

Unrecognized Dermatophyte Infection in Ichthyosis Vulgaris

Maja Grahovac, Dragomir Budimčić

University Department of Dermatology and Venereology, Zagreb University Hospital Center and School of Medicine, Zagreb, Croatia

Corresponding author:

Maja Grahovac, MD
University Department of Dermatology and Venereology
Zagreb University Hospital Center and School of Medicine
Šalata 4
HR-10000 Zagreb
Croatia
m.grahovac@yahoo.com

SUMMARY A case of unrecognized widespread dermatophyte infection associated with ichthyosis vulgaris and atopy is described. Our patient was a young woman in which the diagnosis of ichthyosis vulgaris and atopic dermatitis blocked the recognition of widespread dermatophyte infection for more than six months. The case showed some clinical peculiarities in terms of both extent of lesions and their clinical appearance.

KEY WORDS: chronic dermatophytosis, *Trichophyton mentagrophytes*, ichthyosis vulgaris, atopy

Received: September 16, 2008

Accepted: May 13, 2009

INTRODUCTION

Ichthyoses are a large heterogeneous group of disorders, typically involving most of the body surface and usually presenting at birth or shortly thereafter. Ichthyosis vulgaris (IV) is the most common disorder of keratinization with diffuse scaling and highly variable degree of involvement. The predilection sites are extensor surfaces of the extremities and the trunk. About 25%-33% of patients have atopic dermatitis (AD). When an IV patient has severe pruritus or flexural involvement, the answer is usually AD (1).

Several host conditions such as atopy and epidermal barrier defects like ichthyosis are associated with an increased prevalence of dermatophytosis (2). One of the most prevalent causes

of dermatophytosis is *Trichophyton* infection with ubiquitous occurrence because of its ability to escape the host's immune response.

We present a case of a young woman in which the diagnosis of ichthyosis vulgaris and atopic dermatitis precluded recognition of a widespread dermatophyte infection for more than six months.

CASE REPORT

A 26-year-old female patient with ichthyosis vulgaris and atopic dermatitis from early childhood presented to our hospital with itching rash. The first skin lesions had appeared six months earlier. All aspects of her erythema and scaling were assumed to be caused by atopic dermatitis. In this

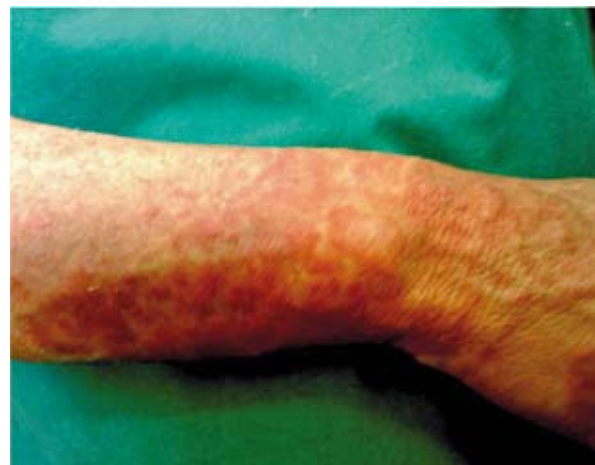


Figure 1. Initial visit; patients trunk and arm before therapy.

period, she had been treated with several corticosteroid creams (betamethasone, alclometasone), which made her lesions even worse. When the patient presented to our Department, dermatological examination revealed, besides dry skin, multiple nummular erythematous plaques on both arms, upper back and left lower leg. The lesions were markedly infiltrated, exalted and showed peripheral scaling (Fig. 1). Direct microscopic examination of scales obtained by scraping the plaques displayed septate hyphae. Finally, *Trichophyton (T.) mentagrophytes* was confirmed by culture. The source of dermatophyte infection was not confirmed. She was treated with antimycotic systemic treatment, terbinafine 250 mg/day and locally with clotrimazole twice a day for one month. After one month, the skin lesions completely resolved and mycologic examinations were negative (Figs. 2 and 3).

DISCUSSION

Defective epidermal differentiation and cornification are observed in various skin disorders, predominantly ichthyoses, a group of skin diseases characterized by generalized scaling of the skin (3). Dermatophytes are keratinophilic fungi able to infect keratinized tissues of human or animal origin, leading to infections that are mainly restrict-



ed to the corneocytes of the skin, hair and nails. These filamentous fungi are usually identified on the basis of clinical features, direct microscopic examinations, and by culture (4). The majority of dermatophyte infections in humans and animals are caused by *Trichophyton rubrum*, followed by *T. mentagrophytes* (5,6). Infections are usually localized and display characteristic clinical features. Besides pathogen-associated virulence factors, several host conditions are associated with an increased prevalence of dermatophytosis (2). In several studies of chronic dermatophytosis, the prevalence of genetic ichthyosis in the form of IV and atopy was observed (2,7-9). In an Indian study (10) of chronic dermatophytosis, IV was observed in 25% of patients. The most common systemic association was atopy in 7.3% of cases. The association between atopy and chronic infection by dermatophytes is well documented down to the molecular level (11). It has been proposed that immune abnormalities in the atopic skin (a shift from T-helper Th1 to Th2 response) might be responsible for the higher prevalence and severity of dermatophyte infections in such patients (5,11-13). However, in his study, Kaaman (1988) has concluded that atopy alone may not be the major contributory factor in chronicity (14). Atopy and barrier defects like those in ichthyoses are known risk factors for dermatophytosis (11). It is thought that due to excessive keratin production, ichthyotic skin provides a more favorable habitat for fungi than normal skin (5,10). It is also seen in other patients that an abnormal process of keratinization itself may increase the chance for dermatophyte infection. Several studies report an increased prevalence of dermatophytosis in patients with isolated disorders of keratinization such as palmar and plantar hyperkeratosis and KID (keratitis, ichthyosis, deafness) syndrome



Figure 2. Patients trunk and arm after two weeks of therapy.

