminate lupus vulgaris presenting as granulomatous folliculitis should be differentiated from

Fig. 7 A 24-year-old man with erosions and abscesses of scrofuloderma tuberculosis.

papulopustular rosacea, perioral dermatitis, sarcoidosis, acneiform secondary syphilis, and various deep fungal infections.[47]

Ulcers and erosions in CTB

Scrofuloderma (Figure 7), EI, orificial TB, and occasionally PNT can present ulcerative erosive lesions.[5,6,8]

A nonhealing wound on the left foot over 3 years in a patient with diabetic mellitus was treated as diabetic foot ulcer without any improvement. The CTB infection was confirmed by bacterial culture and PCR examination, and the wound healed after antitubercular therapy.[48] The condition of a patient who presented with a leg ulcer, digital gangrene, and absent peripheral pulse was initially diagnosed as Takayasu arteritis, but later found as pulmonary TB, with multiple necrotic lung lesions, multiple caseating cervical lymph nodes, and a positive Mantoux test.[49]

We are aware of a patient with true CTB who presented with a chronic leg ulcer via primary inoculation without a history of pulmonary TB or a primary focus of infection being identified (Figure 8).

There is a patient who presented with recurrent oral and genital ulcers, plus erythema nodosum-like skin lesions on her legs, where clinical manifestations fulfilled the diagnostic criteria according to the International Study Group for Behçet's disease. Due to swollen left cervical and supraclavicular lymph nodes, a biopsy was taken from the left cervical lymph, and histopathologic examination showed tuberculous lymphadenitis. The lesions completely resolved after anti-TB therapy. The patient was eventually found to have a Behçet-like syndrome associated with TB infection.[50]

Genital CTB, especially TB of penis, including the glans, foreskin, and penile shaft, should be kept in mind, when considering the diagnosis of genital ulcers in TB-endemic areas.[51–54] Both true CTB and tuberculid have been reported. TB of the vagina and vulva has been described in sexually active and sexually inactive women, and usually presents as painful ulcers. Sometimes, it has been misdiagnosed as a sexually transmitted disease (Figure 9).[51,52]

TB orificialis, involving anal and perianal areas (Figure 10), is caused by autoinoculation of the bacillus into the anal or perianal skin and mucous membranes in patients with active gastrointestinal TB. Misdiagnosis as Crohn disease should be avoided.[55]

There are reports about axillary scrofuloderma and Pott disease with scrofuloderma and psoas abscess, being misdiagnosed and mistreated as hidradenitis suppurativa or acne inversa.[56,57] In reality, chronic hidradenitis suppurativa or acne inversa associated with TB is likely.[58]

Nodules and abscesses in CTB

Nodular lesions in CTB are represented by EI (Figure 11), which is classified as one subtype of tuberculids. Erythema nodosum (EN) can be associated with pulmonary TB but is not classified as CTB.[59] Lesions of EN are usually bilateral on the shins, knees, and ankles and subside within 1 month

Fig. 8 A 50-year-old man with primary inoculation tuberculosis, presenting as a chronic leg ulcer.

Fig. 9 An 18-year-old woman with painful vulvar ulcers found to have tuberculous chancre.

Fig. 10 A 42-year-old woman with orificial tuberculosis involving the anus.

with hyperpigmentation in the absence of ulcers. Lesions of EI occur more commonly on calves, usually ulcerate and resolve with atrophic scars, in a more protracted course. Atypical presentations of EI can mimic EN clinically, occasionally even in histopathology.[60] EI and EN show different responses to anti-TB therapy, in which EI can totally resolve and rarely recurs after a complete course of treatment.[61]

Some studies have suggested that TB may be an important cause of superficial thrombophlebitis or superficial venous thrombosis, with cordlike inflammatory thickening of superficial veins and nodular swellings on the legs.[62] It is considered as a rare variant of tuberculid, being named as nodular granulomatous phlebitis.[63]

