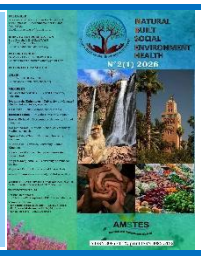
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Paucibacillary Cutaneous Tuberculosis Diagnosed Using GeneXpert MTB/RIF Ultra: A Case Report

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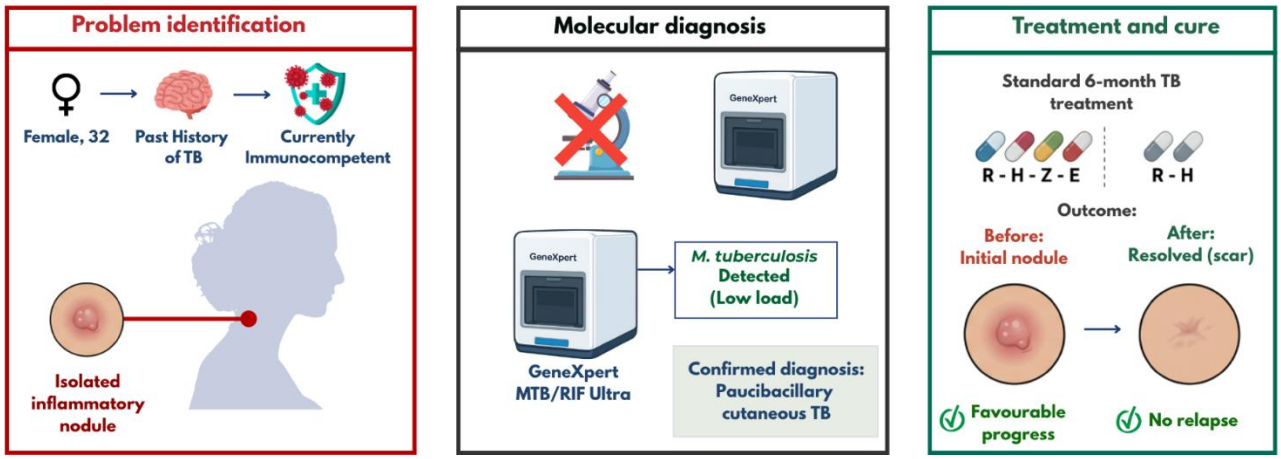
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Graphical abstract

Cutaneous tuberculosis is an uncommon paucibacillary form of extrapulmonary tuberculosis with diverse clinical presentations, complicating diagnosis. We report the case of a 32-year-old immunocompetent woman vaccinated with BCG and previously treated for neuromeningeal tuberculosis, who developed a progressive cervicothoracic skin lesion. Conventional microbiological tests were negative, whereas GeneXpert MTB/RIF Ultra on a skin biopsy detected *Mycobacterium tuberculosis* DNA without rifampicin resistance, confirming secondary scrofuloderma. Standard antituberculosis therapy led to a favourable outcome without recurrence.



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Abstract: Cutaneous tuberculosis is a rare form of extrapulmonary tuberculosis, often paucibacillary and with a polymorphic clinical presentation, making diagnosis difficult. We report the case of a 32-year-old immunocompetent woman, vaccinated with BCG, with a history of treated neuromeningeal tuberculosis, who developed a progressively worsening left cervicothoracic skin lesion. Clinical examination revealed an isolated inflammatory nodule without any alteration in her general condition. Conventional microbiological tests, including sputum smear microscopy and culture, were negative. But, molecular analysis by GeneXpert MTB/RIF Ultra on a skin biopsy detected *Mycobacterium tuberculosis DNA* at a low load, without rifampicin resistance, confirming paucibacillary cutaneous tuberculosis of the secondary scrofuloderma type. Standard antituberculosis treatment resulted in a favourable outcome without relapse. This case highlights the value of molecular techniques in the early diagnosis of atypical forms of extrapulmonary tuberculosis.

Keywords: GeneXpert MTB/RIF, Cutaneous Tuberculosis, Scrofuloderma.

