Crusted Scabies Complicated with Herpes Simplex and Sepsis
Arghavan Azizpour, Maryam Nasimi, Alireza Ghanadan1,2, Fariba Mohammadi, Safoura Shakoei3

Abstract
Crusted scabies is a rare and extremely contagious infestation by Sarcoptes scabiei. Kaposi’s varicelliform eruption (KVE) refers to herpes simplex virus infection superimposed on pre-existing dermatosis such as atopic dermatitis, Darier’s disease, and pemphigus. We report a case of KVE superimposed on crusted scabies in a middle-aged woman. Her condition was complicated with sepsis. She was treated with IV meropenem, vancomycin, and acyclovir and was released 2 weeks later in good condition. To our knowledge, only rare cases of crusted scabies complicated by KVE have been reported.

Key Words: Crusted scabies, kaposi varicelliform eruption, scabies

Introduction
Crusted scabies is an uncommon and severe form of infestation induced by the mite Sarcoptes scabiei var. hominis, which was first described in a leprosy patient. This is characterized by diffuse hyperkeratotic plaques, commonly involving the scalp, face, and intertriginous areas.[1] Crusted scabies is associated with conditions, such as old age, institutionalized hosts, human immunodeficiency virus, human T-cell lymphotropic virus type 1 (HTLV-I), and other immunosuppressive conditions.[2,3] In contrast to conventional scabies, severe pruritus or burrows are rare in these patients.[4,5]

Kaposi varicelliform eruption (KVE) is an extensive herpes simplex virus infection superimposed on pre-existing dermatosis. KVE has been found in several skin diseases including atopic dermatitis, Darier’s disease, and pemphigus diseases.[6,7]

To the best of our knowledge, only one case of crusted scabies in association with KVE without underlying disease has been reported till date.[11]

Herein, we report another case of KVE superimposed upon crusted scabies that was complicated with sepsis in a 43-year-old woman.

Case Report
A 43-year-old woman with a 4-year history of moderate plaque-type psoriasis presented to our hospital with diffuse erythema and scaling. She reported exacerbation of cutaneous lesions 20 days before admission.

She had been treated with the diagnosis of psoriasis exacerbation by topical corticosteroid, oral methotrexate (MTX) 10 mg/week, and prednisolone 5 mg/d in another clinic. However, as the patient had shown no significant improvement after 2 weeks of treatment, she had been referred to our department. She did not complain of significant itching. Family history was non-contributory.

Physical examination revealed large, yellow-erythematous hyperkeratotic plaques on the trunk, extremities, face,
Azizpour, et al.: Complicated crusted scabies

and the scalp [Figure 1a and b]. Microscopic examination of scales revealed numerous scabies mites.

Histopathologic examination revealed crusted epidermis containing numerous mites with superficial invasion of epidermis leading to erosions [Figure 2a].

The patient was diagnosed with crusted scabies. Subsequently, she was treated with topical permethrin 5% cream, which she applied 7 times a week, because of lack of oral ivermectin in our region. Moderate improvement of hyperkeratosis resulted.

The patient developed fever (39°C) and punched out erosions on the face and tongue and clusters of vesicles on the neck appeared on the fourth day of admission. The lesions spread and gradually involved the anterior and posterior surface of the trunk and arms [Figure 3a and b]. Tzanck smear of this punch out lesions was positive for herpes virus.

Histopathologic examination revealed superficial erosions, keratinocytes with glassy appearance of nuclei, and multinucleated giant cells suggestive of viral inclusion bodies [Figure 2b]. Real time polymerase chain reaction (PCR) for herpes simplex virus (HSV) was performed by ready-to-use molecular detection HSV-1/2 RG PCR kit (Qiagen) and showed positive result (Ct 21.60, standard control Ct 35.42) [Figure 2c]. The diagnosis of KVE was confirmed by these tests. Culture from skin lesions and blood culture were positive for Staphylococcus aureus and Citrobacter. The laboratory results revealed anemia, and the erythrocyte sedimentation rate was elevated up to 90 mm/h. The patient was treated with intravenous (IV) colistin (4.5 million units BD), vancomycin (1 g BD), and ciprofloxacin (400 mg IV BD) for bacterial superinfection, and acyclovir 1500 mg/d for herpes infection. However, 2 days later, her condition deteriorated, hemoglobin level decreased from 9.8 to 7.3 g/dl, and creatine phosphokinase and lactic dehydrogenase levels were increased. The patient was transferred to internal medicine clinic for hemolysis due to sepsis. She was treated with IV meropenem (1 g three times daily (TDS)), vancomycin (1 g twice daily BD), and acyclovir. She was released 2 weeks later in good condition.

