Journal of Pakistan Association of Dermatologists. 2026;**36(1)**:128-131.

Case Report

Guttate psoriasis induced by *Staphylococcus aureus* infection

Cut Putri Hazlianda[1], Adina Miltania Tasmil[1]

[1]Dermatology and Venereology Department, Faculty of Medicine Universitas Sumatera Utara, Universitas Sumatera Utara Hospital, Medan, Indonesia.

Abstract

Guttate psoriasis (GP) is an acute form of psoriasis commonly triggered by streptococcal infection, particularly in the pharyngeal or perianal regions. Its etiology involves both environmental and genetic factors that contribute to a complex immune response in the skin. Genetic predisposition, environmental factors, and immune dysfunction all contribute to the pathogenesis of psoriasis with host-microbe interaction governing the progression of this disease. Emerging evidence has indicated that infection is an environmental trigger for psoriasis and plays multiple roles in its maintenance as evidenced by the frequent association between guttate psoriasis onset and acute staphylococcal infection. Different infectious factors act on immune cells to produce inflammatory cytokines that can induce or aggravate psoriasis. We reported a 57-year-old female patient presented with erythematous plaques. The throat swab examination revealed aerobic *Staphylococcus aureus* growth.

Keywords Guttate psoriasis; *Staphylococcus aureus*; Throat swab.

Article Received on 21.12.2024

Revised on 22.10.2025 14.01.2026 17.03.2026

Accepted on 19.03.2026

Published on 30.03.2026

Citation: Hazlianda CP, Tasmil AM. Guttate psoriasis induced by *Staphylococcus aureus* infection. *J Pak Assoc Dermatol*. 2026;**36(1)**:128-131. **Doi- https://doi.org/10.66344/jpad.v36i1.3095**

Introduction

Guttate psoriasis (GP), derived from Latin word gutta meaning “a drop,” is characterized by the sudden appearance of numerous small, drop-shaped papules measuring 0.5–1.5 cm in diameter, predominantly distributed over the upper trunk and proximal extremities.[1,2] This form of psoriasis is most commonly associated with the human leukocyte antigen (HLA)- Cw6 and a preceding streptococcal throat infection. The diagnosis of GP is primarily clinical, based on the presence of characteristic erythematous, scaly, drop like lesions. A recent history of upper respiratory tract infection, particularly due to Streptococcus pyogenes, is often identified as a precipitating factor.[2,3] Throat culture or serologic testing may be performed to confirm streptococcal infection when clinically indicated. Skin biopsy is seldom required but may assist in excluding other papulosquamous disorders. Topical corticosteroids remain the first line therapy for mild cases, whereas moderate to severe disease is managed with phototherapy, either as monotherapy or in combination with topical agents.[3,4] Early identification and treatment of infectious triggers are essential to prevent recurrence and chronicity.[5,6]

Address for correspondence
Dr. Cut Putri Hazlianda
Department of Dermatology and Venereology, Faculty of Medicine Universitas Sumatera Utara, Universitas Sumatera Utara Hospital, Medan, Indonesia.
Email: cut.putri@usu.ac.id

Case Report

A 57-year-old woman presented to our dermatology outpatient clinic with multiple raised reddish plaques on the scalp, trunk, back, hands, and feet for one month. The lesions ranged in size from that of a corn kernel to a coin and were covered with white, scaly surfaces accompanied by itching. The patient reported that the lesions had reappeared during the past month and that she had recently experienced significant psychological stress due to family problems.

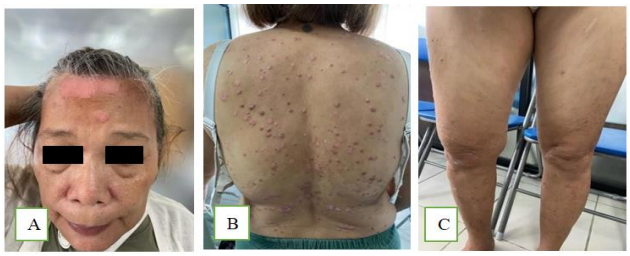

Figure 1 (A-C) Multiple erythematous papules and circumscribed erythematous plaques of military-lenticular size, discrete, covered with white scales were found on the scalp, chest, back, arms and legs, and inferior extremities.

