

A Case of *Staphylococcus simulans* Colonization in a Patient with Psoriasis

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A 20-year-old female patient presented with localized erythematous scaly lichenified plaques on her right lower leg. Despite traditional treatments, the lesions consistently recurred in previously affected anatomical locations. Bacterial culture was performed, and *Staphylococcus simulans* was identified on both lesional and nonlesional skin. Histopathological examination revealed parakeratosis, Munro microabscess, hypogranulosis and regular acanthosis with an elongated rete. Based on clinical and histological findings, the patient was finally diagnosed with possible psoriasis. Although *S. simulans* is a common livestock colonizer, it rarely colonizes human skin. Herein, we report the first case of *S. simulans* colonization in a recurrent psoriatic lesion in previously affected anatomical locations.

Key Words: Psoriasis, *Staphylococcus simulans*

INTRODUCTION

Psoriasis is an immune-mediated chronic inflammatory disease characterized by skin symptoms, articular symptoms, and other systemic symptoms. Although the pathogenesis of psoriasis has not been fully elucidated, it is a multifactorial disease driven by both genetic predisposition and exposure to diverse environmental factors. Triggering factors include biomechanical stress, diet, drugs, alcohol, smoking, and infection¹. *Staphylococcus simulans* (*S. simulans*) is a common livestock colonizer and a rare human colonizer. In this report, we present the case of *S. simulans* colonization in a recurring psoriatic lesion in anatomical areas previously affected.

CASE REPORT

A 20-year-old female patient presented with localized erythematous scaly lichenified plaques on the right lower leg (Fig. 1A). These lesions first appeared nine months ago and spread as she scratched herself constantly because of severe pruritus. She had no relevant personal or family history, and she also had no history of close contact with animals. The potassium hydroxide (KOH) smear was negative. The lesional and nonlesional skin of the right lower leg was rubbed 20 times (10 times in one direction and 10 times perpendicular to this direction) with a cotton stick for bacterial culture. For molecular identification of bacteria, a single colony was inoculated in tryptic soy broth (TSB; 1.7% casein peptone, 0.3% soybean peptone, 0.25% glucose, 0.5% sodium chloride, 0.25% dipotassium phosphate, and 1.5% agar) and cultured

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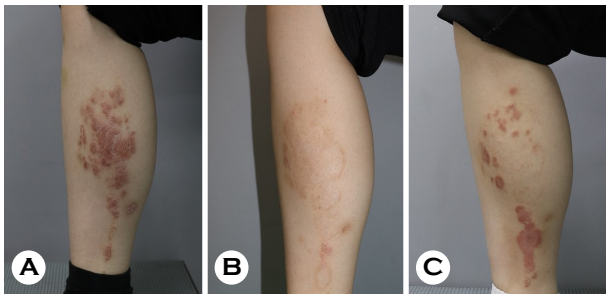


Fig. 1. (A) At the initial visit, multiple erythematous scaly lichenified plaques were identified on the right lower leg. (B) Two months after the initial visit, the lesions markedly improved with postinflammatory hyperpigmentation. (C) Eight months after the initial visit, multiple erythematous scaly plaques recurred on the right lower leg, and a punch biopsy was performed.

at 37°C². Genomic DNA was extracted following the method described in a previous study³. The 16S rDNA region was identified using the universal primers 27F (5'- GAGTTGATCMTGGCTCAG -3') and 1492R (5'- TACGGYTACCTTGTTACGACTT -3')⁴. The PCR product was sequenced for species identification. The resulting sequence was blasted against the nucleotide sequence database of the National Center for Biotechnology Information. The susceptibility tests were performed using the Muller-Hinton broth (MHB) medium and minimal inhibitory concentrations (MICs) were determined per the Clinical and Laboratory Standards Institute guidelines⁵. *S. simulans* was identified on lesional and nonlesional skin. MICs of vancomycin for both isolated strains were 2 µg/ml. MICs of methicillin for both strains were found to be in the 2~4 µg/ml range, indicating that the isolate showed slightly decreased susceptibility to vancomycin and methicillin.

With a clinical impression of lichen simplex chronicus secondary to severe pruritus, we prescribed oral cyclosporine (200 mg/day) and oral antihistamine. After two months of cyclosporine treatment, the lesions improved significantly, leaving postinflammatory hyperpigmentation (Fig. 1B). However, erythematous scaly plaques repeatedly recurred on the same area (Fig. 1C), and punch biopsy was performed. Histopathological evaluation revealed parakeratosis, Munro microabscess, hypogranulosis, and regular acanthosis with an elongated rete (Figs. 2A and 2B). Mild perivascular lymphocytic infiltration was observed in the dermis. Periodic acid-Schiff (PAS) staining was negative. Per our clinical and histological findings, the patient was finally diagnosed with possible psoriasis.

