Asia Pac Allergy. 2022 Apr;12(2):e16
https://doi.org/10.5415/apallergy.2022.12.e16
pISSN 2233-8276·eISSN 2233-8268

Asia Pacific allergy

Case Report

Check for updates

Erythrodermic psoriasis in post-coronavirus disease 2019 patient

Irwan Saputra Batubara * **and Windy Keumala Budianti**

Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

OPEN ACCESS

Received: Sep 12, 2021
Accepted: Apr 18, 2022
Published online: Apr 21, 2022

***Correspondence to**
Irwan Saputra Batubara
Department of Dermatology and Venereology, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia.
Tel: +6281221497863
Email: irwan13294@gmail.com

ORCID iDs
Irwan Saputra Batubara
https://orcid.org/0000-0003-0210-7816
Windy Keumala Budianti
https://orcid.org/0000-0002-9613-7698

Conflict of Interest
The authors have no financial conflicts of interest.

Author Contributions
Conceptualization: Irwan Saputra Batubara. Formal analysis: Irwan Saputra Batubara, Windy Keumala Budianti. Investigation: Irwan Saputra Batubara, Windy Keumala Budianti. Methodology: Irwan Saputra Batubara, Windy Keumala Budianti. Project administration: Irwan Saputra Batubara, Windy Keumala Budianti. Writing - original draft: Irwan Saputra

ABSTRACT

Erythrodermic psoriasis (EP) is characterized by generalized erythema and desquamation affecting more than 75% of body surface area and usually accompanied by systemic symptoms. The triggers are medication withdrawal, drugs reactions, and systemic infections including coronavirus disease 2019 (COVID-19). A 46-year-old man with plaque psoriasis suffered from EP following the sudden discontinuation of medications. He was diagnosed with COVID-19 one month before erythroderma appeared. The body surface area involvement was 96% and psoriasis area severity index was 49.8. His general condition and laboratory examination were within normal limits. He was treated with cyclosporine-A for one month after being healed from COVID-19 with significant improvement. Excessive production of proinflammatory cytokines in COVID-19 plays a role in the pathogenesis of psoriasis. This condition should be managed appropriately to minimize the complication. Cyclosporine-A is the first-line therapy for EP because of its effectiveness and good safety profile. It is also shown a beneficial effect in COVID-19 infection *in vitro*.

Keywords: Erythrodermic psoriasis; COVID-19; Cyclosporine-A; Phototherapy; Cytokines; Hyperinflammatory

INTRODUCTION

Erythrodermic psoriasis (EP) is a severe and rare type of psoriasis with a prevalence of less than 3% of all psoriasis cases, manifested as generalized erythema and desquamation affecting more than 75% of the body surface area. Systemic symptoms including fever, tachycardia, lymphadenopathy, arthralgia, and malaise are common in EP patients. It generally occurs in patients with uncontrolled disease. Triggers of EP are sudden discontinuation of medications such as corticosteroids, drug reactions, and systemic infections, including coronavirus disease 2019 (COVID-19) [1, 2]. COVID-19 is a highly contagious respiratory infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). COVID-19 patients experience a hyperinflammatory state which is thought to affect the course of psoriasis due to excessive release of proinflammatory cytokines. Without proper treatment, complications such as anemia, sepsis, heart failure, and even death can occur [2].

Batubara. Writing - review & editing: Irwan Saputra Batubara, Windy Keumala Budianti.