Introduction

Tuberculosis (TB), a bacterial infection caused by the *Mycobacterium tuberculosis* (MTB) complex, remains a major public health problem worldwide, with a particularly high incidence in developing countries [1,2]. Pulmonary involvement is the most frequent form. Extrapulmonary manifestations, particularly those affecting the meninges, digestive system, osteoarticular system and urogenital system, present a diagnostic challenge because of their anatomo-clinical polymorphism [2,3].

Among these manifestations, cutaneous tuberculosis, most often caused by *Mycobacterium tuberculosis* but occasionally associated with *Mycobacterium bovis* [4], remains a rare entity, representing only 0.14% to 4.4% of tuberculosis cases. It can mimic numerous dermatological conditions, which may result in considerable diagnostic delay [3-5].

Despite the widespread introduction of BCG (Bacillus Calmette-Guérin) vaccination as a mandatory vaccine in the Moroccan National Immunization Programme (PNI), the protection conferred against extrapulmonary forms in adults remains variable and incomplete [6]. In addition, the occurrence of cutaneous forms in immunocompetent patients highlights the complexity of host–pathogen interactions [2,5].

The therapeutic protocol for cutaneous tuberculosis is based on prolonged antibiotic therapy combining several anti-tuberculosis drugs [1,2]. The diagnosis should therefore be considered in any chronic, destructive or verrucous skin lesion showing no signs of healing, even when conventional mycobacteriological tests remain negative [5,6].

We illustrate these particularities through the clinical observation of a young immunocompetent patient vaccinated with BCG, with a history of treated neuromeningeal tuberculosis, who subsequently developed paucibacillary scrofuloderma-type cutaneous tuberculosis. The diagnosis could only be confirmed through a molecular approach using the GeneXpert MTB/RIF Ultra test, despite negative bacilloscopy and culture results, reflecting the diagnostic difficulties and the importance of early management.

Clinical Observation

A 32-year-old woman originally from Morocco, vaccinated at birth with BCG as part of the National Immunization Programme, moved to Italy at the age of 21. At the age of 29, she was diagnosed and treated for neuromeningeal tuberculosis. She was initially hospitalized for

diffuse, intense and persistent headaches associated with a moderate but prolonged fever, followed by impaired consciousness progressing to coma.

The diagnosis was delayed because TB was not initially suspected in the non-endemic context of the patient's country of residence. Cerebral involvement was only confirmed after exploratory surgery, which identified an MTB infection. Following surgical intervention and the initiation of a well-managed antituberculosis treatment regimen, the patient's prognosis improved, leading to complete clinical recovery.

Twenty-two months later, the patient returned to the Kingdom of Morocco and subsequently developed a progressively worsening left cervicothoracic skin lesion. At the onset of the lesion, the patient, unaware of extrapulmonary forms of TB, began several forms of self-medication using ointments and creams obtained from a pharmacy without consultation or prescription. These included H1 antihistamines such as chlorphenoxamine, topical antibiotics such as fusidic acid and chlortetracycline, and topical antifungals such as clotrimazole and econazole. No therapeutic response or clinical improvement was observed, and the patient eventually consulted a dermatologist.



Figure 1. Clinical appearance of paucibacillary scrofuloderma-type cutaneous tuberculosis secondary to neuromeningeal tuberculosis in an immunocompetent patient vaccinated with BCG (2 weeks after onset).

Clinical examination showed that the patient was in excellent general condition, without fever or chills, but presented with a skin lesion in the form of an isolated nodule that had slowly developed over several weeks to more than one month. The lesion was located in the supraclavicular region opposite the left anterior cervicothoracic junction. It was a well-defined, rounded lesion measuring a few centimetres in diameter, slightly raised, with a smooth, shiny and reddish surface. The initial appearance suggested a benign inflammatory lesion resembling an insect bite, without crusting or scaling. The lesion was erythematous, reflecting a localized subcutaneous inflammatory process. It was slightly tender on palpation, without fistulization or discharge (Fig. 1).

