Erythroderma, Scaly Scalp and Nail Dystrophy: A Misleading Association

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Introduction

Erythroderma refers to a generalized or nearly generalized sustained erythema of the skin, involving more than 90% of the body surface accompanied by a variable degree of scaling [1]. Systemic complications of erythroderma include infection, fluid and electrolyte imbalances, thermoregulatory disturbance, high output cardiac failure and acute respiratory distress syndrome. Accordingly, Rapid diagnosis and treatment of erythroderma continues to be a challenge to the physician [2]. According to Xiao-Ying and colleagues in 2010, erythrodermic patients are categorized into four groups: erythroderma due to pre-existing dermatoses, drug induced erythroderma, malignant erythroderma and idiopathic erythroderma [3].

Erythrodermic psoriasis represents 25 % of cases presenting with erythroderma [4]. In addition to the classic clinical picture of erythroderma, clues to identifying erythrodermic psoriasis include history of pre-existing psoriasis, nail abnormalities, scaly scalp and psoriatic arthritis [4-5].

Case Report

A 75 year old female presented to Al- Minya university outpatient dermatology clinic with generalized erythroderma. The condition started 4 years ago with itching associated with few erythematous plaques on the trunk. She did not seek medical advice and she only used topical emollients. The condition gradually progressed to involve most of the body surface area.

Apart from slight mental challenge, general examination was normal. Skin examination showed generalized erythema with areas of oozing and crustations, scaly scalp and dystrophic nails (Figure 1). Routine laboratory investigations revealed no abnormalities apart from mild anaemia and mild hypo-albuminemia.

Based on the triad of erythroderma, scaly scalp and nail dystrophy; the provisional diagnosis was erythrodermic psoriasis, so a biopsy was taken to confirm the diagnosis. The biopsy revealed psoriasiform hyperplasia with the stratum corneum containing numerous scabies mites and eggs (Figure 2). These histopathological findings were confirmed with scalp scraping and KOH staining which revealed numerous mites so, the final diagnosis was crusted scabies (Figure 3).
Figure 2: A) H&E stained skin biopsies showing psoriasiform hyperplasia and mild superficial dermal inflammatory infiltrate. Sarcoptes scabiei eggs and mites were found within the stratum corneum. B) Higher power showing the sarcoptes scabiei egg

Figure 3: KOH scalp scraping showing the sarcoptes Scabiei mite

Discussion

Crusted scabies is a fulminant highly infectious form of scabies with huge number of mites infesting the epidermis [6]. It was first described in 1848 by Danielsen and Boek who considered the disease a form of leprosy endemic to Norway. In 1851, the aetiology was related to the scabies mite and the disease was named “Norwegian scabies”. Later, the term crusted scabies was suggested and became more preferable [7].

Crusted scabies results from failure of the immune system to combat the mite allowing uncontrolled reproduction [8]. Immune-compromised patients and those with immunodeficiency disorders are at increased risk due to impaired cell mediated immune response [9].

Another explanation for crusted scabies is the lack of sensation and normal scratch response which normally helps the removal of mites and destroying the burrows. This explains the increased incidence in the mentally retarded patients [10].

Crusted scabies usually do not present as an acute eruption as in classical scabies. The eruption is slow in onset and insidious in progression. It usually presents as warty dermatosis of the hands and feet. The nails are dystrophic with abundant psoriasis like subungual hyperkeratosis [11]. Burrows seen in the flexures in classic scabies are less apparent. Rarely crusted scabies may also present as psoriasiform dermatitis that gradually progress to involve the entire body ending in erythroderma [12].

In clinically suspected cases, the differential diagnosis between erythrodermic psoriasis and crusted scabies is not easy. In cases of crusted scabies, skin biopsies are misleading as they are only diagnostic if the section contains mites. Meanwhile the pathology of erythrodermic psoriasis lesions is not diagnostic in many cases. Biopsy may show an absent stratum corneum with more prominent dilatation of superficial dermal blood vessels, in addition to the histological features of early psoriatic lesions which include mild epidermal hyperplasia with few neutrophils and red cell extravasations in the papillary dermis which is not conclusive. Parakeratosis and Monro micro-abscesses are usually not preserved, and there may be more spongiosis than is typically seen in classic cases which add to the difficulty of diagnosis [13-15].

When the specimen from the crusted lesion is scraped with a blunt scalpel and placed on a glass slide then a drop of mineral oil and a cover slip are placed on it, microscopic examination reveals numerous mites, eggs and mite faeces (scybala) [16,17].

It is worthy to note that in our case, although we started with the histopathological examination, we were lucky enough to find the
mites. The diagnosis was confirmed by the KOH scrapings which is the easiest office technique.

Although we highlighted in a previous publication the importance of scaly scalp scraping as an easy diagnostic method for crusted scabies, we missed this tip in this case, as we were misled by the provisional diagnosis of psoriatic erythroderma.

Conclusion

Clinical presentation with erythroderma, scaly scalp with or without nail dystrophy should entitle scalp scraping. Although in common practice, scalp scraping is done only as a diagnostic method for suspected cases of tinea capitis, we emphasize the importance of this simple technique as an accurate and rapid method of diagnosis of crusted scabies, avoiding wasting unnecessary time and effort of the patient and the physician.

References