Lymphocutaneous Sporotrichosis Treated with Potassium Iodide with Development of Subclinical Hypothyroidism: Wolff-Chaikoff Effect?

Abstract

Sporotrichosis is a subcutaneous mycotic infection caused by *Sporothrix schenckii* that is acquired by traumatic implantation. The diagnosis is established by demonstration of fungal elements on histopathology and culture. Potassium iodide, azole antifungals, and terbinafine are the treatment options available. In this article, we report a 60-year-old female with lymphocutaneous sporotrichosis that responded well to potassium iodide. However, subclinical hypothyroidism (Wolff–Chaikoff effect) was encountered as a side effect of therapy which was managed with thyroxine replacement. Knowledge about the Wolff-Chaikoff effect (WCE) is important for the dermatologist and reinforces the need for screening and monitoring of thyroid stimulating hormone (TSH) in patients where long duration therapy is being planned.

Keywords: *Itraconazole, potassium iodide, sporotrichosis, Wolff-Chaikoff effect*

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Introduction

Sporotrichosis is a subcutaneous fungal infection that runs a chronic course. Potassium iodide is an established treatment modality for this condition that is both effective and cheap. We encountered a patient with lymphocutaneous sporotrichosis whom we treated with saturated solution of potassium iodide (SSKI). Subclinical hypothyroidism developed and the patient was subsequently started on thyroxine replacement with reduction of SSKI dose. Hypothyroidism is a rare side effect of potassium iodide.[1]

Case Report

A 60-year-old female, resident of Himachal Pradesh, presented with crusted lesions on her right leg of 2 years duration. The patient denied any history of trauma prior to onset of the lesions. The lesion started as a small papule on lower leg and over a period of 2–3 months, similar lesions appeared to involve the whole leg. This was associated with serous discharge and occasional bleeding from the lesions. The patient was a known diabetic on metformin 500 mg twice daily for the last 2 years. There was no history of any other drug intake. There was no personal or family history of thyroid disease or tuberculosis or contact with a tuberculosis patient. Her general and systemic examinations were normal. On local examination, there were multiple erythematous crusted plaques of size ranging from 1 × 1 cm to 1.5 × 1.5 cm over the right lower limb [Figure 1]. These were associated with oozing and were present in a linear pattern. There was no lymphadenopathy.

Complete hemogram and erythrocyte sedimentation rate (ESR) were normal. HIV serology was non-reactive. Chest roentgenogram did not reveal any abnormality and tuberculin skin test was 5 × 5 mm. Smear and culture tests for tuberculosis were negative. Direct microbiological examination from the lesions was negative for fungus. Histopathology from the lesions revealed pseudoepitheliomatous hyperplasia, focal ulceration, and suppurative granulomas in dermis. The granulomas were composed of neutrophils, epithelioid histiocytes, giant cells, and few lymphocytes [Figure 2a and b]. Culture on Sabouraud's Dextrose Agar (SDA) showed growth of *Sporothrix schenckii* [Figure 3].

Based on the above findings, a diagnosis of lymphocutaneous sporotrichosis was made and the patient was started on SSKI. Baseline thyroid function test was

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normal (T3: 0.89 ng/dl, T4: 6.90 µg/dl, TSH: 2.746 IU/ml, thyroid peroxidase antibody (TPOAb): 8.12 IU/ml). SSKI was started at a dose of ten drops three times a day. It was gradually increased every week to reach a dose of 32 drops three times a day after 3 weeks. Serum TSH and electrolytes were done every 3 weeks. However, at this dose TSH was found to be increased (6.281 IU/ml, reference range: 0.550-4.75 IU/ml) whereas levels of T3 and T4 were normal. The dose was reduced and oral thyroxine was started at a dose of 12.5 mcg per day. Two weeks later, TSH returned to normal (4.013 IU/ml). After 12 weeks, there was complete healing of the lesions [Figure 4]. Potassium iodide was stopped at 16 weeks. There was no recurrence 1 year after follow-up. Post-treatment thyroid function tests (TFT) were found to be normal.

