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Case Report

Erythrodermic psoriasis in 57 years old male patients treated with systemic corticosteroid

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Abstract Erythroderma is a severe skin disorder that requires immediate and adequate treatment. The most common cause of erythroderma, which can be detrimental to the patient's life, is psoriasis. We describe a case of erythroderma psoriasis (EP) in a 57-year-old male patient without underlying systemic disease. The patient was diagnosed based on his medical history and physical examination. The management is interdisciplinary and comprehensive. Patients' clinical outcomes improved significantly after receiving topical and systemic corticosteroids.

Key words
Emergency, erythroderma, systemic corticosteroid, erythroderma psoriasis.

Introduction

Erythroderma or exfoliative dermatitis is a skin disorder characterized by generalized erythema on more than 90% of the body surface area.[1,2] The presence of scales in varying degrees and variations is the hallmark of skin exfoliation.[1] Erythroderma is a dermatological emergency and requires treatment in a health facility.[1,3] The pathogenesis of erythroderma can be precipitated by the extension/ expansion of a pre-existing skin disorder, an underlying systemic disease, or an allergic reaction to medications.[1-3] Psoriasis is one of the most prevalent causes of erythroderma, approximately 23-25% of cases.[1,4] The prevalence of erythrodermic psoriasis (EP) in psoriasis patients ranges from 1-2%.[4,5] The development of EP is affected by the interaction between hereditary and environmental factors, including numerous inflammatory mediators.[2,4,5] Management of EP patients are comprehensive, comprising supportive and specific management.[1] The treatment of EP patients is not only focused on controlling the inflammatory reaction but also improving the patient's general status.[1,2,4] We presented a case of erythrodermic psoriasis in a 57-year-old male patient**.**

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Case Report

A 57-year-old male came to the emergency room (ER) with the chief complaint of redness throughout his body since a week prior to admission. The body was covered in red patches, including the head, face, trunk, abdomen, arms, legs, and genitals. The redness in the skin developed in conjunction with skin dryness and exfoliation covered in thick scales. Patients reported itching, burning, and pain with the Visual Analogue Scale (VAS) 6-7. Since then, the patient's oral intake and appetite had decreased. The patient had intermittent fever for two weeks. Prior to the onset of the current complaint, the patient observed several red spots developing on various body areas, consisting of the scalp, both upper and lower arms, the back, and both knees to the front lower legs. Red spots emerged more prominently if the extremity had

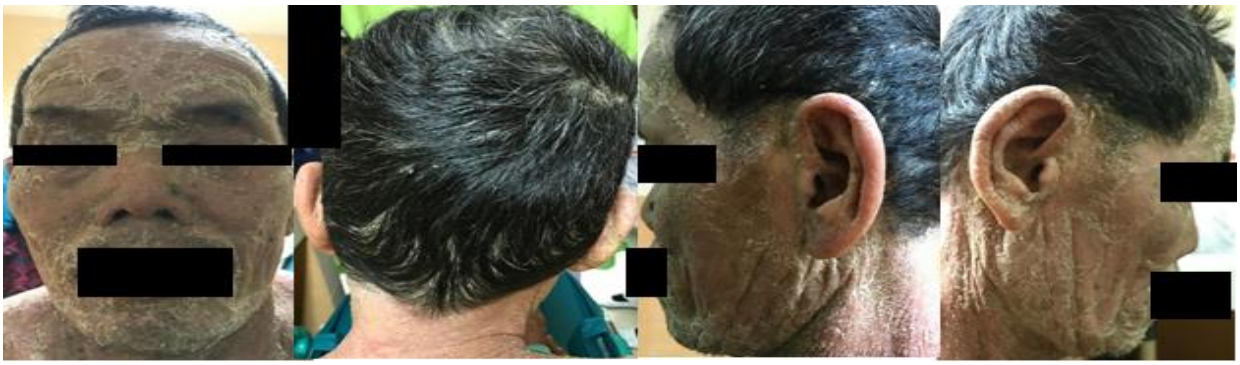

Figure 1 Distribution of generalized lesions in the face and scalp.

