

Generalized erythroderma with crusted plaques in a diabetic female patient

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A 70-year-old female patient presented with generalized erythematous, scaly skin lesions scattered all over the body for 20 days duration associated with slight pruritus. The patient was a known case of uncontrolled diabetes on insulin therapy. There was no past history of any previous skin disease. Cutaneous examination revealed erythematous scaly plaques with crustation scattered on chest, abdomen, back, upper limb and inner aspect of thighs (Fig. 1, 2, 3). General physical and systemic examination did not reveal any abnormality except for



Fig. 3 Crusted erythematous plaques in inner sides of thighs.

diabetes mellitus. Laboratory investigation revealed eosinophilia with high blood glucose level.



Fig. 1 Diffuse areas of erythema, scaling and crustation with areas of lichenification involving chest and neck.



Fig. 2 Crusted erythematous plaques on the abdomen.

What is your clinical differential diagnosis?

1. Psoriasis
2. Erythroderma
3. Cutaneous lymphoma
4. Eczema
5. Crusted (Norwegian) scabies

The specimen from the crusted plaques on the abdomen was scraped with a blunt scalpel and placed on a glass slide. A drop of KOH and a cover slip are placed on it. The microscopic examination revealed presence of scabies mites confirming the diagnosis of crusted (Norwegian) scabies (Fig. 4). Screening test for HIV was negative. Histopathological examination for a biopsy from the abdomen revealed slight perivascular lymphohistiocytic infiltrate. The epidermis showed multiple grooves in stratum corneum, some of these grooves contained



Fig. 4 A scraping for KOH mount from the crusted plaques on the abdomen revealed presence of scabies mites.

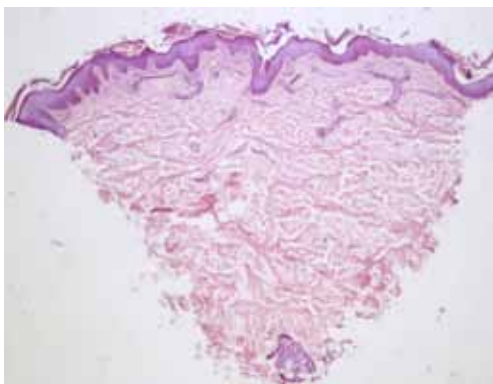


Fig. 5 Superficial lymphohistiocytic infiltrate, multiple grooves in the epidermis.

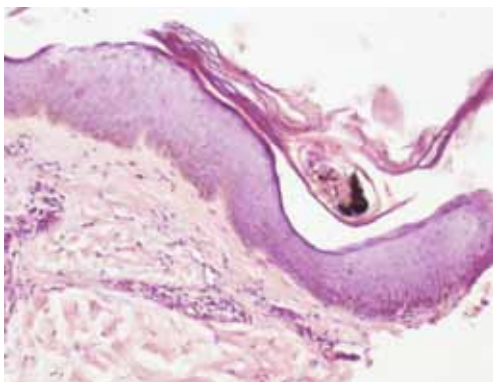


Fig. 6 Within these grooves sub-corneal structure was seen (scabies mite).

scabies mites. These histological features were consistent with a diagnosis of Crusted (Norwegian) scabies (Fig. 5, 6).

The patient was treated with ivermectin 12mg which was repeated after week, antihistaminic and topical gamma benzene hexachloride lotion on first day and repeated after one week. She improved with this treatment.

DISCUSSION

The first description of crusted scabies or Norwegian scabies (NS) was from Norway in the mid 1800s.^{1, 2, 3} It is a clinically distinct and highly contagious form of scabies. In common scabies, the number of parasites infesting the epidermis is relatively small. Such restriction is basically attributable to mechanical destruction of burrows by scratching, regular cleansing and cell mediated immune response of host. Millions of mites colonize the epidermis inducing characteristic hyperplastic changes seen in NS. This results from failure of the host immune response to control the proliferation of mites in the skin leading to hyperinfestation and concomitant inflammatory reaction. Patients chronically treated with immunosuppressive drugs as in post renal transplant patients, or those with HIV, HTLV- I infection are more prone to develop NS. Prolonged use of corticosteroids may induce NS. It has also been described in patients with severe systemic diseases such as leprosy, rheumatoid arthritis, diabetes mellitus, systemic lupus erythematosus, leukemia and in patients who do not scratch either because of an absence of pruritus or due to immobility such as in mental illness, sensory neuropathy, paresis and senility.^{4, 5, 6}

NS is characterized clinically by extensive hyperkeratosis and crusting of the skin especially on the acral areas. The lesions are primarily distributed on the scalp, face, extremities, back and around the nail folds. Pruritus may be moderate to severe. A variable erythema is common evolving sometimes into erythroderma. As in our case, it was presented with erythroderma and crusting. In view of the hyperkeratosis and crusting lesions, NS may be confused with psoriasis, keratosis follicularis, contact dermatitis and seborrhe-

ic dermatitis which may delay on diagnosis and prompt treatment. A delay in diagnosis of even a single case of NS can lead to a massive outbreak of scabies among patients' family members and health care personnels in hospitals and in mental asylums.⁷ Our patient was with known risk factors for the development for NS. She was old age with uncontrolled diabetes.

In histopathology of crusted scabies, the epidermis shows marked orthokeratosis, parakeratosis, acanthosis and dermal cellular infiltrate. The subcorneal layer shows burrows containing female mites. Every section shows many burrows.

The mites can be seen in all stages of development: Adult, larval, nymph and egg. Foci of spongiosis, spongiform pustules and neutrophilic abscess are seen in the spinous zone. Dense superficial and deep perivascular infiltrate of numerous plasma cells are seen in the dermis. Eosinophils and neutrophils are seen around the blood vessels at all levels of the dermis, especially the upper half.^{8,9} NS represents a serious therapeutic problem. It is very resistant to treatment, relapses frequently. In contrast to classical scabies where a single application of topical scabicide would suffice, treatment application needs to be repeated every four to seven days. The introduction of oral ivermectin to therapeutic armamentarium has revolutionized the treatment of scabies.¹⁰ Our patient was treated with ivermectin 12mg which was repeated after week along with topical gamma benzene hexachlorid lotion (GBHC) with improvement in her condition considerably.

Our message is that, the possibility of crusted scabies as a cause for erythroderma should be in mind especially, in immunocompromized patients even if they do not itch.

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