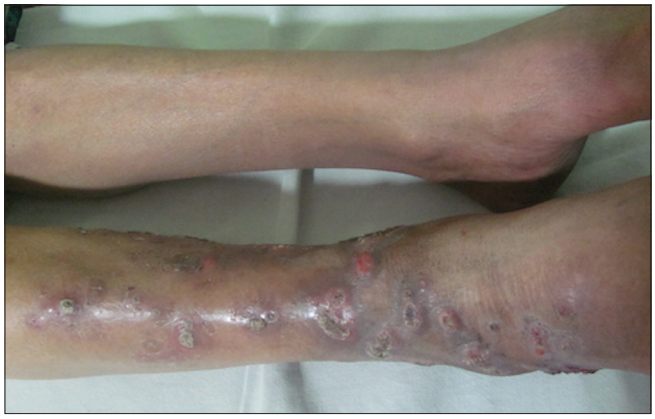

Figure 1: Erythematous crusted oozy plaques over right lower limb

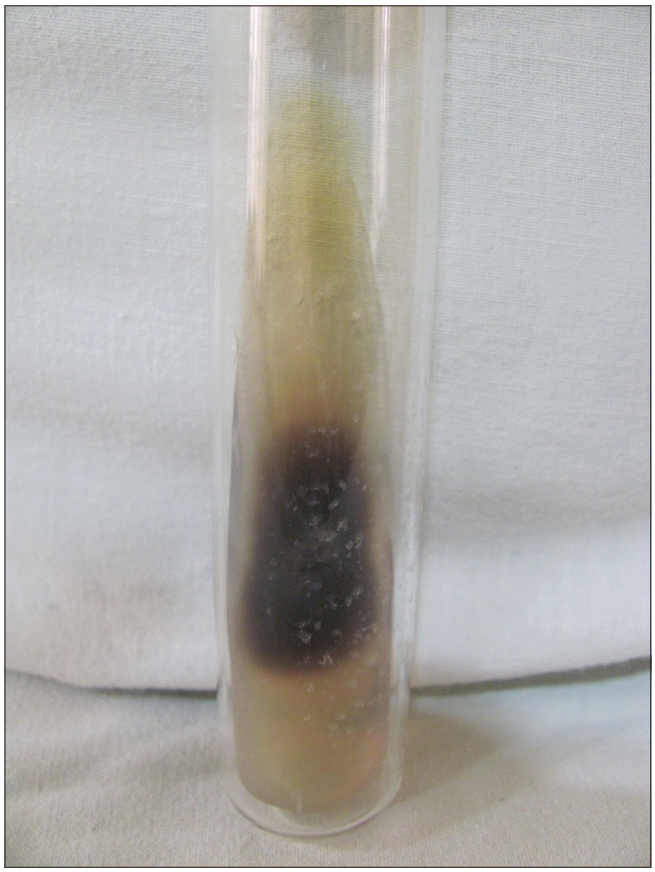

Figure 3: Colonies of *Sporthrix schenkii*: Filamentous hyaline with dark color in the center, obtained after incubation in Sabouraud's dextrose agar for 7 days

Discussion

Sporotrichosis is a deep mycotic infection caused by the fungus *S. schenckii*. The organism is usually acquired by traumatic implantation into the skin in people exposed to decaying vegetative matter. Although our patient denied history of trauma prior to onset of lesions, her occupation and residence cannot rule out the possibility of accidental trauma.

The spectrum of clinical findings varies in sporotrichosis. The manifestations can be cutaneous or systemic, the latter being rare. The involvement in cutaneous type can be either fixed or lymphocutaneous in which lesions occur along the lymphatic cord. The latter type should be differentiated from other causes of nodular lymphangitis which include atypical mycobacterial infection, cutaneous anthrax, nocardiosis, leishmaniasis, and other systemic mycosis. The diagnosis can be confirmed by isolation of the organism on culture media.

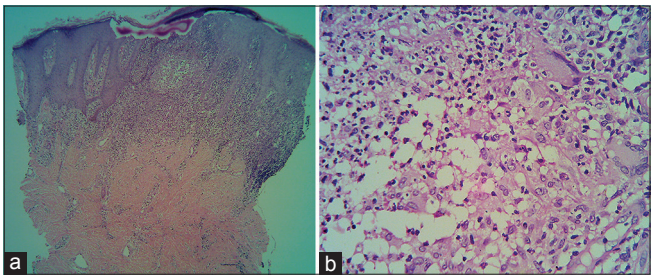

Figure 2: (a) Pseudoepitheliomatous hyperplasia and focal ulceration, underlying dermis showing dense diffuse inflammatory infiltrate (Hemotoxylin and Eosin, ×40). (b) Suppurative granulomas composed of central neutrophils with karyorrhectic debris surrounded by epithelioid histiocytes, giant cells and few lymphocytes (Hemotoxylin and Eosin, ×400)