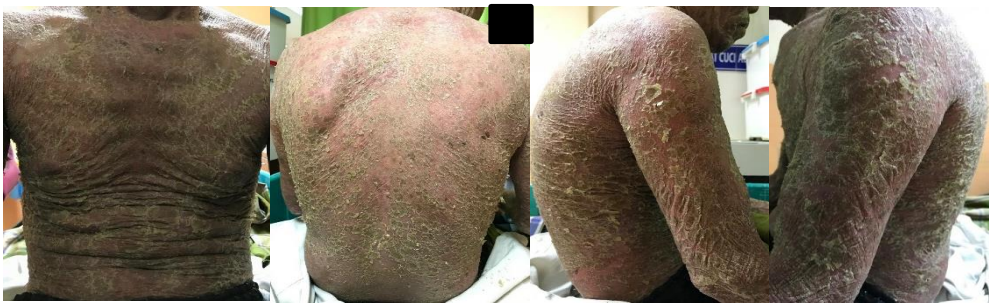

Figure 2 Distribution of generalized lesions in the trunk and upper arms.

been subjected to trauma or impact. They were also accompanied by thick white scales and pruritus and eventually spread to the entire body.

Six months ago, the patient experienced a similar complaint. The patient had previously been treated and received medications from the hospital. The patient did not adhere to the treatment plan consistently. He denied any history of hypertension, diabetes mellitus, cancer, atopic disease, drug allergy, drug abuse, or weight loss.

General Condition

On physical examination, vital signs were unremarkable: blood pressure of 108/78 mmHg, a pulse rate of 108 beats per minute, palpable, strong, and regular, a respiratory rate of 20 cycles per minute, a temperature of 38.4^{o}C, an oxygen saturation level of 98% in room air, and a normal anthropometric status based on Asia-Pacific criteria (bodyweight of 65 kg, a height of 165 cm, and BMI of 24). The examinations on the heart, lungs, abdomen, and lymph nodes were all within normal limits.

Dermatological Status

Head, face, trunk, back, abdomen, superior and inferior extremities, and genitalia: erythematous patches and generalized exfoliation (>90% of body surface area), accompanied with well-defined thick-coated white-yellow scales.

Fingernail and toenail: salmon patch (+), onycholysis (+).

The results of the hematological examination showed hemoglobin of 14.3 g/dL, hematocrit of 42.6%, leukocytes of 12,600/uL, platelets of 339,000/uL, differential count of 0/1/2/78/15/4, the random blood sugar of 117 mg/dL, electrolytes sodium of 142 mmol/L, potassium of 4.1 mmol/L, chloride of 92 mmol/L, albumin of 2.75 g/dL, SGOT of 58 U/L, SGPT of 46 U/L, urea of 75 mg/dL, creatinine of 1.07 mg/dL. Posteroanterior chest x-ray

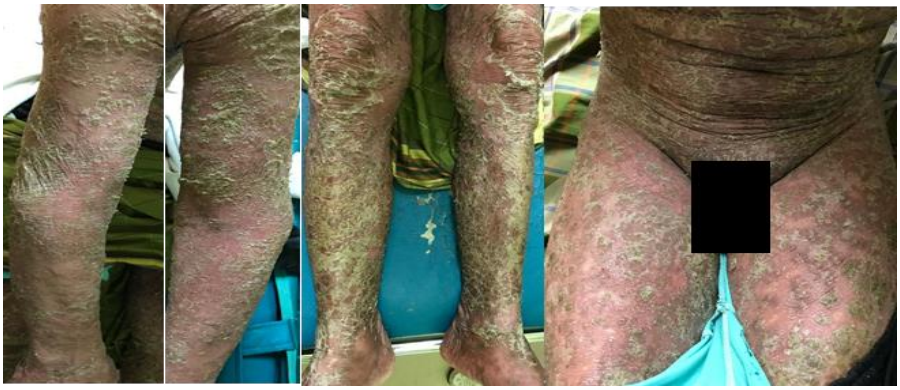

Figure 3 Distribution of generalized lesions in the lower arms, lower legs, abdomen, and genitalia.