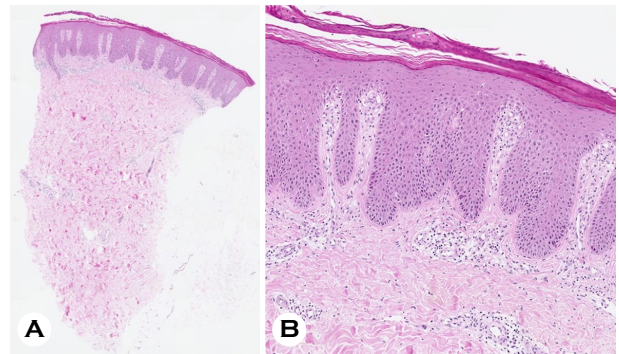


Fig. 2. (A-B) Histopathological examination showed parakeratosis, Munro microabscess, hypogranulosis, and regular acanthosis with an elongated rete present in the epidermis. Mild perivascular lymphocytic infiltration was observed in the papillary dermis (H&E, ×20; H&E, ×200).

DISCUSSION

It has been suggested that infection may play a role in triggering and maintaining psoriasis. In particular, the association between streptococcal throat infections and guttate psoriasis is well-established. Additionally, a possible link has been suggested between exacerbations of existing plaque psoriasis and streptococcal infections⁶. Streptococcal infections can act as both inducing and aggravating factors of psoriasis, possibly by stimulating T-cell proliferation through superantigens and promoting the migration of skin-homing T cells via molecular mimicry between streptococcal and skin-specific epitopes^{7,8}. Among *Staphylococcus* species, *Staphylococcus aureus* (*S. aureus*) infection have been found to be implicated in the disease activity of psoriasis. Its superantigens (particularly staphylococcal enterotoxin-A) play a role in the pathogenesis of psoriasis by stimulating T-cell signaling and interferon-gamma production⁹. In addition, bacteria such as *Helicobacter pylori*, *Escherichia coli*, and *Pseudomonas aeruginosa*, viruses such as the human immunodeficiency virus and hepatitis virus, and fungi such as *Malassezia* and *Candida* may play a role in the pathogenesis and exacerbation of psoriasis¹⁰. Further research is needed to determine their exact roles and the mechanisms involved.

To date, there have been no reported associations between *S. simulans* and psoriasis. *S. simulans* is a coagulase-negative *Staphylococcus* that is usually identified in animals such as cattle, sheep, and goats¹¹. However, unlike its common occurrence in livestock, it rarely colonizes human skin, and there have been only a few cases in which it has acted as a human pathogen causing skin infections^{11,12}, toxic shock syndrome¹³,

osteoarticular infections^{14,15}, native valve endocarditis¹⁶, urinary tract infections^{17,18}, and pleural empyema¹⁹. On the other hand, *S. simulans* may function as commensal bacteria, contributing to the maintenance of skin homeostasis rather than acting as a pathogen by producing auto-inducing peptides that block colonization and acute infection by methicillin-resistant *S. aureus*²⁰. In our case, the use of immunosuppressants such as cyclosporine, along with repeated hand irritation on the lesion, may have affected the skin microbiome or rendered the lesion vulnerable to secondary infection by *S. simulans*. However, because *S. simulans* colonization was observed in both lesional and nonlesional skin, it is unlikely to be solely attributable to secondary infection from the external environment. Herein, to the best of our knowledge, we report the first case of *S. simulans*, a rare human skin colonizer and common livestock colonizer, in a recurrent psoriatic lesion in previously affected anatomical locations. Whether or not the colonization of *S. simulans* has pathophysiological significance in the pathogenesis and recurrence of psoriasis in this patient remains undetermined. Further research is needed to elucidate the underlying mechanisms and the significance of the results.

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CONFLICT OF INTEREST

In relation to this article, we declare that there is no conflict of interest.

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PATIENT CONSENT STATEMENT

The patient provided written informed consent for the publication and the use of her images.