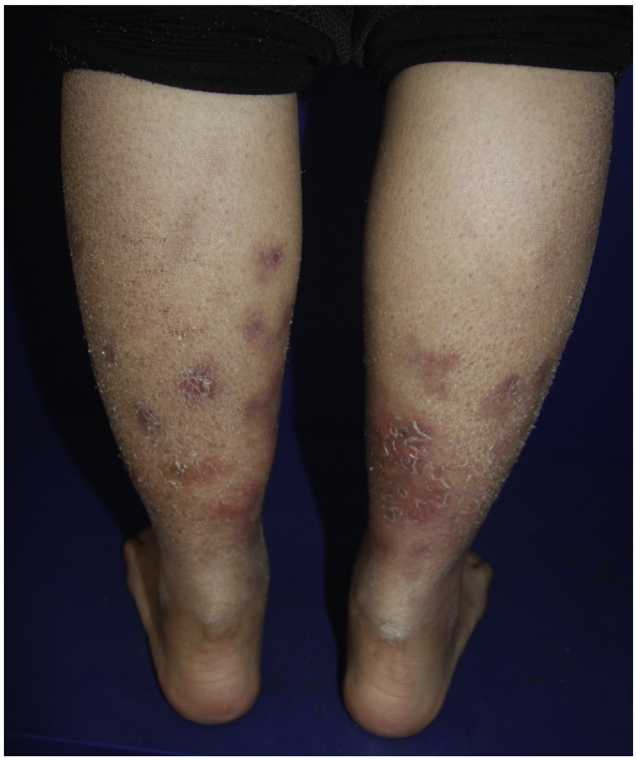

Fig. 11 A 35-year-old woman with erythema induratum of Bazin on both calves.

Tuberculous gumma, also known as metastatic tuberculous abscess, is characterized by nontender and fluctuant subcutaneous abscesses, appearing as single or multiple lesions on the trunk, extremities, or head.[64,65] Regional adenopathy is usually absent, which can be a differentiating sign for a staphylococcal abscess or other bacterial infections. Tuberculous gumma often originates from hematogenous spread in patients with immunosuppression or malnutrition. Acid-fast bacilli are typically detected in a tuberculous gumma, and the pathology demonstrates central ulceration with abundant caseous necrosis, surrounded by a rim of giant cells and macrophages. Differential diagnoses may include staphylococcal abscess, nocardiosis, atypical mycobacterial infections, chromomycosis, sporotrichosis or other deep fungal infections, leishmaniasis, leprosy, panniculitis, and syphilitic gumma.

Cellulitis-like CTB has been described in a patient with systemic lupus erythematosus being treated with oral cyclophosphamide and prednisolone,[66] and in a patient with acute military TB.[67]

Treatment and prognosis

The antituberculous drugs isoniazid, rifamycin, pyrazinamide, and ethambutol are the first-line treatment for all types of CTB,[68] following the standard regimen of WHO recommendation for the treatment of new cases of pulmonary TB.[5] For true CTB, the intensive phase begins and lasts for 2 months, followed by the maintenance phase for 4 months. Drug doses used in the intensive phase for adults and teenagers are rifampicin (450 mg/day for <50 kg body weight and 600 mg/day for >50 kg body weight), isoniazid (300 mg/day), pyrazinamide (1,500 mg/day), and ethambutol (750 mg/day for <50 kg body weight and 1,000 mg/day for >50 kg body weight), whereas doses for children are rifampicin (10-20 mg/kg per day), isoniazid (10-15 mg/kg per day), and pyrazinamide (30-40 mg/kg per day). Drug doses used in the maintenance phase are rifampicin (600 mg/day for adults and 10 mg/kg per day for children) and isoniazid (600 mg/day for adults and 10 mg/kg per day for children).[16] Additional surgical excision of refractory lesions and correction of deformities may be needed for recalcitrant cases.[69]

Treatment of tuberculids follows the same regimens recommended for true CTB. For EI, a longer period of treatment with isoniazid maintaining for up to 2 years is proposed.[8] Dapsone, potassium iodide, and doxycycline have been reported as adjuvants to treat the inflammation in EI, whereas corticosteroids or tuberculin protein in various dilutions for desensitization are occasionally used.[70]

True CTB and tuberculids can be cured following the standard anti-TB regimens. The recurrence of CTB after medical cure may be a concern, although available data are scarce. In

our 5-year followup of eight patients with EI, one of them (12.5%) showed recurrence after standard antituberculous therapy (unpublished data). More long-term studies are needed to understand the recurrence rates of various CTB under current treatment recommendations, especially in immunocompromised patients.

Conclusions

Among the classic great imitators, including syphilis and leprosy, CTB is becoming the foremost challenge to dermatologists in daily practice, not only due to the diversifying manifestations, multiple transmission pathways, perplexing pathogenesis, ambiguity in diagnosis, prolonged treatment course, and emergence of multiple drug resistance, but also due to the global migration and increasing use of immune targeting treatment in patients with malignancies and autoimmune or chronic inflammatory diseases. CTB should be considered in the differential diagnosis, and a systemic examination should be required of patients with unusual, atypical, therapeutically resistant, long-lasting skin disorders, especially in the TB-endemic areas.

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