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Case Report Open Access

Methicillin-Resistant Staphylococcus Aureus Toe Web Infection in a Patient with Atopic Dermatitis; Association with Disease Activity

Aslı Bilgiç[1], **Özge Turhan**[2] **and Özlem Dicle**[1*]

[1]*Department of Dermatology and Venereology, School of Medicine, Akdeniz University, Antalya, Turkey*

[2]*Associate Professor, Department of Infectious Diseases and Clinical Microbiology, School of Medicine, Akdeniz University, Antalya, Turkey*

***Corresponding author:** Özlem Dicle, Department of Dermatology and Venereology, School of Medicine, Akdeniz University, 07070, Antalya, Turkey, Tel: 90 242 2496707; Fax: 90 242 2274490; E-mail: odicle@akdeniz.edu.tr

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Case Report

An otherwise healthy 64 year-old man with 5 years history of atopic dermatitis (AD) presented with itching, excoriated erythematous scaly plaques involving the popliteal fossae, antecubital fossae, periorbital area, the neck and the dorsal aspect of his hands and feet, elbows, and knees with maceration of the toe-webs (Figure 1). The patient received intensive inpatient treatment prior to presentation to our clinic; he had been treated with high potency topical steroids, topical pimecrolimus, emollients and oral antihistamines for long periods and several cycles of oral corticosteroids with some improvement.

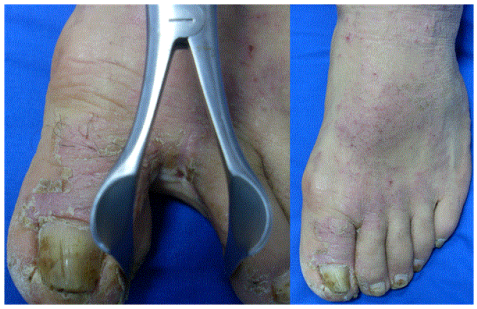

Figure 1: Maceration of the toe web and erythematous scaly plaques on the dorsal surface of the foot.

A histopathologic examination of the biopsy taken from left antecubital fossae, supported the diagnosis of atopic dermatitis. Laboratory examination revealed mild blood eosinophilia [6, 9%, (N: 0.90-2.90)], and high IgE level [1076 IU/mL (N: 0.00-87.00)]. Complete blood analysis, routine biochemical tests and blood glucose levels showed normal values. Potassium hydroxide test and Wood's light examination for the evaluation of interdigital maceration with respect to frequent infectious condition such as tinea pedis and erythrasma gave negative results. However, a swab for bacteriologic pathogens was found positive for Methicillin-resistant *Staphylococcus aureus (MRSA).* The patient was treated with oral fusidic acid at a dose of 500 mg 3 times daily according to the antimicrobial resistance pattern of the pathogen and local drug availability. His symptoms of atopic dermatitis improved after this therapy combined with desloratidine treatment twice a day, for ten days (Figure 2).

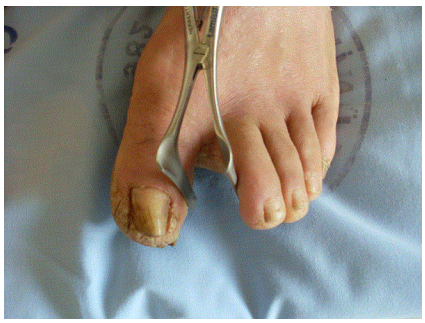

Figure 2: After treatment with oral fusidic acid

Atopic dermatitis is an inflammatory skin disease which has usually chronic course with relapses. Although the pathogenesis is not fully understood, the disease is mediated by disrupted skin barrier and immune defects involving skin integrity, protein abnormality and low levels of antimicrobial peptides in epithelium [1]. Therefore, atopic dermatitis can be aggravated by environmental factors such as allergens, stress and infections.