CASE REPORT

A 46-year-old man visited Dermatology Clinic with chief complaints of itchy and painful generalized redness plaques and desquamations on the entire body for the last 2 weeks. He also complained of weakness and mild fever. His complaint appeared 1 month after he was confirmed of COVID-19 infection. In May 2021, he was diagnosed with COVID-19 by real-time-polymerase chain reaction (RT-PCR) and hospitalized for 14 days. On day 10th of hospitalization, he was tested negative for COVID-19 by RT-PCR examination. During hospitalization, he received azithromycin 1×500 mg and oseltamivir 2×75 mg daily. He has been diagnosed with psoriasis vulgaris since 2018 and underwent narrowband ultraviolet B phototherapy with the last dose was 2,667 mJ/cm^2. He also routinely applied topical corticosteroid for skin lesions and tar preparation for the scalp. Due to hospitalization, followed by 14 days of self-isolation, he did not undergo phototherapy and applied topical medication for approximately one month. After he began the phototherapy, his lesions worsened and became erythrodermic. The erythrodermic appeared 4 weeks after he was confirmed of COVID-19 infection. On physical examination, the vital signs and body mass index were normal. The body surface area involved was 96%, and the psoriasis area severity index (PASI) was 49,8 (**Fig. 1A-D**). He had no joint symptoms, and a rheumatologist examination showed no arthritis. Laboratory studies was as follows: haemoglobin 12.2 g/dL, haematocrit 37.1%, thrombocyte 362.000/µL, leucocyte 8.800/µL, urea 20.4 mg/dL, creatinine 0.9 mg/dL, glomerular filtration rate 102.1 mL/min/1.73 m^2, SGOT 15 µ/L, SGPT 13 U/L, and glucose 98 mg/dL. Chest x-ray examination was normal. The patient was given cyclosporine-A (CyA) with an initial dose of 100 mg (1.3 mg/kg) daily for 2 weeks. Because the lesions were improved without complaining of side effects, the dose was increased to 150 mg (2 mg/kg) daily for another 2 weeks. Upon revisited, the lesions were significantly improved with body surface area involvement was 12%, PASI 8, and improvement of delta PASI 81.8% (**Fig. 2A-D**). The patient has given written informed consent regarding the publication of his photos.

DISCUSSION

Although the prevalence of EP is low, it is a potentially life-threatening condition that should be treated appropriately. The trigger of EP is sudden medication withdrawal, drug reactions, and systemic disease, including leukemia, T-cell lymphoma, human immunodeficiency virus, gout arthritis, and COVID-19 [1, 2]. Hyperinflammatory state in COVID-19 is caused by excessive production of cytokines, such as C-reactive protein, interleukin (IL)-2, -6, -7, -10,

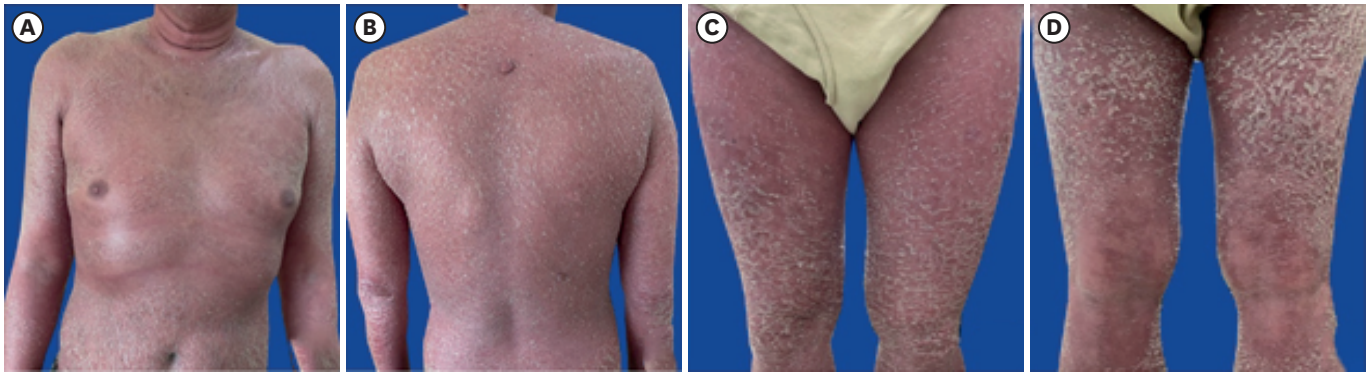

Fig. 1. (A-D) Generalized erythema and desquamation affecting 96% of patient's body surface area.