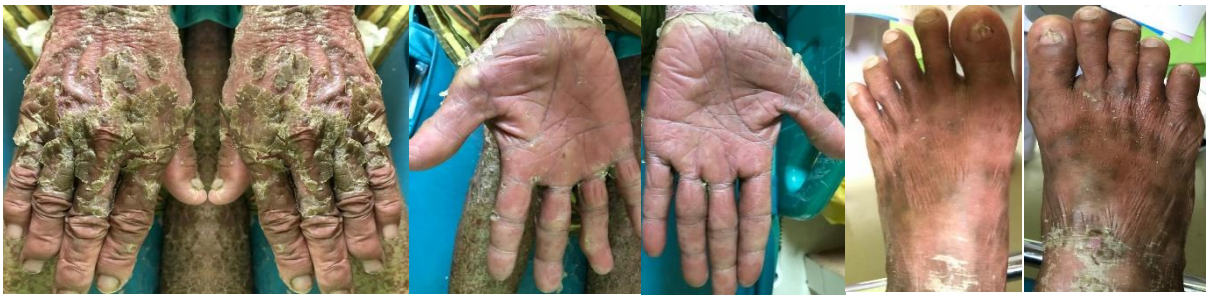

Figure 4 Distribution of generalized lesions in both hands, feet, and the nails.

radiology examination revealed mild cardiomegaly.

The patient's diagnosis was established based on his medical history and dermatological examination, which revealed skin lesions suggestive of erythroderma. The patient's erythroderma cause was identified as psoriasis vulgaris. The differential diagnosis included erythroderma secondary to seborrheic dermatitis. Histopathological examination was not performed on this patient due to the absence of supporting facilities.

Additionally, the Internal Medicine division was also consulted regarding the current case. The patient received systemic management as IV methylprednisolone 125 mg in divided doses administered 62.5mg b.i.d. The methyl-prednisolone dose was gradually tapered until it reached 16 mg/ day. Additional systemic treatments included normal saline 0,9% 500 mL every 12 hours, the antibiotic moxifloxacin 400 mg IV, paracetamol 500 mg IV as the analgesic, cetirizine 10 mg orally, and albumin a capsule t.i.d orally. Patients also obtained topical treatment, including desoximetasone cream 0,25%, topical gentamicin twice a day, Vaseline album, and normal saline 0,9% dressing every four hours for 15 minutes. Clinical improvement was achieved after the medical regimen, as evidenced by diminished erythema, hyperpigmented areas on the skin, and the absence of exfoliative skin.

Discussion

Erythroderma is an emergency that requires specific treatment in a hospital.[1–3] The status of erythroderma patients might be stable or acute to life-threatening. The mortality rate of erythroderma cases ranges between 3-60%.[1] Erythroderma can be induced by various etiologies, including the progression of skin diseases (psoriasis, pityriasis rubra pilaris, seborrheic dermatitis, contact dermatitis, pemphigus foliaceous, bullous pemphigoid, lichen planus), underlying systemic disease

(leukemia, T-cell lymphoma, Human Immunodeficiency Virus (HIV) infection), and allergic reactions to certain drugs/ chemicals.[1,4,6] Aqil *et al.*[7] in their investigation, found that erythroderma was most commonly caused by the expansion of skin disease (43.5%), notably psoriasis (27.2%). A similar study, conducted by Cesar *et al*.[8] on 103 patients with erythroderma revealed that the most prevalent cause of erythroderma in patients was also related to the progression of skin disease (65%) and mainly psoriasis (44.7%). Erythroderma psoriasis can occur due to the evolution of psoriasis disease, excessive treatment, or rapid discontinuation of previous treatment (rebound phenomenon).[1,2,4,5] Erythroderma psoriasis triggers comprise of various conditions, including environmental (trauma, burns, emotional stress, smoking, alcoholism, infections), systemic disorders, and medications or chemicals (lithium, antimalarials, cotrimoxazole, infliximab, acitretin, corticosteroids, tar, lithium, antimalarials, cotrimoxazole, infliximab, acitretin, corticosteroids, tar).[4,5] The pathophysiology of EP involves interactions among cytokines, inflammatory mediators, and adhesion molecules on host cells. The main cytokines and cell adhesion molecules that have a role in the development of EP are interleukin-23 (IL-23), IL-17A, IL-4, IL-10, IL-13, Tumor Necrosis Factor (TNF), Intercellular Adhesion Molecule-1 (ICAM-1), Vascular Adhesion Molecule-1 (VCAM-1), and E-selectin. Cytokines and inflammatory mediators trigger the differentiation of numerous inflammatory cells, such as keratinocytes, Langerhans cells, macrophages, Th2 cells, and Th17 cells. Activation of these cells generates an inflammatory reaction, dilatation of blood vessels, and widespread desquamation.[2,4,6,9]