In the context of a recent history of treated central nervous system tuberculosis (CNST), this clinical and anamnestic presentation, together with negative HIV serology, suggested secondary cutaneous TB despite the atypical clinical appearance of the lesion. The patient was referred for specialized bacteriological investigations. Initially, direct examination (bacilloscopy) and mycobacteriological cultures performed at a local medical biology laboratory on skin samples (swabs) were negative.

A mycobacteriological re-evaluation carried out at Ibn Sina Hospital, confirmed this paucibacillary profile, with absence of growth in culture on specific Löwenstein–Jensen medium and negative microscopy, showing no acid-fast bacilli (AFB) after modified Ziehl–Neelsen staining. These analyses were performed on a skin biopsy specimen of the lesion obtained by the treating dermatologist.

In contrast, molecular analysis using the GeneXpert MTB/RIF Ultra assay, performed on the same skin biopsy specimen, revealed within less than two hours the presence of *Mycobacterium tuberculosis* (MTB) DNA detected at a very low level, without detection of the *rpoB* gene associated with rifampicin resistance (RR), thus confirming the diagnosis of cutaneous tuberculosis. Additional investigations were recommended, but no other active systemic involvement was identified (Fig 2).



Figure 2. Molecular diagnosis performed on a skin biopsy specimen using the GeneXpert MTB/RIF Ultra assay, a real-time nested PCR technique enabling the detection of *Mycobacterium tuberculosis* and rifampicin resistance.

The diagnosis was scrofuloderma-type cutaneous TB. It was considered a secondary paucibacillary tuberculosis occurring in an immunocompetent individual vaccinated with BCG, in the context of post-treatment neuromeningeal tuberculosis. The patient was referred to a CDTMR (Centre for the Diagnosis and Treatment of Respiratory Diseases) in Casablanca to receive free treatment.

The therapeutic protocol consisted of standard four-drug antituberculosis therapy combining Rifampicin, Isoniazid, Pyrazinamide and Ethambutol for two months (2RHZE), followed by a four-month maintenance phase with Rifampicin and Isoniazid (4RH). The clinical course was favourable, with progressive regression of the skin lesion, although noticeable scarring remained (Fig. 3).

It should also be noted that the patient showed no recurrence of TB during follow-up, particularly beyond three months after completion of treatment. No cutaneous involvement or other extrapulmonary localization of tuberculosis was observed.

