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Case Report

Unveiling crusted scabies: From identification to intervention

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Abstract

Crusted scabies is a rare and highly contagious form of scabies marked by an overwhelming infestation of Sarcoptes Scabiei mites in the skin, especially in immunocompromised, disabled and malnourished individuals. It is characterized by large crusted lesions, extensive yellow to brown scales and thick hyperkeratosis. The condition poses a significant diagnostic challenge due to its resemblance to other dermatological disorders, often leading to delayed treatment and increased risk of transmission.

Here, we present the case of a 70-year-old male with yellowish-brown hyperkeratotic lesions. The patient has a known history of uncontrolled type 2 diabetes mellitus and hypothyroidism for one year, which likely contributed to disease susceptibility and severity. Despite its rarity, early recognition and timely intervention are essential to prevent outbreaks, especially in institutionalized and immunocompromised populations.

This case highlights the challenges in diagnosing and treating crusted scabies, aiming to increase awareness among healthcare providers and emphasize the importance of vigilance in identifying and addressing this rare condition.

Keywords: Crusted scabies, Burrow, Delta sign, Jetliner with contrail sign, Permethrin, Ivermectin.

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1. Introduction

Scabies is a parasitic infestation caused by human-specific ectoparasite Sarcoptes scabiei var. hominis and presents with skin lesions of varying severity associated with itching. It has worldwide distribution, particularly in densely populated, tropical, economically disadvantaged regions, as well as in areas with inadequate healthcare services. It has been recognized as a neglected tropical disease. Crusted scabies, formerly referred to as "Norwegian" scabies, is a rare and more severe variant of scabies. It is commonly seen in individuals who are immunocompromised, malnourished, or disabled.[1]

It is frequently reported in immunocompromised patients especially in those using corticosteroids, infected with HIV or HTLV-1, organ transplant recipients and individuals with hematologic malignancies. It develops primarily due to inability of immune system to control the proliferation of the mites.[2]

It typically does not present as an acute eruption, as in classical scabies. Instead, it develops gradually and progresses slowly. Itching, a common symptom of scabies, is usually minimal or absent.[3]

It is marked by extensive crusted lesions, widespread scaling, hyperkeratosis, and extensive mite infestation. Crusted scabies poses a significant public health concern, affecting not only individuals but also families and entire communities. It spreads through direct contact with infected individuals and also through indirect contact with contaminated materials such as bedding and clothing. If not diagnosed promptly, crusted scabies can lead to severe complications, including secondary impetigo, cellulitis, and sepsis, with a high risk of mortality. Due to its hyper infestation, it has the potential to trigger major outbreaks, making it highly contagious.[1,3]

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2. Case Report

Here is a case of a 70-year-old male who presented with widespread yellow-brown thickened lesions over the body since past two months, accompanied by mild itching. The lesions first appeared on the hands and gradually spread to other areas of the body. He had been applying topical corticosteroids to the lesions for five weeks, but they showed no improvement. He also had a one-year history of uncontrolled type 2 diabetes mellitus and hypothyroidism.

On examination, multiple well to ill defined hyperkeratotic plaques of variable size, covered with yellowish-brown crusts were observed on the bilateral hands and feet, lower abdomen, thighs, groin, penis, scrotum, and buttocks, resembling a 'built-up sand' appearance (**Figure 1a,b,c**). The face was spared from any lesions. Laboratory investigations revealed an elevated HbA1c level (10.4%), increased TSH (11.41 mIU/L), and low haemoglobin (10.5 g/D). Other baseline investigations were within normal limits. Serological tests for HIV, hepatitis B, and hepatitis C were nonreactive.

Figure 1: Distinct, yellowish-brown hyperkeratotic crusted lesions with a "built-up sand" appearance observed on; **a:** Dosrum of both feet; **b:** Buttocks, and; **c:** Right hand; **d:** Regression of hand lesions post treatment.

