



Erythroderma Following Systemic Corticosteroid Therapy Caused by Crusted Scabies in Immunoglobulin a Nephropathy

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Abstract

We here report a case of a 15-year old boy developed systemic diffuse erythema caused by crusted scabies, which appeared during her treatment with systemic corticosteroid therapy for IgA nephropathy. At first, skin lesions were misdiagnosed as drug eruption or exfoliative dermatitis and its conventional treatment is ineffective. For further diagnosis, pathological examination of skin lesion was taken and demonstrated scabies in stratum corneum. Treatments with sulphur ointment for 2 weeks led to the complete resolution of his skin condition. To conclude, the possibility diagnosis as scabies should be considered in nephrotic patients, who were applied with prolonged use of corticosteroids and associated skin lesions and pruritus.

Keywords: Nephropathy; Erythroderma; Corticosteroid therapy; Crusted scabiei

Introduction

Crusted scabies is a rare variant of classic scabies and has a high mortality of up to 50% over 5 years [1]. Crusted scabies usually associated with underlying immunodeficiency. Crusted scabies is characterized by presence of huge number of sarcoptes scabiei in the horny layer of the epidermis. Typical skin lesions are the thick horny layering and warty crusts [2]. However, with the topical or systemic use of corticosteroids, the clinical manifestation of crusted scabies may be masked, and misdiagnosed as other skin diseases, such as erythroderma, exfoliative dermatitis and eczema etc and it is always challenge to make the correct diagnosis of crusted scabies being challenge. Here we report a case of 15-year old boy with IgA nephropathy developed systemic diffuse erythema caused by crusted scabies.

Case Presentation

A 15-year old boy with a history of IgA nephropathy was admitted to our inpatient because of systemic diffuse erythema, scaling and itching for 2 months. About 2 months ago, he was diagnosed with IgA nephropathy by renal biopsy and always took multiple drugs including 20 mg/d methylprednisolone orally and traditional Chinese Medicine. Then he presented with generalized erubescence and scaling of the scalp, neck, trunk, arms, forearms, thighs, genitalia, gluteal region, dorsum of hands and webs of fingers (Figure 1A). The cuticle of both hands and feet was obviously thickened and cracked without any seepage (Figure 1B). We treated he as drug eruption and exfoliative dermatitis by oral administration of 20 mg/d methylprednisolone and several antihistamine drugs for 2 months and applied glucocorticoid ointment, boric acid ointment etc. All the drugs for IgA nephropathy except methylprednisolone were discontinued. However, these treatments were without any improvement.

For further diagnosis, skin biopsy of a erythematous lesion in belly was taken and showed the typical pathological characteristics of erythroderma and scabies. Pathological examination showed parakeratosis, scabies in stratum corneum, stratum spinosum hypertrophy, sponge edema, dilating blood vessels, the density of the perivascular mononuclear cell infiltrating with eosinophils in the dermis (Figure 2). The diagnosis of erythroderma was made, which was caused by crusted scabies. After treated with sulphur ointment, namely applied the whole body except face twice a day for

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Figure 1: Clinical photograph showing skin lesions of peeling skin over the (A) face and (B) bilateral lower limbs.

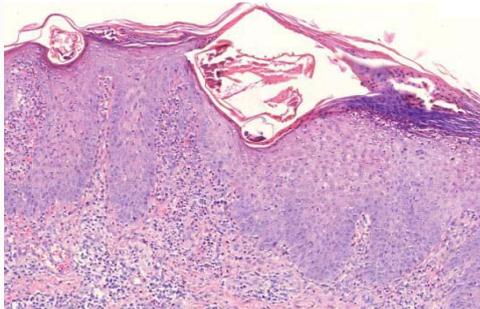


Figure 2: Pathological examination showed parakeratosis, scabies in stratum corneum, stratum spinosum hypertrophy, the density of the perivascular mononuclear cell infiltrating with eosinophils in the dermis.

3-4 days per week without bathing, the generalized erubescence was restored 90%, and intense itching had greatly relieved. Only applied sulphur ointment for 2 weeks, the lesions were cured.

Discussion

Erythroderma is the condition where almost the whole body surface characterized by erythema and desquamation. The causes of erythroderma can be classified as the following: eczemas, psoriasis, drugs, malignancy, other causes which only account for 0.5% cases, including scabies, lichen planus and some genetic disease etc [3]. The diagnosis of erythroderma is not difficult. The most important is looking for the cause of erythroderma and then treat it. Besides, we should observe the curative effect, after routine treatment of taking orally and externally glucocorticoids and. And when treating without improvement, we need to find out all the probably common and rare causes by pathological examination and other techniques so as to facilitate diagnosis and treatment disease, avoid missed diagnosis and misdiagnosis. Early finding the causes of erythroderma and effective management significantly reduce mortality and morbidity of this potential dermatologic emergency [4].

The key clinical feature of this case is realizing that unusual infections should be considered in patients receiving systematic steroids. Steroid use and nephropathy may be risk factors in developing disseminated forms of otherwise infection diseases. Crusted scabies is a rare, highly contagious uncommon form of scabies contagious. It is related to the debilitating skin condition. Clinically, crusted scabies is a hyperkeratotic skin disease and it could generalize to erythroderma [5,6].

Crusted scabies may be a complication of immunosuppressant therapy and it may also appear in immunocompromised patients, such as in patients with prolonged use of corticosteroids, both topically and systemically, as our case. We have hypothesized that such

nephrotic patients may have an immunosuppression predisposing them to hyperinfestation of scabies. The possibility of development scabies should be considered in nephrotic patients with prolonged use of corticosteroids and associated skin lesions and pruritus. It is very important to make a early correct diagnosis of crusted scabies for its contagious nature, and high mortality either from secondary sepsis or from the underlying predisposing condition present in many patients.

Prolonged use of corticosteroids in patients with IgA nephropathy is associated with an increased risk of crusted scabies infection, whereas failure to control scabies effectively can further aggravate the nephropathy disease. Some cases of epidemic scabies and acute glomerulonephritis were reported [7]. Concerning the causes of acute glomerulonephritis in scabietic patients, there are two points of view. One is suggested that acute glomerulonephritis is caused by secondary bacterial infection. The skin lesions were caused by scratching. Another point of view is that scabies together with immunoglobulin form immune complex causing the glomerular injury and leading the nephritis [8].

We could say that nowadays this diagnosis become challenge for the both urologist and dermatologists. The aim to report this case is to provide more details of the clinical features, and to increase the vigilance among physicians in patients with IgA nephropathy associated with crusted scabies infection.

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