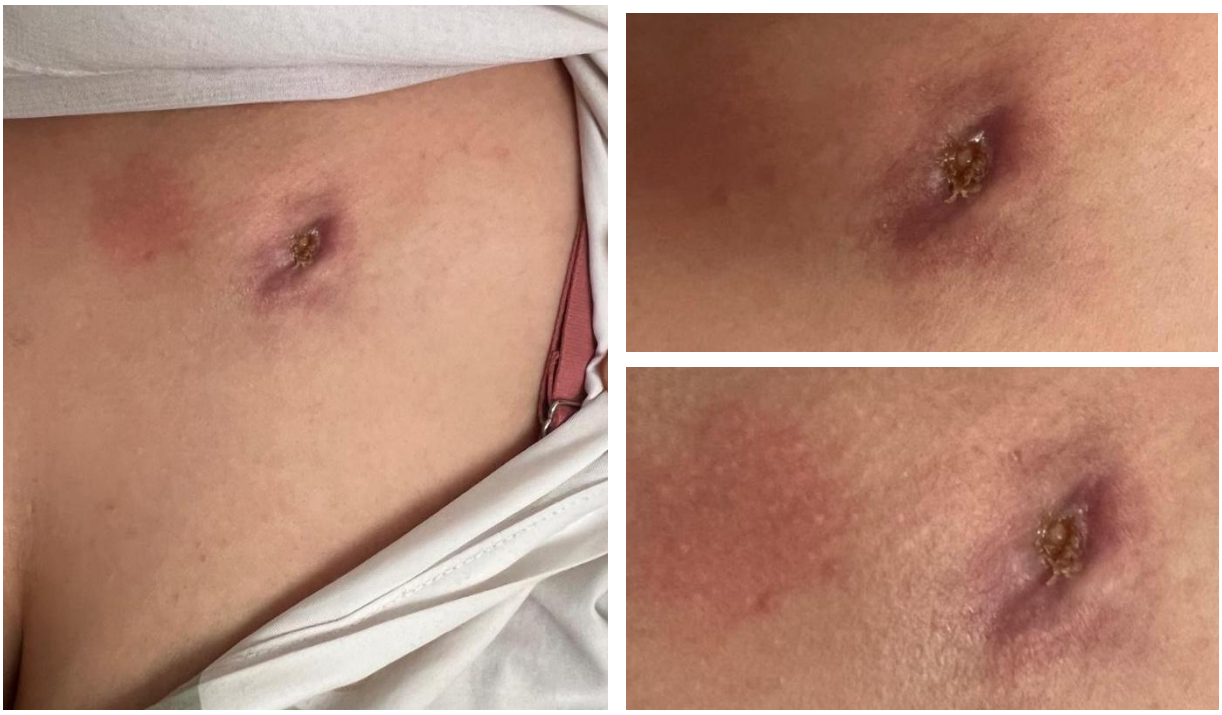


Figure 3. Clinical appearance of paucibacillary scrofuloderma-type cutaneous tuberculosis secondary to neuromeningeal tuberculosis in an immunocompetent patient vaccinated with BCG (after skin biopsy and at the initiation of antituberculosis quadritherapy).

Discussion

We report a clinical case of paucibacillary scrofuloderma-type cutaneous tuberculosis occurring in a young immunocompetent patient vaccinated with BCG at birth, in the particular context of a recent history of treated neuromeningeal tuberculosis, with diagnostic delay favoured by initial self-medication.

Cutaneous tuberculosis is a rare form of secondary tuberculosis, representing less than 1–1.5% of extrapulmonary TB cases [7]. Its polymorphic clinicopathological presentation depends on several factors, including the virulence of the *Mycobacterium tuberculosis* strain, the route of exogenous or endogenous contamination, and the immune status of the host [5,8].

Exogenous contamination mainly results from direct inoculation of *Mycobacterium tuberculosis* (Koch's bacillus) into the skin from the external environment, generally through a breach in the skin barrier such as a wound or trauma. This route of contamination occurs most often in non-immune individuals and may be responsible for clinical forms such as tuberculous chancre and verrucous tuberculosis, in which the skin lesion represents the primary site of infection [1,5,8,9].

In contrast, endogenous contamination results from dissemination of the tubercle bacillus from a pre-existing internal tuberculous focus, whether active or latent. This mycobacterial spread may occur through haematogenous, lymphatic or contiguous routes from a deep focus such as lymph node, osteoarticular or visceral tuberculosis, and is therefore responsible for secondary forms such as scrofuloderma or lupus vulgaris [5,8-10].

In our patient, the clinical history strongly suggests endogenous dissemination related to the previous neuromeningeal focus, raising the possibility of reactivation or secondary bacillary spread despite previously well-conducted treatment. This observation is consistent with data from the literature describing delayed secondary localizations after deep tuberculosis [5,8-10].

Scrofuloderma classically corresponds to the direct extension of an underlying tuberculous focus, which may be lymph node, osteoarticular or other deep involvement. Clinical forms without an identifiable active deep focus, as observed in our case, have nevertheless been described, suggesting either haematogenous dissemination or local reactivation of MTB. This hypothesis is reinforced by the 22-month disease-free interval following treatment for neuromeningeal tuberculosis [5,8-10] (Table 1).

Clinically, the location and misleading initial presentation of the skin lesion, mimicking a benign inflammatory condition, also contributed to delayed diagnosis. Early forms of scrofuloderma may present as non-specific nodules before progressing to the typical ulcerations and fistulas [5,8,9]. This variability in clinical presentation requires a high level of suspicion, particularly in patients with a history of tuberculosis. This explains the interest shown by the treating dermatologist, who suspected cutaneous tuberculosis and referred the patient for laboratory confirmation before initiation of the antituberculosis treatment protocol.

