

CHRONIC GENITAL ULCER IN A TEENAGER REVEALS ORIFICIAL CUTANEOUS TUBERCULOSIS

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Abbreviations **TB** = tuberculosis; **TCO** = tuberculosis cutis orificialis.

Case report. A 15-year-old boy was admitted to the pediatric ward at Dr. Soetomo General Hospital with a genital wound. He was referred to the dermatology and venereology department for redness, purulent lesions, and a genital ulcer that had persisted for one month. Initially, small red pustules had appeared in the genital area; these progressively worsened, spread, enlarged, and evolved into ulceration. The patient complained of pain and easy bleeding. His mother also reported white patches in the mouth resembling oral ulcers for the past two weeks, associated with painful chewing and progressive thickening and spread. Additionally, the patient had intermittent fever for one month, a dry cough for two days without dyspnea, and significant weight loss. He denied abdominal pain, diarrhea, dysuria, hematuria, or penile or scrotal swelling. There was no history of chronic illness, allergies, high-risk behaviors, TB exposure, or similar family disease. Growth and development were normal.

Three weeks before admission, the patient had undergone penile necrotomy at another facility, but the lesions continued to worsen. Previous treatments included IV fluids, ranitidine, metamazole, metronidazole, nystatin, turmeric tablets, and sucralfate.

When attending the hospital visit he appeared weak but alert; on physical examination, weight was 45 kg, height 150 cm, BMI 20 kg/m². Tachycardia was noted (115 bpm), with normal respiratory rate and temperature. No anemia, jaundice, lymphadenopathy, or organ abnormalities were present. Dermatologic examination revealed white plaques on the tongue without erosion; the genital area showed multiple ulcers of varying sizes with pus and slough, without induration or necrosis. The pubic region had papules, pustules, and a 2 × 0.5 × 0.5 cm ulcer with minimal pus (Fig. 1).

Laboratory findings: Hb 12.1 g/dL (ref. 13.3-16.6), WBC 10.56 × 10³/μL (3.37-10.0), platelets 491,000/μL (150-450), BUN 6.4 mg/dL (6-20), albumin 3.53 g/dL (3.5-5.0). Gram stain from the genital ulcer revealed Gram-positive rods; the tongue sample showed no fungi or hyphae. Tzanck/Giemsa showed no Donovan bodies; HIV, VDRL, TPHA, and HSV 1/2 IgG/IgM were all negative.

Histopathology showed partially ulcerated acanthosis with dense lymphocytic and neutrophilic dermal infiltrate, with no epithelioid cells, granulomas, or malignancy. Tissue culture was negative for *M. tuberculosis*, GeneXpert on sputum was negative, while GeneXpert on tissue was positive for *M. tuberculosis* (rifampicin sensitivity indeterminate); Mantoux was positive (20 mm, turbid).

The combination of histopathology and molecular diagnostics allowed a definitive diagnosis of tuberculosis cutis orificialis (TCO) despite atypical clinical presentation and negative microscopy and culture.

After diagnosing genital ulcer due to orificial cutaneous tuberculosis and oral candidiasis, the patient received intensive antituberculosis therapy for 2 months (isoniazid 300 mg/day, rifampicin 450 mg/day, ethambutol 750 mg/day), followed by 4 months of isoniazid + rifampicin (450 mg/day), along with nystatin for the oral lesions.

On day 8, sudden ulcer bleeding occurred and was controlled with epinephrine-soaked gauze; Hb subsequently fell to 7.2 g/dL, requiring transfusion.

By day 12, the lesions had improved significantly. After 20 days, the ulcers were dry, reduced in size, and no new lesions were present; oral plaques had resolved completely. The patient was discharged on day 20, and at follow-up the ulcers had fully healed, leaving only hypopigmented macules on the genitalia and pubic area (Fig. 2).



Fig. 1



Fig. 2

Fig. 1, 2: Ulcerated orificial cutaneous tuberculosis in a 15-year-old boy (Fig. 1) and after specific therapy (Fig. 2).

