Section: Dermatology

Case Report

CASE OF SCROFULODERMA

Surendra Singh Bhati[1], Ujjwal Jharia[2]

[1]Professor, Department of Dermatology, Venereology and Leprosy at Index Medical College Hospital & Research Centre, Indore, India.
[2]Junior Resident, Department of Dermatology, Venereology and Leprosy at Index Medical College Hospital & Research Centre, Indore, India.

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Corresponding Author:
Dr. Ujjwal Jharia,
Junior Resident, Department of Dermatology, Venereology and Leprosy at Index Medical College Hospital & Research Centre, Indore, India.
Email: doctorujjwaljharia@gmail.com

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ABSTRACT

Scrofuloderma, a cutaneous manifestation of tuberculosis, is an uncommon but clinically significant presentation of mycobacterial disease. It usually results from the direct extension of Mycobacterium tuberculosis from an underlying infected lymph node or osseous structure to the overlying skin. The condition is characterized by chronic granulomatous inflammation, leading to skin abscesses, ulcers, and fistulae. Due to its nonspecific clinical features, scrofuloderma often poses significant diagnostic challenges, as it can mimic other dermatological or infectious conditions. This case report outlines the clinical progression of a patient diagnosed with scrofuloderma in a tertiary care hospital. It highlights the diagnostic challenges faced during evaluation and describes the comprehensive use of investigative tools to confirm the diagnosis. Furthermore, this report discusses the management plan, focusing on the efficacy of a tailored antitubercular treatment regimen.

Keywords: Chronic granulomatous inflammation, scrofuloderma, cutaneous manifestation, tuberculosis.

INTRODUCTION

Tuberculosis (TB), caused by Mycobacterium tuberculosis, continues to be a significant infectious disease globally, presenting a major public health challenge. While it is primarily recognized for its pulmonary involvement, TB can affect nearly any organ system, resulting in a diverse range of clinical manifestations.[1,2] Among these, cutaneous TB is an uncommon yet clinically significant form, encompassing a spectrum of dermatological presentations due to mycobacterial infection of the skin. Scrofuloderma, also referred to as tuberculosis cutis colliquativa, represents a distinct form of cutaneous TB, typically arising from the direct extension of an underlying TB infection from lymph nodes, bones, or other organs to the skin.[3-7]

Scrofuloderma, the focus of this discussion, is a rare but clinically relevant manifestation of cutaneous TB. While TB remains a pressing global health issue, with approximately 10.6 million new cases and 1.6 million deaths reported worldwide in 2021, scrofuloderma constitutes a small fraction of these cases, underscoring its rarity.[8] Despite significant progress in TB diagnostics and treatment, particularly in resource-limited regions with limited healthcare access, diagnosing scrofuloderma can be challenging due to its infrequency and nonspecific clinical features.[9] Furthermore, the emergence of multidrug-resistant Mycobacterium tuberculosis strains adds to the complexities of treatment, emphasizing the need to identify and manage all TB forms, including its cutaneous variants.[10] Although specific prevalence data on scrofuloderma may be limited, case reports and literature highlight sporadic instances, underscoring the importance of maintaining a high level of clinical suspicion and awareness of this condition.

Scrofuloderma is predominantly observed in children and young adults, as these age groups are more susceptible due to immunological immaturity. It often serves as an indicator of an underlying tuberculosis (TB) infection that may have gone undiagnosed. The development of scrofuloderma arises from the immune response to Mycobacterium tuberculosis, leading to chronic granulomatous lesions that progress to ulceration and discharge through the skin. This progression causes considerable morbidity in affected individuals and represents a public health concern due to its potential for transmission, emphasizing the critical need for early detection and intervention.[3]

The pathogenesis of scrofuloderma involves the invasion of the skin by TB infection from an

adjacent focus, such as an infected lymph node or bone. This extension is driven by the immune system's attempt to contain the infection, which paradoxically results in tissue inflammation and necrosis.[11] Clinically, scrofuloderma is characterized by nodules that gradually enlarge, become fluctuant, and eventually rupture, producing ulcers with characteristic seropurulent discharge. These lesions are frequently accompanied by systemic TB symptoms, such as fever, unexplained weight loss, and general malaise. In certain instances, the cutaneous symptoms may precede the recognition of systemic TB, making it imperative to maintain clinical vigilance.[12,13]

The diagnosis of scrofuloderma relies on integrating clinical evaluations, histopathological analyses, microbiological tests, and imaging studies to determine the extent of systemic involvement. Histopathologically, scrofuloderma is distinguished by granulomatous inflammation, typically with caseation necrosis, a hallmark of tuberculosis (TB) infection.[12] However, due to overlapping clinical and histological features, diagnosing scrofuloderma can be challenging, necessitating differentiation from other granulomatous conditions.