Table 1. Clinical forms of cutaneous tuberculosis [5]

Clinical form	Mode of acquisition	Main lesions
Lupus vulgaris	Primary or secondary infection by direct extension or lymphohaematogenous spread	Centrifugal extension, serpiginous plaques, purplish-yellow colouration, scaling, atrophy
Warty tuberculosis	Secondary infection by autoinoculation	Keratotic plaques with irregular borders and dystrophic scarring
Miliary tuberculosis	Primary or secondary infection by massive haematogenous dissemination	Diffuse papules followed by vesicles and crusts
Papulonecrotic tuberculid	Tuberculid occurring during the course of tuberculosis	Dermal papules followed by necrotic pustules and atrophic varioliform scars
Lichen scrofulosorum	Tuberculid associated with lymph node or bone tuberculosis	Lichenoid perifollicular papules and plaques, yellowish or reddish-brown
Tuberculous gumma	Secondary infection by haematogenous dissemination	Erythematous-violaceous subcutaneous nodules and ulcers
Bazin's erythema induratum	Tuberculid occurring during chronic tuberculosis	Bluish-red subcutaneous nodules, sometimes ulcerated, mainly on the lower limbs
Erythema nodosum	Tuberculid occurring during tuberculosis	Painful nodules
Tuberculous chancre	Primary infection by local inoculation	Painless ulceration
Scrofuloderma	Secondary infection by contiguous extension from a neighbouring focus	Erythematous-violaceous nodules, initially painless and mobile, later adherent with suppuration
Orificial tuberculosis	Secondary infection by autoinoculation from a primary focus	Nodules followed by painful ulcerations, abscesses and fistulae

In the Kingdom of Morocco, cutaneous tuberculosis remains relatively common, mainly represented by scrofuloderma and gumma tuberculosis, as demonstrated by hospital series from the university hospitals of Fez and Casablanca, with high proportions reaching 67% and 58% of cases, respectively [11]. This distribution contrasts with that reported in French and Chinese series, where tuberculids, particularly Bazin's erythema induratum, are more frequently observed [11]. These differences may be explained by epidemiological, immunological and environmental variations among the studied populations, as well as differences in bacillary load profiles and diagnostic contexts.

Although effective against severe forms of tuberculosis in children, particularly tuberculous meningitis, BCG vaccination does not prevent the occurrence of various extrapulmonary forms

in adults [7-9,12]. Several scientific studies have shown that BCG modulates clinical severity rather than the risk of infection, which could explain the paucibacillary nature and relatively localized course observed in our patient [7-9,12].

From a diagnostic standpoint, our clinical case illustrates the difficulty of mycobacteriological confirmation of paucibacillary cutaneous forms using conventional bacteriological diagnostic techniques. Negative smear microscopy and culture results on Löwenstein–Jensen medium are indeed frequent, with positivity rates below 30% in some series. According to the WHO, the sensitivity of culture on solid Löwenstein–Jensen medium is estimated at approximately 75–80% [5,8,13]. Similar findings have been reported in several recent studies, confirming that conventional microbiological diagnostic methods have limited yield in cutaneous tuberculosis, often making diagnosis difficult and delayed [8,14].

In this context, molecular techniques, particularly the GeneXpert MTB/RIF Ultra assay, play a crucial role. This real-time nested PCR enables rapid, sensitive and specific detection of mycobacterial DNA, even in samples with low bacterial load, with markedly higher sensitivity than microscopy and culture, considered the gold standard [1,13]. A meta-analysis by Denkinger *et al.* [15], comparing the technical performance of GeneXpert MTB/RIF with culture as the gold standard, confirmed its value in extrapulmonary forms, including cutaneous tuberculosis, with a significant improvement in diagnostic yield [15]. This comparative study reported a specificity of 98.7%, a sensitivity of 83.1%, and a high positive predictive value of 98% [15]. The presence of polymerase inhibitors in tested specimens, including biological fluids and biopsies, may explain the reduction in sensitivity observed in some situations [16]. In our case, the GeneXpert MTB/RIF assay nevertheless confirmed the diagnosis despite negative conventional tests and a low mycobacterial load in the specimen, findings that do not exclude the diagnosis of cutaneous tuberculosis.