Discussion. Tuberculosis (TB) is a chronic infection primarily caused by *Mycobacterium tuberculosis* and can affect nearly any organ, although pulmonary involvement is the most common. Extrapulmonary TB accounts for 8-24% of cases, while cutaneous TB remains rare, representing only 1.5-3% of such manifestations (3). Despite the global TB burden of 9.9 million new cases reported in 2020 – concentrated in India, China, Indonesia, the Philippines, and Pakistan – cutaneous TB constitutes only a small fraction, with prevalence ranging from 0.03–0.06% in low-burden regions to 0.1-2% in high-burden areas (1, 4). A slight male predominance has been reported, and in several Asian and African countries, nearly half of cases occur in children and young adults. The rising incidence of cutaneous TB parallels increases in HIV infection and multidrug-resistant TB (1).

Cutaneous TB encompasses several clinical forms influenced by host immunity, bacterial load, and route of infection, including tuberculosis verrucosa cutis, inoculation TB, scrofuloderma, lupus vulgaris, miliary TB, metastatic tuberculous abscesses, tuberculids, and the exceptionally rare tuberculosis cutis orificialis (TCO). TCO presents as chronic, punched-out mucocutaneous ulcers involving the oral, nasal, or anogenital regions and typically results from autoinoculation of bacilli from an internal tuberculous focus. Although more common in immunosuppressed individuals, primary TCO without identifiable internal disease has been documented (1, 3).

In the present case, a boy presented with a painful genital ulcer persisting for one month, without any history of sexual contact, TB exposure, or immunosuppression. Because genital ulcers in adolescents are more commonly attributed to sexually transmitted infections, the initial diagnostic work-up targeted these etiologies. Direct smears for acid-fast bacilli, Donovan bodies, and Tzanck cells were negative; serologic testing for syphilis and HSV was also nonreactive. Unexpectedly, GeneXpert performed on ulcer tissue detected *M. tuberculosis* DNA, establishing the diagnosis of TCO. Clinically, TCO is characterized by irregular, punched-out ulcers with yellowish granules, seropurulent discharge, and easy bleeding, which may be solitary or multiple. No internal TB focus was identified

in this patient. This finding is consistent with previous reports indicating that internal sources may be clinically silent or undetectable due to the paucibacillary nature of the disease. Although rare, primary TCO without an identifiable autoinoculation source has been described (6-8).

Histopathologic examination revealed ulcerated epidermis with dense lymphocytic and neutrophilic infiltrates but no granulomas, findings compatible with early or atypical cutaneous TB. Histology in cutaneous TB is notoriously variable, ranging from neutrophilic inflammation to classic caseating granulomas. Granuloma formation depends on disease stage, host immunity, and bacterial load; exogenous forms may begin as neutrophil-rich lesions, whereas endogenous forms often exhibit nonspecific patterns (5, 9-11). This variability underscores the need to integrate clinical, microbiologic, and molecular data to achieve diagnostic certainty (9, 12).

Diagnostic challenges in cutaneous TB are largely attributable to the low sensitivity of conventional tests. Tuberculin skin testing has limited reliability, and both direct microscopy and mycobacterial culture often yield negative results due to the paucibacillary nature of lesions, inadequate sampling, slow organism growth, or technical limitations (9, 13, 14). In contrast, molecular diagnostics – particularly the GeneXpert MTB/RIF assay – have significantly improved detection of cutaneous TB by providing rapid, highly specific identification of *M. tuberculosis* and rifampicin resistance. Sensitivity in extrapulmonary specimens ranges from 53-95%, and many reported cases demonstrate GeneXpert positivity despite negative cultures. In our patient, tissue GeneXpert positivity with concurrent negative sputum results reflected localized disease, a pattern typical of cutaneous forms (15, 16).

Management follows standard antituberculosis therapy guidelines: a 2-month intensive phase with rifampicin, isoniazid, pyrazinamide, and ethambutol, followed by a 4-month continuation phase with rifampicin and isoniazid. Pediatric dosing is weight-based, and ethambutol may be omitted unless drug resistance is suspected. Most cutaneous TB lesions show clinical improvement within 4-6 weeks, with complete healing occurring over 1-5 months. Treatment failure warrants reevaluation for alternative diagnoses or drug-resistant TB (2, 17).

Conclusion. Cutaneous tuberculosis remains a rare but clinically significant form of extrapulmonary TB, often presenting with nonspecific features that may obscure timely diagnosis. The identification of *Mycobacterium tuberculosis* through tissue-based molecular testing highlights the crucial role of nucleic acid amplification techniques in detecting paucibacillary disease, particularly when conventional microbiologic and histopathologic methods are negative.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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