(15-18). The amount of keratin is considered the most important factor for dermatophytosis affinity for palms and soles.

We present a case of a young woman in which the diagnosis of ichthyosis vulgaris and atopic dermatitis precluded recognition of widespread dermatophyte infection for more than six months. Similar experience has been described by Sheetz and Lynch, where fungal infection proceeded unrecognized for many years and all aspects of scaling were assumed to be caused by ichthyosis (19). Another two reports suggest that dermatophyte infection is rarely recognized in patients with congenital ichthyosis (20,21).

CONCLUSION

The dermatophyte infection presented in this report was initially misdiagnosed because of its



Figure 3. Patients arm after one month of therapy

atypical clinical aspect and widespread localization. The relationship of fungal infection and ichthyosis vulgaris, although worthy to note, is infrequently reported. Therefore, tinea should always be excluded, especially in patients suffering from ichthyosis and atopy widespread erythematous plaques.

References

1. Braun-Falco O, Plewig G, Wolff HH, Burgdorf WHC. Ichthyoses: Dermatology. Second, completely revised edition. Berlin:Springer;2000. pp. 713-22.
2. Hay RJ. Chronic dermatophyte infection. I Clinical and mycological features. Br J Dermatol 1982;106:1-7.
3. Hoffjan S, Stemmler S. On the role of the epidermal differentiation complex in ichthyosis vulgaris, atopic dermatitis and psoriasis. Br J Dermatol 2007;157:441-9.
4. Frealle E, Rodrigue M, Gantois N, Aliouat CM, Delaporte E, Camus D, *et al.* Phylogenetic analysis of *Trichophyton mentagrophytes* human and animal isolates based on MnSOD and ITS sequence comparison. Microbiology 2007;153:3466-77.
5. Hoetzenecker W, Schanz S, Schaller M, Fierlbeck G. Generalized tinea corporis due to *Trichophyton rubrum* in ichthyosis vulgaris. J Eur Acad Dermatol Venereol 2007;21:1129-31.
6. McGregor JM, Hamilton AJ, Hay RJ. Possible mechanisms of immune modulation in chronic dermatophytoses: an *in vitro* study. Br J Dermatol 1992;127:233-8.
7. Hay RJ, Brostoff J. Immune responses in patients with chronic *Trichophyton rubrum* infections. Clin Exp Dermatol 1977;2:373-80.

8. Hay RJ. Failure of treatment in chronic dermatophyte infections. *Postgrad Med J* 1979;55:608-10.
9. Jones HE, Reinhardt JH, Rinaldi MG. Immunologic susceptibility to chronic dermatophytosis. *Arch Dermatol* 1974;110:213-20.
10. Sentamilselvi G, Kamalam A, Ajithadas K, Janaki C, Thambiias AS. Scenario of chronic dermatophytosis: an Indian study. *Mycopathologia* 1997;140:129-35.
11. Ludwig RJ, Woodfolk JA, Grundmann-Kollm NN, Enzensberger R, Runne U, Platts-Mills TA, *et al.* Chronic dermatophytosis in lamellar ichthyosis: relevance of a T-helper 2-type immune response to *Trichophyton rubrum*. *Br J Dermatol* 2001;145:518-21.
12. Slunt JB, Taketomi EA, Woodfolk JA, Hayden ML, Platts-Mills TA. The immune response to *Trichophyton tonsurans*: distinct T cell cytokine profiles to a single protein among subjects with immediate and delayed hypersensitivity. *J Immunol* 1996;157:5192-7.
13. Decken K, Kohler G, Palmer-Lehmann K, Wunderlin A, Mattner F, Magram J, *et al.* Interleukin-12 is essential for protective Th1 response in mice infected with *Cryptococcus neoformans*. *Infect Immun* 1998;66:4994-5000.
14. Kaaman T. Hand, foot and nail disease – a common manifestation of chronic dermatophytosis. *Mycoses* 1988;31:613-16.
15. Hazen PG, Walker AE, Stewart JJ, Carney JF, Engstrom CW, Turgeon KL. Keratitis, ichthyosis and deafness (KID) syndrome; management with chronic oral ketoconazole therapy. *Int J Dermatol* 1992;31:58-9.
16. Ginter G, Soyer HP. Atypical erythrokeratoderma with deafness, keratitis and double mycotic infection. *Z Hautkr* 1988;63:951-7.
17. Chopra A, Maninder S, Gill SS. Hyperkeratosis of palms and soles: clinical study. *IJDVL* 1997;63:85-8.
18. Nielsen PG. Hereditary palmoplantar keratoderma and dermatophytosis in the northernmost county of Sweden (Norrbotten). *Acta Derm Venereol Suppl (Stockh)* 1994;188:1-60.
19. Sheetz K, Lynch PJ. Ichthyosis and dermatophyte fungal infection. *J Am Acad Dermatol* 1991;24(2 Pt 1):321.
20. Shelley ED, Shelley WB, Schafer RL. Generalized *Trichophyton rubrum* infection in congenital ichthyosiform erythroderma. *J Am Acad Dermatol* 1989;20:1133-4.
21. Agostini G, Geti V, Difofo EM, Gianotti B. Dermatophyte infection in ichthyosis vulgaris. *Mycoses* 1992;35:197-9.