Another important aspect of this clinical observation is the diagnostic delay associated with self-medication without medical consultation. Our patient applied several topical treatments, including antibiotics, antifungals and H1 antihistamines, without medical advice or a precise diagnosis. This contributed to masking the clinical signs and delaying specialized management. This phenomenon is widely described in developing countries, where unrestricted access to medications promotes diagnostic delay and encourages self-medication, with initial reliance on symptomatic treatments without medical consultation or prescription [17].

One study showed that the time to diagnosis (TD-TEP) for extrapulmonary tuberculosis is largely patient-related, with a mean of 31.23 ± 43.55 days and a median of 17.5 days. Several factors influence this delay, including advanced age, male sex, rural residence and lack of awareness of the disease [18].

Another Moroccan multicentre study conducted in 2021 revealed that the distribution of patients with cutaneous tuberculosis, according to the interval between lesion onset and the first medical consultation, was characterized by a mean delay of approximately 11.5 months, reflecting a particularly marked diagnostic delay. This prolonged delay is explained by several factors, including the initial neglect of lesions by patients because of their often painless and non-specific nature, together with limited awareness of this condition among primary care physicians. In addition, the waiting time for access to specialist consultation, which may range from 2 to 8 months depending on healthcare facilities and regions, further contributes to prolonged diagnostic delay [19].

From a therapeutic standpoint, management is based on the standard regimen recommended by the World Health Organization (2RHZE/4RH) and detailed in the National Tuberculosis Control Programme of the Moroccan Ministry of Health and Social Protection. This regimen remains effective in the majority of cases. The favourable outcome observed in our patient, with disappearance of the lesion during treatment, confirms the high sensitivity of the bacillus and the efficacy of the therapeutic protocol. Persistence of scarring nevertheless remains common, particularly in advanced cutaneous forms [20,21]. Finally, the absence of recurrence three months after completion of treatment, together with the negative staging investigations, confirms the isolated nature of this secondary cutaneous lesion. This observation underlines the importance of early diagnosis and appropriate therapeutic management in order to avoid complications, sequelae and recurrences.

On the occasion of World Tuberculosis Day on 24 March 2026, the Moroccan Ministry of Health and Social Protection issued a ministerial statement reminding the public that nearly 53% of tuberculosis cases in Morocco are extrapulmonary, emphasizing the importance of increased vigilance regarding these often misleading and atypical presentations [22]. In this context, it is essential that clinicians from all specialties systematically include extrapulmonary tuberculosis in the differential diagnosis of any chronic, atypical or unusual lesion, particularly as tuberculosis is endemic in the Kingdom of Morocco. Several studies have also shown that the concomitant association of pulmonary tuberculosis with a typical clinical presentation and an extrapulmonary form remains relatively uncommon [22].

Finally, this case highlights the major and indispensable contribution of molecular diagnostic tools, particularly the GeneXpert MTB/RIF assay recommended by the World Health Organization, which enables rapid, sensitive and reliable detection of *Mycobacterium tuberculosis*, including in paucibacillary forms, thus contributing to early and appropriate management [1].

Conclusion

This clinical case illustrates the diagnostic challenges associated with cutaneous forms of tuberculosis, particularly in their biologically paucibacillary and clinically atypical presentation in an immunocompetent individual vaccinated against tuberculosis. Our work highlights the point that negative results obtained with conventional bacteriological methods do not exclude the diagnosis and underlines the crucial contribution of molecular techniques, notably the GeneXpert MTB/RIF Ultra assay, for rapid and reliable confirmation.

The occurrence of secondary scrofuloderma after treated neuromeningeal tuberculosis also reminds clinicians of the possibility of late dissemination or reactivation, thus justifying prolonged clinical vigilance. In addition, the initial self-medication contributed to diagnostic delay, emphasizing the importance of greater patient awareness. Finally, this case underlines the need always to include extrapulmonary tuberculosis in the differential diagnosis of chronic skin lesions in endemic areas such as the Kingdom of Morocco.

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