In patients with atopic dermatitis, skin barrier defects promotes skin colonization by microbes, such as *Staphylococcus aureus* and lead to further inflammation and persistence of the T-helper 2-dependent immune response. Moreover, cell wall components of *Staphylococcus aureus* may trigger the production of chemokines leading to increased infiltration of inflammatory cells [2]. It is well known that patients with atopic dermatitis experience high rates of nasal and flexural colonization of their skin surfaces by *Staphylococcus aureus*, but limited data is available regarding the colonization by MRSA. However atopic dermatitis patients require more frequent use of topical and systemic antibiotics, which can lead to the development of greater microbial resistance in this population and the resulting colonization by resistance microorganisms, such as MRSA [3]. In the last 10 years, some studies and cases on MRSA infections in pediatric atopic dermatitis patients reported and some of them showed association with the presence of MRSA and increased severity of atopic dermatitis [4-7]. On the other hand, although foot intertrigo are known to be mostly caused initially by dermatophytes, *Staphylococcus aureus* is not a rare condition. In the study by Karaca et al., the most common pathogen of foot intertrigo was found to be coagulase-negative staphylococci [8]. To the best of our knowledge, the presented case is the first case of toe-web infection caused by MRSA in a patient with atopic dermatitis.

Oral agents recommended for treatment of MRSA infections include clindamycin, doxycycline, co-trimoxazole, rifampicin and fusidic acid and all have variable in vitro activity against MRSA isolates. Fusidic acid which has been in clinical use for the treatment of staphylococcal infections including MRSA, in countries where it is available, in comparison, has a favorable adverse effect profile. MRSA is also emerging in Turkish hospitals, in the one of the largest epidemiological study done in Turkey susceptibility rates for fusidic acid were found to be very high (91.9 %) based on antibiotic sensitivity tests [9]. Therefore, since neither serious nor complicated skin infections nor identifiable risk factors were present in our patient, oral fusidic acid treatment was preferred and efficient response was rapidly achieved.

In all symptomatic toe web infections, the presence of gram-negative and positive bacteria should be investigated and the presence of this type of skin infection in patients with atopic dermatitis, particularly those unresponsive to conventional therapy should alert the clinician as the underlying aetiology, and intervention should be directed accordingly.

References

1. Irvine AD, McLean WH, Leung DY (2011) Filaggrin mutations associated with skin and allergic diseases. N Engl J Med 365: 1315-1327.
2. Jinnestal CL, Belfrage E, Bäck O, Schmidtchen A, Sonesson A (2014) Skin barrier impairment correlates with cutaneus Staphylococcus aureus colonization and sensitization to skin-associated microbial antigens in adults patients with atopic dermatitis. Int J Dermatol 53: 27-33.
3. Lipnharski C, d'Azevedo PA, Quinto VP, Bessa G, Bonamigo RR (2013) Colonization by S. aureus increases the EASI and the number of appointments by patients with atopic dermatitis: cohort with 93 patients. An Bras Dermatol 88: 518-521.
4. Hon KL, Leung AK, Kong AY, Leung TF, Ip M (2008) Atopic dermatitis complicated by methicillin-resistant Staphylococcus aureus infection. J Natl Med Assoc 100: 797-800.
5. Jagadeesan S, Kurien G, Divakaran MV, Sadanandan SM, Sobhanakumari K, et al. (2014) Methicillin-resistant Staphylococcus aureus colonization and disease severity in atopic dermatitis: A cross-sectional study from South India. Indian J Dermatol Venereol Leprol 80: 229-234.
6. Suh LM, Honig PJ, Yan AC (2006) Methicillin-resistant Staphylococcus aureus skin abscesses in a pediatric patient with atopic dermatitis: a case report. Cutis 78: 113-116.
7. Tang CS, Wang CC, Huang CF, Chen SJ, Tseng MH, et al. (2011) Antimicrobial susceptibility of Staphylococcus aureus in children with atopic dermatitis. Pediatr Int 53: 363-367.
8. Karaca S, Kulac M, Cetinkaya Z, Demirel R (2008) Etiology of foot intertrigo in the District of Afyonkarahisar, Turkey: a bacteriologic and mycologic study. J Am Podiatr Med Assoc 98: 42-44.
9. Yildiz O, Çoban AY, Sener AG, Coskuner SA, Bayramoglu G, et al. (2014) Antimicrobial susceptibility and resistance mechanisms of methicillin resistant Staphylococcus aureus isolated from 12 Hospitals in Turkey. Annals of clinical Microbiology and Antimicrobials 13: 44.