Granulomatous diseases, such as sarcoidosis, leishmaniasis, fungal infections (e.g., histoplasmosis, sporotrichosis), and other mycobacterial infections (e.g., atypical mycobacteria), can mimic scrofuloderma, further complicating diagnosis.

Sarcoidosis, identified by non-caseating granulomas, may resemble cutaneous tuberculosis (TB), with systemic manifestations assisting in distinguishing the two. Leishmaniasis, a disease caused by protozoan parasites, can mimic scrofuloderma; a thorough travel history and appropriate serological testing are valuable for diagnosis. Fungal infections, such as histoplasmosis, sporotrichosis, and infections caused by atypical mycobacteria, present similar diagnostic challenges; differentiation often relies on histopathological evaluation, microbial cultures, and molecular diagnostic techniques.

In endemic regions, a high level of clinical suspicion, supported by multidisciplinary collaboration between dermatologists, infectious disease specialists, and radiologists, is vital for accurate diagnosis and effective treatment.[14]

The management of scrofuloderma necessitates a multidisciplinary approach, often involving dermatologists, infectious disease experts, and occasionally surgeons when advanced disease requires intervention. The cornerstone of treatment is antituberculosis therapy (ATT), adhering to standard TB treatment protocols. Treatment duration and drug combinations depend on factors such as the strain's drug susceptibility, the patient's clinical response, and any indication of multidrug-resistant TB.[12] The primary aim of therapy is to eliminate the infection, prevent disease transmission, and minimize complications such as scarring or disfigurement. Early intervention is crucial for reducing morbidity and preventing long-term sequelae associated with cutaneous TB manifestations.

CASE REPORT

A 24-year-old male patient visited the outpatient Department of Dermatology, Venereology, and Leprosy at Index Medical College Hospital & Research Centre, Indore, presenting with a gradually increasing, painless swelling of his neck and hand that had been progressing over six months. He reported a history of multiple hospital visits for fever, chest discomfort with sweating over the past six months, along with significant weight loss. Initially appearing as a small, firm nodule, the lesion progressively enlarged, ulcerated, and started discharging a serous fluid. Upon examination, there were multiple well-defined crusted plaques with ulcerations and discharge on an erythematous base over the left side of the neck and the right dorsal aspect of the hand. The patient also described systemic symptoms, including fever, night sweats, weight loss, and a persistent cough.

Figure 1 and Figure 2 illustrates prominent, confluent nodule on the neck and dorsal surface of the hand, respectively; with the overlying skin exhibiting violaceous discoloration in some areas and ulceration in others. Certain parts of the lesion had broken down, forming sinuses that discharged seropurulent material. No additional similar lesions were detected elsewhere on the body. The patient reported no known contact with individuals diagnosed with active tuberculosis.