REFERENCES

1. Yan D, Gudjonsson JE, Le S, Maverakis E, Plazyo O, Ritchlin C, et al. New frontiers in psoriatic disease research, Part I: Genetics, environmental triggers, immunology, pathophysiology, and precision medicine. *J Invest Dermatol* 2021;141:2112-2122.e3
2. Doyle JE, Mehrhof WH, Ernst RR. Limitations of thio-glycolate broth as a sterility test medium for materials exposed to gaseous ethylene oxide. *Appl Microbiol* 1968; 16:1742-1744
3. Chapaval L, Moon DH, Gomes JE, Duarte FR, Tsai SM. An alternative method for *Staphylococcus aureus* DNA isolation. *Arq Bras Med Vet Zootec* 2008;60:299-306
4. Suzuki MT, Giovannoni SJ. Bias caused by template annealing in the amplification of mixtures of 16S rRNA genes by PCR. *Appl Environ Microbiol* 1996;62:625-630
5. Clinical and Laboratory Standards Institute. Methods for dilution antimicrobial susceptibility tests for bacteria that grow aerobically, 10th ed.; approved standard M07-A10.; Wayne, PA, USA, 2015
6. Gudjonsson JE, Thorarinnsson AM, Sigurgeirsson B, Kristinnsson KG, Valdimarsson H. Streptococcal throat infections and exacerbation of chronic plaque psoriasis: a prospective study. *Br J Dermatol* 2003;149:530-534
7. Valdimarsson H, Thorleifsdottir RH, Sigurdardottir SL, Gudjonsson JE, Johnston A. Psoriasis--as an autoimmune disease caused by molecular mimicry. *Trends Immunol* 2009;30:494-501
8. Sigurdardottir SL, Thorleifsdottir RH, Valdimarsson H, Johnston A. The association of sore throat and psoriasis might be explained by histologically distinctive tonsils and increased expression of skin-homing molecules by tonsil T cells. *Clin Exp Immunol* 2013;174:139-151
9. Nielsen MB, Odum N, Gerwien J, Svejgaard A, Bendtzen K, Bregentholt S, et al. Staphylococcal enterotoxin-A directly stimulates signal transduction and interferon-gamma production in psoriatic T-cell lines. *Tissue Antigens* 1998;52:530-538
10. Zhou S, Yao Z. Roles of Infection in Psoriasis. *Int J Mol Sci* 2022;23:6955
11. Shields BE, Tschetter AJ, Wanat KA. *Staphylococcus simulans*: An emerging cutaneous pathogen. *JAAD Case Rep* 2016;2:428-429
12. Tous Romero F, Gutiérrez García-Rodrigo C, Velasco Tamariz V, Llamas Martín R. Acute infection by *Staphylococcus simulans* in the hand of a man. *JAMA Dermatol* 2016;152:1060
13. Goda K, Kenzaka T, Hoshijima M, Yachie A, Akita H.

- Toxic shock syndrome with a cytokine storm caused by *Staphylococcus simulans*: a case report. BMC Infect Dis 2021;21:19
14. Désidéri-Vaillant C, Nédelec Y, Guichon JM, Le Louarn S, Noyer V, Sapin-Lory J, et al. *Staphylococcus simulans* osteitis in a diabetic patient. Diabetes Metab 2011;37: 560-562
 15. Mallet M, Loiez C, Melliez H, Yazdanpanah Y, Senneville E, Lemaire X. *Staphylococcus simulans* as an authentic pathogenic agent of osteoarticular infections. Infection 2011;39:473-476
 16. Vallianou N, Evangelopoulos A, Makri P, Zacharias G, Stefanitsi P, Karachalios A, et al. Vertebral osteomyelitis and native valve endocarditis due to *Staphylococcus simulans*: a case report. J Med Case Rep 2008;2:183
 17. Drobeniuc A, Traenkner J, Rebolledo PA, Ghazaryan V, Roupheal N. *Staphylococcus simulans*: A rare uropathogen. IDCases 2021;25:e01202
 18. Shishido M, Kitaoka H, Watanabe K, Fujimoto M, Kumagai T. Acute focal bacterial nephritis caused by *Staphylococcus simulans*. Cureus 2022;14:e31241
 19. Lal A, Akhtar J, Ullah A, Abraham GM. First case of pleural empyema caused by *Staphylococcus simulans*: Review of the literature. Case Rep Infect Dis 2018;2018: 7831284
 20. Brown MM, Kwiecinski JM, Cruz LM, Shahbandi A, Todd DA, Cech NB, et al. Novel peptide from commensal *Staphylococcus simulans* blocks methicillin-resistant *Staphylococcus aureus* quorum sensing and protects host skin from damage. Antimicrob Agents Chemother 2020;64:e00172-20