Figure 2: Direct microscopic examination of potassium hydroxide (KOH)–mounted skin scrapings revealed Sarcoptes scabiei mites at 40x magnification

Direct microscopic examination of KOH mounted skin scrapings revealed the presence of mites and eggs (**Figure 2,3**) On dermoscopy, multiple curvilinear burrows arranged in noodle-like pattern, displaying the grey-edged line sign (red arrow), delta sign (green arrow), and jetliner with contrail sign (blue arrow) were seen. (**Figure 4**). Histopathology revealed hyperkeratosis, acanthosis, scabies mite in stratum corneum (black arrow) and mixed inflammatory infiltrate composed of plasma cells and lymphocytes in dermis. (**Figure 5**)

Figure 3: Direct microscopic examination of potassium hydroxide (KOH)–mounted skin scrapings revealed Sarcoptes scabiei mites at 40x magnification mite eggs at 40x magnification, 4.

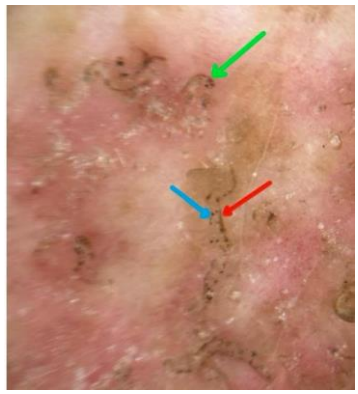

Figure 4: Dermoscopy showing numerous curvilinear burrows arranged in a noodle-like pattern, displaying the grey-edged line sign (red arrow), delta sign (green arrow), and jetliner with contrail sign (blue arrow)

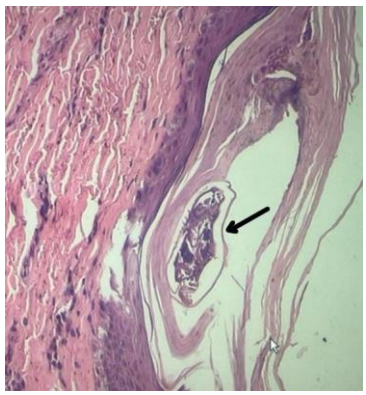

Figure 5: Histopathology showing *Sarcoptes scabiei* mite (black arrow) within the epidermis using hematoxylin and eosin (H&E) staining at 10x magnification.

Patient was prescribed oral ivermectin 12 mg on days 1, 2, 8, 9, and 15, along with daily application of 5% permethrin for seven days, followed by twice-weekly applications for the next three weeks. Supportive treatment consisted of a keratolytic agent (3% salicylic acid in white petroleum

ointment), emollients, and antihistamines. Close contacts of the patient were also treated. Resolution of lesions and improvement of patient's condition was seen after completion of treatment. (**Figure 1d**)

3. Discussion

Crusted scabies is a rare, highly contagious and severely debilitating form of disease characterized by extensive hyperkeratosis and crusting of skin. It is also known as Norwegian scabies, as it was first documented by Boeck and Danielssen in Norwegia in 1848 among leprosy patients.[3] This condition occurs as a result of the host's impaired lymphocytic response, allowing excessive mite infestation within the stratum corneum.[4] It is commonly seen in individuals with impaired T-cell immunity or those who have reduced skin sensation and a diminished ability to physically remove the mites.[5] It is primarily transmitted either through close direct contact or indirect transmission via contaminated items like clothing or bedspreads.[1]

Typically, localized thick crusted plaques that varies in thickness from 3 to 15 mm are seen. These crusts are creamy, grey, yellow-brown, or yellow-green in colour and are tightly attached to the skin. It can be complicated by secondary bacterial infections like impetigo, ecthyma, cellulitis and lymphangitis.[3,5]

Its morphology closely resembles conditions such as seborrheic dermatitis, atopic dermatitis, psoriasis, dyshidrotic eczema, hyperkeratotic eczema, palmoplantar keratoderma, Sézary syndrome, and erythrodermic mycosis fungoides and should therefore be clinically distinguished from them.[6]