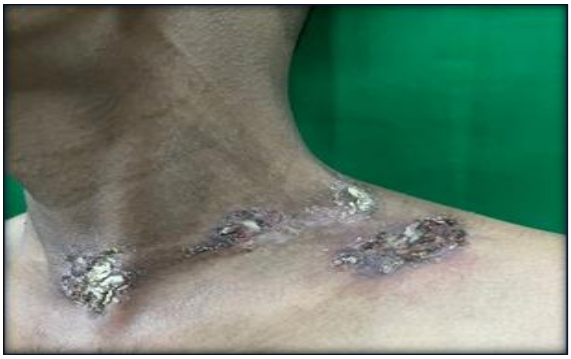

Figure 1: Lesions with pus-discharging sinuses on the neck

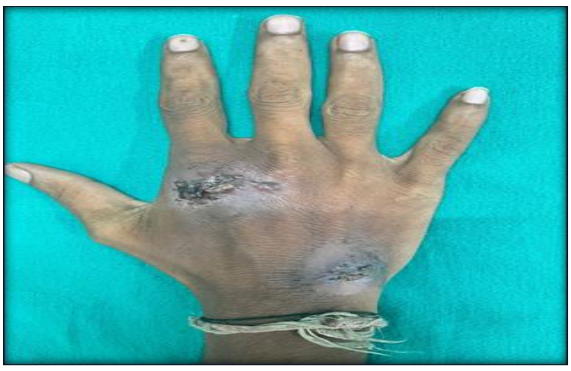

Figure 2: Lesions with pus-discharging sinuses on the dorsal surface of the hand

Investigations

During the investigation phase, the patient underwent an extensive series of tests to confirm the diagnosis of scrofuloderma. An incisional skin biopsy revealed granulomatous inflammation with caseating necrosis, which is characteristic of tuberculosis (TB). Despite the absence of pulmonary involvement, as evidenced by a normal chest X-ray and computed tomography (CT) scan, the Mantoux test was positive (>10 mm induration at 72 hours), indicating either latent or active mycobacterial infection. However, the QuantiFERON-TB Gold (IGRA) test was negative. Ultrasound (USG) of the neck revealed multiple lymph nodes measuring 0.5 × 1.0 cm. On clinical examination, multiple well-defined crusted plaques with ulcerations and discharge on an erythematous base were observed over the left side of the neck and the dorsal aspect of the right hand.

Table 1 summarizes the patient's investigation results and key findings across various parameters. The complete blood count (CBC) revealed a hemoglobin level (Hb%) of 10.8 g/dL, indicating mild anemia. The mean corpuscular hemoglobin concentration (MCHC) was 32.2 g/dL, while the mean corpuscular volume (MCV) was reduced at 71.3 fL, suggesting microcytosis. The red blood cell (RBC) count was 4.72 × 10^{12}/L, and the white blood cell (WBC) count was elevated at 10,100 cells/µL. The platelet count was slightly low at 4.32 × 10^{9}/L, below the normal range. In the coagulation profile, the activated partial thromboplastin time (APTT) was prolonged to 52.3 seconds, while both the prothrombin time (PT) and international normalized ratio (INR) were within normal limits. A peripheral smear showed a normocytic normochromic blood picture. Thyroid function tests were normal, with free triiodothyronine (FT3), free thyroxine (FT4), and thyroid-stimulating hormone (TSH) all within the reference range. Kidney function tests indicated normal urea and creatinine levels, while liver function tests demonstrated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) levels within the normal range, alongside normal total bilirubin levels. Random blood sugar was slightly elevated at 123 mg/dL. Virology testing for hepatitis B surface antigen (HBsAg), hepatitis C virus (HCV), and human immunodeficiency virus (HIV) returned negative results. These comprehensive investigations, in conjunction with clinical findings, confirmed the diagnosis of scrofuloderma, enabling the initiation of targeted antituberculosis therapy.

Table 1: Clinical findings of various parameters of the patient

Test	Patient's Value	Reference Value
Hb	10.5 g/dL	12-15 g/dL
MCHC	32.1 g/dL	32-36 g/dL
MCV	71.2 fL	80-100 fL
RBC count	4.70	4.5-5.5x10^{12}/L
WBC count	10110 cells/µL	4000-11000 cells/µL
Platelet count	4.30 x 10^{9}/L	150-450x10^{9}/L
APTT	52.1 seconds	30-40 seconds
PT	13.1 seconds	11-14 seconds
INR	1.15	0.8-1.2
Peripheral smear	Normocytic normochromic	N/A
FT3	3.92 pg/mL	2.0-4.4 pg/mL
FT4	1.58 ng/dL	0.93-1.7 ng/dL
TSH	2.32 µIU/mL	0.27-4.2 µIU/mL
Urea	21 mg/dL	10-50 mg/dL
Creatinine	0.8 mg/dL	0.6-1.2 mg/dL
ALT (SGPT)	18 U/L	< 35 U/L
AST (SGOT)	21 U/L	< 35 U/L
Total bilirubin	0.5 mg/dL	0.1-1.2 mg/dL
Random blood sugar	124 mg/dL	< 140 mg/dL
HBsAg	Negative	N/A
HCV	Negative	N/A
HIV	Negative	N/A

Figure 3 displays a histopathological section of scrofuloderma stained with routine hematoxylin and eosin. The image reveals a reduced thickness of the epidermis and significant alterations in the underlying tissue. Notable findings include areas of caseating necrosis, the presence of Langhans-type giant cells with a characteristic "horseshoe" arrangement of nuclei, and clusters of epithelioid histiocytes encircled by mononuclear inflammatory cells. These features are consistent with granulomatous inflammation typically observed in tuberculosis.