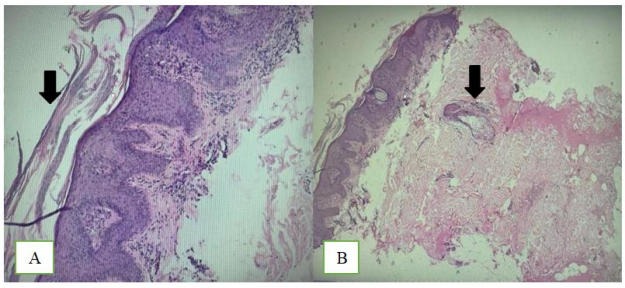

Figure 2 (A-B) Histopathological examination showed stratified squamous epithelium with normal nuclear morphology along with hyperkeratosis and suprapapillary thinning squamous. The basement membrane was intact. The subepidermal layer showed there were neutrophilic inflammatory cells infiltrates.

On dermatological examination, multiple erythematous papules and well circumscribed erythematous plaques of miliary to lenticular size were observed on the scalp, thoracic, vertebral, brachial (bilateral), and lower extremity regions. The lesions were discrete and covered with silvery white scales. The Auspitz sign was positive. The Psoriasis Area and Severity Index (PASI) score was 15.9, consistent with severe psoriasis, while the Dermatology Life Quality Index (DLQI) score was 6, indicating a moderate impact on quality of life. Laboratory evaluation at Prof. Dr. Chairuddin Panusunan Lubis Hospital included a complete blood count, liver and kidney function tests, throat swab culture, microbial sensitivity test, and histopathological examination. The hematologic and biochemical parameters were within normal limits. Throat swab culture revealed aerobic S. aureus growth. The sensitivity test indicated resistance to ampicillin, penicillin, and piperacillin.

Histopathological examination of a lesional skin biopsy showed stratified squamous epithelium with normal nuclear morphology. The epidermis exhibited regular hyperkeratosis with thinning of the suprapapillary plates. The basement membrane remained intact, and neutrophilic infiltrates were present within the suprabasal layer, findings consistent with psoriasis. The patient was treated with oral antihistamines; a high-potency topical corticosteroid combined with 3% salicylic acid applied to the body and extremities, 1% low-potency topical corticosteroid cream for facial lesions, topical urea, antifungal shampoo applied twice weekly for scalp lesions and multivitamins.

At fourth week follow up visit in November 2022, the patient reported improvement in redness and scaling, though mild itching persisted. Dermatological examination revealed reduced and thinned papules and circular erythematous plaques, discrete and covered with fine scales, distributed over the scalp, chest, back, and lower extremity areas. The PASI score had decreased to 7.5 (moderate psoriasis), and the DLQI score improved to 2 (mild effect), indicating partial remission and a significant improvement in the patient's quality of life.

Discussion

The patient described in this report presented with guttate psoriasis, which appeared to be triggered by S. aureus infection. Although guttate psoriasis is most commonly associated with streptococcal throat infection and the presence of HLA-Cw6, other bacterial infections, including S. aureus, may act as precipitating factors. Infectious agents can stimulate immune cells to release pro-inflammatory cytokines, thereby exacerbating psoriatic inflammation.[5,7] Several studies have demonstrated that staphylococcal superantigens contribute to development of psoriatic lesions. The pathophysiology of psoriasis vulgaris has been linked to bacterial superantigens such as staphylococcal enterotoxin A (SEA), which selectively binds to major histocompatibility complex (MHC) class II molecules. These molecules

are expressed at high concentrations on T cells within psoriatic skin lesions, promoting abnormal T-cell activation and cytokine release.[8]

The diagnosis of guttate psoriasis is primarily clinical, based on dermatological examination and patient history.[6,8] Supporting investigations, such as throat swab or bacteriological culture, may assist in identifying infectious triggers. In selected cases, complete blood count and histopathological examination may provide additional diagnostic value. Patients often report symptoms of upper respiratory tract infection such as cough, rhinorrhea, or sore throat preceding the onset of the eruption.[5,8]

