Title
Crusted scabies of the scalp in a patient with systemic lupus erythematosus

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Authors
Yee, Brittany E
Carlos, Casey A
Hata, Tissa

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Abstract

**Background:** Crusted scabies is a severe, hyperkeratotic, psoriasiform disorder associated with immune suppression. Affected individuals typically present with crusted hyperkeratotic lesions in a variety of locations. This condition can lead to severe complications: institutional outbreaks and secondary bacterial infections associated with sepsis and high mortality.

**Main observations:** A 37-year-old woman with a 12-year history of systemic lupus erythematosus treated with prednisone, methotrexate, and plaquenil presented with a three-week history of a painful scalp rash with adherent yellow scale. Skin biopsy and tissue culture were consistent with a diagnosis of crusted scabies with superficial bacterial infection. The patient was treated with oral ivermectin and permethrin cream, as well as ciprofloxacin for the bacterial infection. At one-week follow-up, the scalp was no longer tender and hyperkeratotic plaques had significantly improved. At one-month follow-up, the affected scalp demonstrated further improvement with decreasing erythema and alopecia with follicular ostia.

**Conclusions:** Our case highlights the atypical presentation of crusted scabies with primary scalp involvement and need for vigilance in recognizing and appropriately treating this condition to prevent the consequences of longstanding infection. Combination treatment with ivermectin and permethrin is appropriate management for this condition.

**Key Words:** crusted scabies, scalp, systemic lupus erythematosus

Introduction

Crusted scabies typically presents as a psoriasiform hyperkeratotic dermatosis of the hands, feet, nails, face, neck, scalp, and trunk [1]. It is typically associated with several congenital and acquired immunocompromised conditions including human immunodeficiency virus (HIV), hematologic malignancy, neurologic illnesses, and connective tissue diseases [2, 3], including systemic lupus erythematosus (SLE) [4-8].

Herein, we report an atypical case of crusted scabies with primary involvement of the scalp in a patient with systemic lupus erythematosus. Her crusted scabies was likely caused by immunosuppression from autoimmune disease and long-term prednisone treatment.
Case synopsis

A 37-year-old woman with a 12-year history of SLE treated with prednisone 15 mg daily, methotrexate 12 mg once weekly, and hydroxychloroquine 400 mg daily presented with a three-week history of a painful scalp rash. Otherwise, the patient’s medical history was unremarkable. Physical exam revealed a red plaque with adherent yellow scales on the occipital scalp (Figure 1).

The initial differential diagnoses included psoriasis or impetiginized psoriasis; the patient complained of severe pain. She was started on fluocinolone acetonide 0.01% topical oil and doxycycline for suspicion of impetiginized psoriasis. Skin biopsy of the occipital scalp was performed owing to the atypical nature of her pain. Biopsy demonstrated marked hyperkeratosis with numerous mites and ova within the keratotic crust with an underlying inflammatory infiltrate with eosinophils consistent with crusted scabies (Figure 2). Tissue culture grew heavy enterobacter cloacae. The patient was treated with oral ivermectin (administered at 0.2 mg/kg on days 1, 2, 8, 9, 15, 22, and 29), permethrin cream (applied nightly for 7 days continuous, then twice weekly for two weeks), and ciprofloxacin for enterobacter infection. At one-week follow-up, the scalp was no longer tender and the hyperkeratotic plaques had significantly improved (Figure 3A). At one-month follow-up, her occipital scalp demonstrated some blotchy erythema and alopecia with follicular ostia (Figure 3B). The patient had symptomatic improvement with decreased itching and pain, and symptoms have since completely resolved.
Crusted scabies is a severe hyperkeratotic, psoriasiform dermatosis. Unlike typical scabies, adults with crusted scabies may lack the characteristic rash or itching. Sites of presentation have been reported on the scalp, face, neck, extremities, trunk, hands, and feet [1, 9, 10]. Crusted scabies has also presented as onychodystrophy, making the diagnosis sometimes extremely difficult [11]. Owing to its presentation, this condition is often misdiagnosed as psoriasis, eczema, ichthyosis, or a drug reaction [1]. These hyperkeratotic plaques can carry up to 4000 mites per gram of skin compared to about 20 mites on the entire skin of individuals with ordinary scabies [12]. This hyper-infestation can lead to institutional outbreaks. Additionally, these plaques are known to slough off and cause fissuring, which predisposes to secondary bacterial infections [10] associated with sepsis and higher mortality [12, 13]. Our patient’s primary scalp involvement demonstrates an atypical presentation of crusted scabies, which should be considered in patients who are immunosuppressed. The presence of hyperkeratotic scalp plaques without any other areas of involvement may mimic the appearance of psoriasis and pose as a significant diagnostic challenge. In addition, the patient’s severe pain and super infection in her immunocompromised state highlight the need for appropriate care to avoid further morbidity and possible mortality.

Combination treatment including oral ivermectin and topical agents such as permethrin or benzyl benzoate is generally recommended for this highly contagious scabies variant [1, 13-15]. However, no comparative studies of the safety and efficacy of different therapies for crusted scabies have been performed [13, 16]. Our patient’s infection responded well to the combination of oral and topical treatments. An additional goal of treatment would be to lessen any immunosuppression in those patients who may tolerate dosage adjustments.

Conclusions

Our case highlights the atypical presentation of crusted scabies of the scalp alone and need for vigilance in recognizing and appropriately treating this condition to prevent the consequences of longstanding infection. Combination oral ivermectin and topical permethrin cream provided significant clinical improvement within one-week of treatment. Medications are generally well tolerated, and early diagnosis and treatment can prevent significant morbidity in this often misdiagnosed disease.

References