The diagnosis can be further confirmed through microscopic examination of skin scrapings, dermoscopy, and histopathological analysis. Various eponyms are used to describe characteristic dermoscopic manifestations of scabies. The first is the Delta sign, characterized by brown triangular structures representing the mite's head and anterior legs. Another notable sign is the Jetliner with Contrail Sign, where the mite's body appears translucent, and the air-filled burrow resembles a jetliner with a contrail. Noodle Pattern or Millipede-like structure, depicts multiple burrows and is a key dermoscopic sign. The grey-edged line sign features a dark line on the outer wall of the curved burrow, formed by melanin-rich fecal material left by the mite. Histopathological examination can further establish the diagnosis by identifying mites or their fragments in the subcorneal layer of the tissue section.[7]

In our case, the patient presented with extensive, thick, yellow-brown crusted lesions covering large areas of the body. Given the minimal itching and multiple risk factors—including diabetes, hypothyroidism, and improper topical corticosteroid use—crusted scabies was considered a key differential diagnosis. Characteristic dermoscopic findings, along with the visualization of mites on microscopic examination and histopathology, further confirmed our diagnosis.

Numerous factors like immunosuppression, heavy mite burden, thick hyperkeratotic plaques, and reduced absorption of topical treatments due to extensive crusting makes the treatment challenging.[1] The key principles in management involve isolating patients until complete recovery, choosing an efficient treatment plan using scabicidal drugs and keratolytic agents, providing symptomatic relief with antihistamines and antibiotics for secondary infections and educating patients and their families.[8]

Crusted scabies requires a more aggressive treatment approach compared to classic scabies. The preferred scabicidal treatment is a combination of topical permethrin and oral ivermectin, which cause paralysis and death of mite by disrupting the neuronal sodium channel repolarization and blocking gamma-aminobutyric acid (GABA) receptors respectively. 5% permethrin is used daily every night for 12 h duration for seven days, then twice weekly until resolution along with oral ivermectin (200 µg/kg) on day 1, 2, 8, 9, and 15. In more severe cases, additional doses of ivermectin can be added on day 22 and 29 after initiating the treatment.

Other adjuvant drugs like keratolytic agents such as 5–10% salicylic acid and 40% urea are needed to remove crusts and thereby increase the effectiveness of a topical scabicidal agent.[8,9]

Effective management of scabies also requires the implementation of certain general measures. The patient must be strictly isolated to prevent outbreaks, while close contacts and hospital staff should take necessary precautions to avoid infestation. In addition to treating patients, it is crucial to also treat family members and individuals who are in close contact, irrespective of their symptoms. Environmental decontamination also plays an essential role Items such as sofas, bedding, towels, and clothes should all be dry-heated for ten minutes at 600°C or cleaned in water at temperatures above 500°C. Furniture and floors should be cleaned using a vacuum cleaner, while non-washable items can be treated with insecticides.[8,10]

A literature review by LJ Robert et al. on 78 patients of crusted scabies demonstrated that notable decline in mortality in cases was primarily attributed to the intensified ivermectin regimen. Furthermore, the early initiation of antibiotics for suspected secondary bacterial sepsis significantly contributed to improved patient outcomes. This comprehensive approach highlights the critical role of intensive management in effectively managing severe cases of crusted scabies.[3]

4. Conclusion

Crusted scabies being a highly transmissible disease, requires early diagnosis and aggressive management to prevent complications and outbreaks. A combination of oral ivermectin, topical scabicides, and thorough environmental

decontamination is needed to eliminate scabies infestation. Timely intervention, multidisciplinary care, and simultaneous treatment of close contacts are needed to achieve complete resolution and reducing the risk of recurrence. Raising awareness among medical professionals can facilitate early detection and timely treatment, ultimately improving patient outcomes and public health.

5. Source of Funding

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6. Conflict of Interest

None.

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