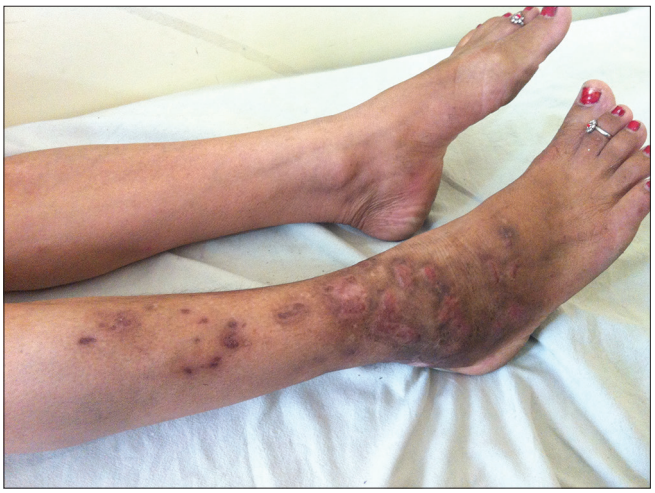

Figure 4: Complete healing of lesions at 12 weeks of treatment

There are no studies comparing the treatment options for sporotrichosis which include potassium iodide, azole antifungals, terbinafine, and localized hyperthermia. SSKI was used as a standard therapy for sporotrichosis. However, itraconazole has taken over as the drug of choice for uncomplicated cutaneous sporotrichosis.[2] It is used in a dose of 100-200 mg/day for a period of 3-6 months.[3]

We chose to treat our patient with SSKI (1gm/ml) which is a cheap and effective treatment option for sporotrichosis. SSKI is usually well-tolerated. However, it can cause cutaneous side effects like urticarial, acneiform, nodular, purpuric and erythematous skin lesions, and gastrointestinal symptoms in the form of nausea and vomiting.[4] At high doses it can also cause iodism that manifests as sneezing, coryza, parotid gland swelling, increased salivation and lacrimation, and noninflammatory lid edema.[5]

Ten weeks after start of treatment, our patient was found to have raised TSH levels (6.281 IU/ml) and was started on oral thyroxine 12.5 mcg daily. The dose of SSKI was reduced and TSH levels returned to normal after 2 weeks.

The homeostasis of thyroid hormone secretion is maintained by two regulatory mechanisms. Firstly, there is negative feedback mechanism via the hypothalamic-pituitary axis with secretion of TSH from pituitary in response to thyrotropin-releasing hormone (TRH) from the hypothalamus.[6] The second is the regulatory mechanism that exists in the thyroid itself that maintains a pool of organic iodine within the thyroid gland. This is known as autoregulation. Inbuilt autoregulation mechanism also maintains a storage pool of organic iodine in the thyroid gland and ensures that it produces enough thyroid hormone for the patient to remain euthyroid (escape phenomenon).

Excess iodide causes inhibition of organic binding of iodide in the thyroid gland which results in cessation of thyroid hormone synthesis. This is known as the Wolff–Chaikoff effect. If the autoregulation is deficient, hypothyroidism occurs as escape from WCE cannot occur.[6]

Various theories have been proposed for WCE including role of hydrogen peroxide and inhibition of the inositol triphosphate response to TSH.[7,8] WCE has clinical implication for the dermatologists using SSKI. Before starting the patient on SSKI, the personal or family history of thyroid or autoimmune disease should be sought. Medications such as amiodarone can affect thyroid function, hence it is important to inquire whether the patient is on any such medication. Since the dermatologist use KI for a long duration (usually more than a month), a screening TSH should be done. A shorter duration of therapy can allow escape from the WCE, hence baseline TSH may not be done in such cases. In cases of iodide induced hypothyroidism, KI should be discontinued and alternate therapy should be used. Thyroid hormone levels come back to normal within 1 month of iodide withdrawal.[9]

In our patient, TSH levels came back to normal after 1 month of stopping KI, even after withdrawal of thyroxine which further reinforces the fact that KI can induce hypothyroidism even in patients with no known risk factors. The decision to treat this or not is made by the treating physician.

Knowledge about the WCE is important for the dermatologists. It reinforces the need for screening and monitoring of TSH in patients where long duration therapy is being contemplated.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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