**INTRODUCTION**

Atopic dermatitis (AD) pathophysiology is complex and results from skin barrier dysfunction and an unregulated immune response, and this condition is influenced by genetic and environmental factors. Patients with AD are also at increased risk of bacterial, fungal, and viral skin infections which can lead to invasive infections if left untreated. Furthermore, their greater predisposition to developing skin infections would seem to be related to various contributing factors including skin barrier defects, a decrease in antimicrobial peptides (AMPs), an increased skin pH, or Th2 cytokines (such as IL-4 and IL-13).

**CASE REPORT**

A 9-year-old Caucasian boy suffering from chronic AD since the age of 1 year, who had spent a lot of time at a horse riding center, visited the clinic in January 2019. He reported worsening pruritus and scabs on his face, trunk, and extremities along with a diffused skin dryness (Figures 1 and 2) and a very serious itchy plaque on his lower left limb (Figure 3). As no laboratory test is needed to identify atopic dermatitis, the diagnosis was made by examining the skin and reviewing the boy’s medical history. The patient had already undergone skin prick tests for respiratory or food allergies, and patch tests for contact dermatitis, all of which resulted negative. In addition, the serum level of total immunoglobulin E was measured and was found to be within the normal range. He was treated for a long time with both topical steroids (including hydrocortisone butyrate 0.1%, mometasone furoate 0.1%) and inhibitors of calcineurin (tacrolimus and pimecrolimus) with minimal benefit. Afterward, he was treated with deflazacort oral solution at a dosage of 1 mg/kg/day for 20 days in several cycles. Despite the treatment, his disease often flared up again.

When he came for his first checkup, the patient had visible Dennie-Morgan lines, inflamed, erythematous papules, and crusty plaques scattered over 75% of his face and body, resulting in an eczema area and severity index (EASI) score of 19.
He also had a reddish plaque on his left leg, which was resistant to both steroidal and antimicrobial topical treatments. We therefore decided to perform a culture test of a skin scraped specimen which revealed an *Enterococcus faecalis* (*E. faecalis*) on the lesion. An antibiogram test allowed us to choose the most effective antibiotic drug. This subsequently led to a rapid improvement of the infected skin lesion. The patient had spent a substantial period of time at the horse riding center and had therefore came into frequent contact with horses and their excrement. Wolny-Koładka (2018) and Graves et al.(2008) identify some species *Staphylococcus* and *Enterococcus* spp. from the ground of a horse riding center. This all confirmed the high probability of contracting Staphylococcal and Enterococcal when exposed to soil contaminated with horse feces, as in the case of our patient.4,5

According to recent insight on physiopathology, it is well known that AD is associated with decreased production of AMPs in the skin, which are a group of molecules which act as protection against bacteria, fungi, and viruses.6,7 The reduction might be due to the suppressive effect of Th2 cytokines (present in higher quantities in AD) and a relative decrease in IL-17 (an inducer of AMPs).3,8

Among skin infections, the most common bacterial skin infections in AD are caused by *Staphylococcus aureus* (*S aureus*) followed by *Streptococcus pyogenes* whose virulence is due to staphylococcal enterotoxins (superantigens). More than 80% of *S aureus* isolated from AD patients are superantigen-producing.9

Patients with AD are at increased risk of colonization by methicillin-resistant *S. aureus* (MRSA), compared with the general population.10 MRSA skin and soft-tissue infections lead to a loop of AD.11

Like bacterial infections, AD patients are at a higher risk of eczema herpeticum (EH) caused by the herpes simplex virus and fungal infection, such as tinea or yeast.12
3 | CONCLUSION

Skin barrier defects, a decrease in AMPs, increased skin pH, or Th2 cytokines are potential contributing factors to the amplified risk of skin infections in AD, especially those caused by *S. aureus*. Conversely, our patient developed a skin infection caused by *E. faecalis*, which is a bacterium typically present in the gut and bowel. The combination of environmental factors (such as frequent contact with animal excrement) and a well-documented higher susceptibility of AD patients to skin infections go some way to explaining this unusual skin condition.

CONFLICT OF INTEREST

Authors have no conflict of interest.

AUTHOR CONTRIBUTION

MG: conceived the article and wrote the paper. TC, BA, and CM: critically revised the manuscript. DP: gave important intellectual contribution to the final version of the article.

CONSENT STATEMENT

A written informed consent was obtained from the patient.

ORCID

Giovanna Malara https://orcid.org/0000-0001-6210-4479

Pietro Denisi https://orcid.org/0000-0003-4714-4717

REFERENCES

1. Guttmann-Yassky E, Nograles KE, Krueger JG. Contrasting pathogenesis of atopic dermatitis and psoriasis Part II: immune cell subsets and therapeutic concepts. *J Allergy Clin Immunol*. 2011;127:1420-1432.
2. Langan SM, Abuabara K, Henrickson SE, Hoffstad O, Margolis DJ. Increased risk of cutaneous and systemic infections in atopic dermatitis—a cohort study. *J Invest Dermatol*. 2017;137(6):1375.
3. Ong PY, Leung DY. Bacterial and viral infections in atopic dermatitis: a comprehensive review. *Clin Rev Allergy Immunol*. 2016;51(3):329-337.
4. Wolny-Kolarzka K. Resistance to antibiotics and the occurrence of genes responsible for the development of methicillin resistance in *Staphylococcus* bacteria isolated from the environment of horse riding centers. *J Equine Vet Sci*. 2018;61:65-71.
5. Graves A, Weaver RW, Entry J. Characterization of enterococci populations in livestock manure using BIOLOG. *Microbiol Res*. 2009;164(3):260-266.
6. Gallo RL, Murakami M, Ohtake T, Zaiou M. Biology and clinical relevance of naturally occurring antimicrobial peptides. *J Allergy Clin Immunol*. 2002;110:823-831.
7. Leung DYM. Superantigens, steroid insensitivity and innate immunity in atopic eczema. *Acta Derm Venereol Suppl (Stockh)*. 2005;15:11-15.
8. Wolk K, Mitsui H, Witte K, et al. Deficient cutaneous antibacterial competence in cutaneous T-cell lymphomas: role of Th2-mediated biased Th17 function. *Clin Cancer Res*. 2014;20:5507-5516.
9. Leung DY, Hanifin JM, Pariser DM, et al. Effects of pimecrolimus cream 1% in the treatment of patients with atopic dermatitis who demonstrate a clinical insensitivity to topical corticosteroids: a randomized, multicentre vehicle-controlled trial. *Br J Dermatol*. 2009;161(2):435-443.
10. Avena-Woods C. Overview of atopic dermatitis. Am J Manag Care. 2017;23:S115-S123.
11. Ong PY. Recurrent MRSA infections in atopic dermatitis. *J Allergy Clin Immunol Pract*. 2014;2:396-399.
12. Siegfried EC, Hebert AA. Diagnosis of atopic dermatitis: mimics, overlaps, and complications. *J Clin Med*. 2015;4(5):884-917.