Therapeutic options for guttate psoriasis include topical and systemic agents. First-line treatment involves topical corticosteroids, vitamin D analogues, and tar preparations, while systemic non-biologic therapies such as retinoids, cyclosporine, and methotrexate serve as second-line options.[7,8] The primary goal of treatment is to achieve complete lesion clearance and long-term remission. In many cases, guttate psoriasis resolves spontaneously within three to four months, particularly when associated with streptococcal infection, and the overall prognosis remains favorable.[9,10]

Conclusion

This case highlights guttate psoriasis potentially triggered by S. aureus infection, an uncommon but clinically significant finding. While streptococcal infection remains the most established precipitating factor, this report emphasizes the importance of considering other bacterial pathogens as possible triggers in susceptible individuals. Recognition of infectious sources and timely antimicrobial management, combined with appropriate dermatologic therapy, can lead to substantial clinical improvement and reduction in disease severity. Early identification and treatment of triggering infections are crucial to prevent recurrence and to achieve long term remission in patients with guttate psoriasis.

Declaration of patient consent Authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship None.

Conflict of interest No conflict of interest.

Author's contribution

CPH: Substantial contributions to conception and design, acquisition of data, analysis & interpretation of data. Drafting the article and revising it critically.

AMT: Substantial contributions to conception and design, acquisition of data, analysis & interpretation of data. Drafting the article and revising it critically.

All authors has given final approval of the version to be published and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

References

1. Goh C, Cheng C, Agak G, Zanglein AL, Graber EM, Thiboutot DM, Kim J. Acne vulgaris. In: Kang S, Amagai M, Bruckner AL, Enk AH, Margolis DJ, McMichael AJ, Orringer JS, editors. Fitzpatrick's Dermatology in General Medicine. 9th ed. New York: McGraw-Hill Companies; 2019. p.1391–1412.
2. Zhou S, Yao Z. Roles of infection in psoriasis. *Int J Mol Sci*. 2022;**23(12)**:6995. doi:10.3390/ijms23126995.
3. Zhou T, Koussiouris J, Kim L, Vender R. Management of guttate psoriasis: a systematic review. *J Cutan Med Surg*. 2024;**28(6)**:577–84. doi:10.1177/12034754241266187.
4. Prameswari FU, Prakoeswa CRS, Ferriastuti W. Narrowband ultraviolet B (NB-UVB) phototherapy in psoriasis: a retrospective study. *Berkala Ilmu Kesehatan Kulit dan Kelamin*. 2022;**34(2)**:99–108. doi:10.20473/bikk.v34i2.2022.99–108.
5. Fernandez-Figueras MT, Puig L. Histopathological diagnosis of psoriasis and psoriasiform dermatitides. *Diagn Histopathol*. 2025;**31(2)**:87–97. doi:10.1016/j.mpdhp.2024.11.005
6. Miao M, Yan J, Sun Y, Liu J, Guo S. Psoriasis: unraveling disease mechanisms and advancing pharmacological and nanotechnological treatments. *J Inflamm Res*. 2025;**18**:2045–72. doi:10.2147/jir.s506103.

7. Rizzetto G, Molinelli E, Radi G, Cirioni O, Brescini L, Giacometti A, et al. MRSA and skin infections in psoriatic patients: therapeutic options and new perspectives. *Antibiotics (Basel)*. 2022;**11(11)**:1504. doi:10.3390/antibiotics11111504
8. Cho M, Yang J, Jung WH, Kim HS. A case of Staphylococcus simulans colonization in a patient with psoriasis. *J Mycol Infect*. 2024;**29(1)**:7–10. doi:10.17966/JMI.2024.29.1.7
9. Sitton B, Walker T, Mital R, Varra V, Kaffenberger J. Antistreptococcal treatment of psoriasis: a systematic review. *Arch Dermatol Res*. 2024 Jun 8;**316(7)**:363. doi:10.1007/s00403-024-03051-8.
10. Miura S, Igarashi A, Okazaki H, et al. Staphylococcus aureus and Streptococcus pyogenes induce psoriasis-related transcriptomes augmented by IL-17A and TNF-α. *J Invest Dermatol*. 2023;**143(8)**:1613-7.e5.