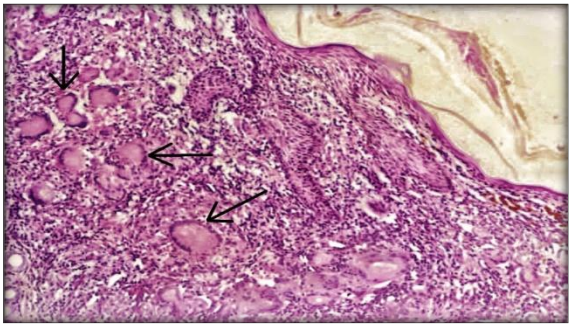

Figure 3: Histopathological image of the scrofuloderma.

Treatment

Antitubercular treatment was initiated promptly, consisting of a fixed-dose combination of standard first-line antitubercular drugs (HRZE): isoniazid 75 mg, rifampicin 150 mg, pyrazinamide 400 mg, and ethambutol 275 mg. Based on the patient's weight band, four tablets were administered once daily during the first eight weeks (intensive phase). This was followed by a continuation phase lasting 16 weeks, comprising isoniazid 75 mg, rifampicin 150 mg, and ethambutol 275 mg, aimed at eradicating the infection and preventing multidrug-resistant (MDR) TB development. Comprehensive wound care was also provided for the ulcerated skin lesions, focusing on enhancing healing and preventing secondary infections. This treatment plan highlights the significance of a multidisciplinary approach, combining effective pharmacological therapy with supportive care, to optimize outcomes in managing scrofuloderma.

Outcome and follow-up

The patient showed remarkable clinical improvement within the first 7–8 days of antituberculosis therapy, with a noticeable reduction in lesion discharge and early signs of healing in the ulcerated areas.

DISCUSSIONS

Scrofuloderma arises due to skin infection adjacent to a tuberculous focus, which may involve peripheral ganglion TB or TB of the bone, joint, or testicles. Clinically, it is characterized by subcutaneous, painless, and slowly enlarging nodules that evolve into ulcers and fistulous tracts with serous, purulent, or caseous discharge.[12] This case underscores the importance of considering TB in the differential diagnosis for persistent, chronic skin lesions, particularly in regions where TB remains endemic.[15]

Other distinct forms of cutaneous TB caused by direct infection with Mycobacterium tuberculosis include lupus vulgaris, tuberculosis verrucosa cutis, tuberculous chancre, and tubercular gumma. These differ from tuberculides, which are hypersensitivity reactions to TB elsewhere in the body, such as erythema induratum (nodular vasculitis), lichen scrofulosorum, and papulonecrotic tuberculid. Scrofuloderma, however, is unique in that it presents as asymptomatic swellings that persist for months before softening and ulcerating, as seen in our patient. This manifestation results from infection and degeneration of skin overlying a superficial tuberculous focus, typically affecting cervical lymph nodes.[16]

Although systemic symptoms such as fever, weight loss, or chronic cough—commonly associated with active TB—were absent in this case, the patient exhibited a positive Mantoux test and confirmed the presence of mycobacteria in the lesion discharge, supporting a diagnosis of cutaneous TB.

In a study by Umapathy et al,[17] it was noted that the drug resistance patterns of isolated tubercle bacilli, including initial multidrug resistance (to isoniazid and rifampicin), mirrored resistance patterns observed in pulmonary TB cases in controlled clinical trials. In our case, while multidrug-resistant (MDR) TB was absent, a four-drug antitubercular regimen proved to be an effective treatment strategy. This combination therapy successfully eradicated the mycobacterial infection, reducing the risk of drug resistance and recurrence of scrofuloderma.

CONCLUSION

In conclusion, this case underscores the imperative to consider TB in the spectrum of granulomatous skin diseases, advocating for prompt diagnosis, efficacious treatment strategies, and the mitigation of potential complications. Moreover, it highlights the pivotal role of early detection, precise diagnosis, and meticulous management in ameliorating patient outcomes and mitigating the burden of TB-related skin manifestations. Notably, the uniqueness of this case lies in its contribution to the existing body of knowledge by elucidating the diagnostic and therapeutic challenges encountered in managing scrofuloderma. By delineating the clinical course and outcomes, this report offers novel insights into the complexities of TB-associated cutaneous manifestations. Thus, it accentuates the significance of continued vigilance, interdisciplinary collaboration, and innovative approaches in confronting TB-related dermatological conditions.

Declarations

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