https://doi.org/10.5415/apallergy.2022.12.e16

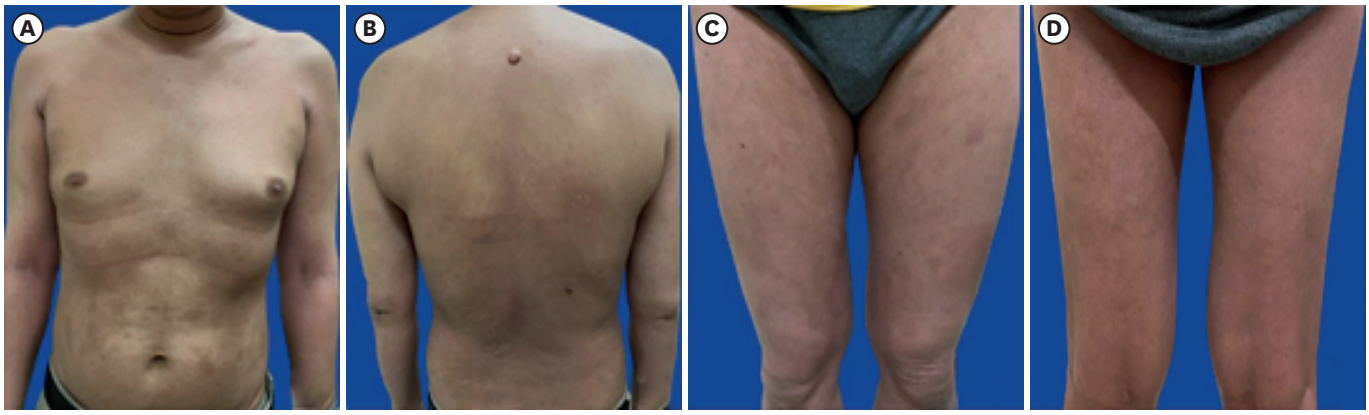

Fig. 2. (A-D) Rapid improvement of skin lesions following cyclosporine therapy for 1 month.

granulocyte-colony stimulating factor, interferon-inducible protein 10, and tumor necrosis factor-alpha. These cytokines play a role in the pathogenesis of psoriasis and worsen the disease [2, 3]. A case of psoriasis exacerbation in COVID-19 was first reported in a 71-year-old woman who received oseltamivir and hydroxychloroquine [4]. Psoriasis exacerbation in this patient might be caused by COVID-19 infection and hydroxychloroquine, and the latter is known to stimulate the differentiation and growth of keratinocytes [5]. A case series of 52 COVID-19 patients with skin diseases found that 9.6% of patients had psoriasis. The burden of emotional stress during pandemics increases the incidence of psoriasis exacerbations. In addition, immunosuppressive drugs for psoriasis increase the risk of COVID-19 infection [6]. The initial management of EP consists of supportive therapy and immunosuppressive medication. In unstable patients, hospitalization should be considered. CyA is recommended as the first-line therapy for EP. It inhibits transcription of IL-2, thereby suppressing the proliferation of T cells. The dose ranges from 1.5 to 5 mg/kg daily [7]. It demonstrated complete remission in 67% EP patients with a mean dose of 4.2 mg/kg daily over 6.3 months [8]. Combination of CyA with methotrexate, alefacept, or etretinate is also effective for EP [7]. *In vitro* study showed that CyA inhibits replication of several types of coronaviruses, including SARS CoV2 [9, 10]. A nonimmunosuppressive analogue of CyA, Alisporivir, showed inhibition effect of SARS-CoV2 *in vitro* [11]. CyA was also preferred as immunosuppression for renal allograft recipients with COVID-19 [12]. It is generally well tolerated with side effects including gastrointestinal symptoms, headache, and impaired renal function. Contraindications of CyA are hypersensitivity reactions, severe renal disease, hypertension, and malignancy [7]. In this patient, there were no contraindications and side effects during the first 2 weeks of therapy. Thus, the dose was increased to 150 mg daily. Phototherapy was discontinued because it triggers the koebnerization in EP [7].