Discussion

Crusted scabies is a severe form of skin infestation. Unlike conventional scabies, pruritus in crusted scabies may be mild or absent. Because of to the atypical clinical presentation, the diagnosis is frequently delayed.\(^1\)

Several underlying diseases may predispose patients to crusted scabies including acquired immune deficiency syndrome (AIDS), HTLV1 infection, T-cell lymphoma, leukemia, Down syndrome, iatrogenic immunosuppression, heavy ethanol use, past leprosy, moderate immunosuppression in transplant recipients, systemic lupus erythematosus, rheumatoid arthritis, diabetes mellitus, malnutrition, various neuropathies,\(^2\) and topical corticosteroids.\(^3\)
Reactive keratinocytic hyperplasia marked by fine lamellar desquamation, and commonly, palmoplantar hyperkeratosis and nail involvement result from colonization of epidermis by mites.\[1,4\]

Accordingly, clinical presentation of the disease may lead to misdiagnosis with other inflammatory, non-infectious dermatosis such as erythrodermic psoriasis, hyperkeratotic eczema, and T-cell lymphoma as seen in our case.\[1\]

Crusted scabies must be considered in any patient with generalized erythrodermic scaling dermatitis.

Our patient was previously misdiagnosed as psoriasis, and at the time of her visit to our clinic, she was receiving topical clobetasol propionate and oral MTX. She had then developed punched out lesions consistent with eczema herpeticum and had been treated with intravenous acyclovir.

KVE is commonly described as painful, edematous, and disseminated vesiculopustules on the skin affected with a pre-existing skin disease. These lesions progress to painful punched out erosions that are susceptible to secondary bacterial colonization.\[6\]

KVE has been described in atopic dermatitis (eczema herpeticum), Darier’s disease, pemphigus, pityriasis rubra pilaris, Hailey–Hailey disease, cutaneous T-cell lymphoma, seborrheic dermatitis, psoriasis, Wiskott–Aldrich syndrome, congenital ichthyosiform erythroderma, and Sézary syndrome.\[6,7,9\] To our knowledge, only three crusted scabies patients with KVE have been reported till date. These patients had a history of cutaneous lymphocytic leukemia, renal transplantation, and diabetes mellitus, respectively;\[3\] however, the patient cited in this case had no past medical history of any such diseases.

KVE is a potentially life-threatening condition because of secondary sepsis or underlying disease.\[2\]

Patients with crusted scabies have usually an underlying immunosuppressive disease. However, 40% of the patients lack an identifiable risk factor.\[2\] Skin breakdown related to crusted scabies and herpes infection was the most likely source of bacteremia.

In conclusion, crusted scabies remains a debilitating condition with potentially high mortality. The mortality rate of crusted scabies has been reported to be up to 50% over 5 years.\[2\] A delay in diagnosis and treatment leads to spreading of the infestation with increasing risk of sepsis.

**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.

**Financial support and sponsorship**

This study was supported financially by the Tehran University of Medical Sciences, Tehran, Iran.

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Sunderkotter C, Feldmeier H, Fölster-Holst R, Geisel B, Klinke-Rehbein S, et al. S1 guidelines on the diagnosis and treatment of scabies - short version. J Dtsch Dermatol Ges 2016;14:1155-67.
2. Roberts LJ, Huffam SE, Walton SF, Currie BJ. Crusted scabies: Clinical and immunological findings in seventy-eight patients and a review of the literature. J Infect 2005;50:375-81.
3. Shaw K, Smith S, Driscoll M. Scabies herpeticum: Crusted scabies with herpes simplex superinfection. G Ital Dermatol Venereol 2017;152:192-3.
4. Yelamos O, Mir-Bonafé JF, López-Ferrer A, Garcia-Muret MP, Alegre M, Puig L. Crusted (Norwegian) scabies: An under-recognized infestation characterized by an atypical presentation and delayed diagnosis. J Eur Acad Dermatol Venereol 2016;30:483-5.
5. Jungbauer FH, Veenstra-Kyuchukova YK, Koeze J, Krujit Spanjer MR, Kardaun SH. Management of nosocomial scabies, an outbreak of occupational disease. Am J Ind Med 2015;58:577-82.
6. Vora RV, Pilani AP, Jivani NB, Kota RK. Kaposi varicelliform eruption. Indian Dermatol Online J 2015;6:364-6.
7. Fujii M, Takahashi J, Honma M, Ishida-Yamamoto A. Kaposi’s varicelliform eruption presenting with extensive skin lesions and sepsis. J Dermatol 2017;44:1180-1.
8. Bilan P, Colin-Gorski AM, Chapelon E, Sigal ML, Mahé E. [Crusted scabies induced by topical corticosteroids: A case report]. Arch Pediatr 2015;22:1292-4.
9. Lehman JS, el-Azhary RA. Kaposi varicelliform eruption in patients with autoimmune bullous dermatoses. Int J Dermatol 2016;55:e136-40.