In conclusion, EP is a potentially life-threatening condition that needs prompt management to prevent morbidity and mortality. CyA was shown to be rapidly effective and well tolerated for therapy of EP in the post-COVID-19 patient.

REFERENCES

1. Singh RK, Lee KM, Ucmak D, Brodsky M, Atanelov Z, Farahnik B, Abrouk M, Nakamura M, Zhu TH, Liao W. Erythrodermic psoriasis: pathophysiology and current treatment perspectives. Psoriasis (Auckl) 2016;6:93-104.
PUBMED | CROSSREF

2. Elmas ÖF, Demirbaş A, Kutlu Ö, Bağcıer F, Metin MS, Özyurt K, Akdeniz N, Atasoy M, Türsen Ü, Lotti T. Psoriasis and COVID-19: a narrative review with treatment considerations. Dermatol Ther 2020;33:e13858.
PUBMED | CROSSREF
3. Zhang F, Li H, Zhou Y, Gu Y, Wang L. Caveolin-1 expression in different types of psoriatic lesions: analysis of 66 cases. Indian J Dermatol 2014;59:225-9.
PUBMED | CROSSREF
4. Kutlu Ö, Metin A. A case of exacerbation of psoriasis after oseltamivir and hydroxychloroquine in a patient with COVID-19: Will cases of psoriasis increase after COVID-19 pandemic? Dermatol Ther 2020;33:e13383.
PUBMED | CROSSREF
5. Said A, Bock S, Lajqi T, Müller G, Weindl G. Chloroquine promotes IL-17 production by CD4+ T cells via p38-dependent IL-23 release by monocyte-derived Langerhans-like cells. J Immunol 2014;193:6135-43.
PUBMED | CROSSREF
6. Kutlu Ö, Metin A. Dermatological diseases presented before COVID-19: are patients with psoriasis and superficial fungal infections more vulnerable to the COVID-19? Dermatol Ther 2020;33:e13509.
PUBMED | CROSSREF
7. Lo Y, Tsai TF. Updates on the treatment of erythrodermic psoriasis. Psoriasis (Auckl) 2021;11:59-73.
PUBMED | CROSSREF
8. Management of erythrodermic psoriasis with low-dose cyclosporin. Studio Italiano Multicentrico nella Psoriasi (SIMPSO). Dermatology 1993;187 Suppl 1:30-7.
PUBMED | CROSSREF
9. Pfefferle S, Schöpf J, Kögl M, Friedel CC, Müller MA, Carbajo-Lozoya J, Stellberger T, von Dall'Armi E, Herzog P, Kallies S, Niemeyer D, Ditt V, Kuri T, Züst R, Pumpor K, Hilgenfeld R, Schwarz F, Zimmer R, Steffen I, Weber F, Thiel V, Herrler G, Thiel HJ, Schwegmann-Wessels C, Pöhlmann S, Haas J, Drosten C, von Brunn A. The SARS-coronavirus-host interactome: identification of cyclophilins as target for pan-coronavirus inhibitors. PLoS Pathog 2011;7:e1002331.
PUBMED | CROSSREF
10. Pizzorno A, Padey B, Dubois J, Julien T, Traversier A, Dulière V, Brun P, Lina B, Rosa-Calatrava M, Terrier O. In vitro evaluation of antiviral activity of single and combined repurposable drugs against SARS-CoV-2. Antiviral Res 2020;181:104878.
PUBMED | CROSSREF
11. Softic L, Brillet R, Berry F, Ahnou N, Nevers Q, Morin-Dewaele M, Hamadat S, Bruscella P, Fourati S, Pawlotsky JM, Ahmed-Belkacem A. Inhibition of SARS-CoV-2 Infection by the cyclophilin inhibitor alisporivir (Debio 025). Antimicrob Agents Chemother 2020;64:e00876-20.
PUBMED | CROSSREF
12. Kemmner S, Guba MO, Schönermarck U, Stangl M, Fischereder M. Cyclosporine as a preferred calcineurin inhibitor in renal allograft recipients with COVID-19 infection. Kidney Int 2020;98:507-8.
PUBMED | CROSSREF