The onset of EP cases is often between 40-60 years, and the ratio is more significant in males than in females.[1,10] Li *et al*.[11] reported, among 260 individuals with erythroderma, approximately 55% had psoriasis as the aetiology, with a mean age of 46.92±16.71 years and a male to female ratio of 2.67. Erythroderma psoriasis incidence was also age-related in Morocco[7] and Portugal,[8] with a mean age of 41.9 and 50.9 years, respectively, and was predominated by men. Erythroderma psoriasis is defined clinically by the appearance of widespread erythematous patches or plaques (>90% of body surface area), which may or may not be accompanied by scales developing between 2- and 6-days following erythema.[1] Patients with EP may present pitting, an oil-drop or salmon patch, Beau's line, brittle nail, hyperkeratosis, onychodystrophy, or onycholysis.[4,6] The inflammatory response damages the skin barrier, resulting in systemic symptoms and severe complications such as fever, chills, tachycardia, pruritus, lymphadenopathy, fatigue, malaise, myalgia, arthralgia, insomnia, fluid and electrolyte balance disturbances, and dehydration.[1,3,4] The diagnosis can be clinically based on the presence of inflammatory skin lesions displaying generalized erythema with thick scales and history of psoriasis. Moreover, lymphocytes and eosinophils, dilated capillaries, hyperkeratosis, parakeratosis, acanthosis, spongiosis, and microabscesses of Munro may also assist in confirming the diagnosis.[4,6] Other examinations, such as laboratory and radiology, do not benefit in diagnosis but do assist in determining the severity of the disease and patient care.[1,3]

As for this case, a 57-year-old male patient reported a chief complaint of red patches all over the body for one week before admission accompanied by flaky skin with thick scales. The nails showed salmon patches and onycholysis. Before the current complaint occurred, the patient had a history of erythematous patches accompanied by thick, layered scales that only appeared in several parts

of the body, especially the back and extensor extremities. Koebner phenomenon was also found in patients.[1,6] The history of prior red patch lesions before they generalized are the characteristic of psoriasis vulgaris. These patches eventually became widespread throughout the body until now. The current complaint had also happened once six months ago. The patient received previous treatment at the hospital until his skin condition improved but was lost from follow-up. From his medical history and physical examination, the primary diagnosis was erythroderma caused by the progression of the previous psoriasis vulgaris disease. Non-adherence to prior therapy was considered to be the trigger of psoriasis exacerbations to erythroderma. Anggarini *et al*.[12] reported a case of erythroderma induced by an expansion of seborrheic dermatitis in a 60-year-old male patient. In cases of erythroderma due to seborrheic dermatitis, the predilection location is in areas rich in sebaceous glands, namely hairy scalp, eyebrows, nasolabial folds, ears, ear canal, the upper middle part of the chest, and back. It is also characterized by generalized erythema (90-100%) and oily yellowish-white scales.[12,13] The diagnosis of seborrheic dermatitis erythroderma was ruled out in this case. Histopathological examination was not performed on the patient due to the limited resource and facility. Laboratory investigations in the patient revealed an inflammatory response characterized by leukocytosis, an increase in segment neutrophils, and hypoalbuminemia due to extensive desquamation and protein loss.

The management principles for EP cases are classified into general and specific management. General management comprises assessing patients' risk factors and comorbidities, monitoring for symptoms of infection, correcting imbalanced body fluids, proteins, and electrolytes, and reestablishing normal skin barrier function.[2,5,14] Vasodilation of large blood vessels increase blood flow to the skin, resulting in hypothermia in patients with EP.[3] Preventing hypothermia in patients can be accomplished by providing warm or thick blankets and monitoring their temperature frequently.[1] Vasodilation also increases the heart's work rate, which should be monitored via vital signs.[1,3] Fluid administration is intended to maintain the patient's hydration status in the presence of excessive fluid evaporation, which can result in severe dehydration. Imbalances in electrolytes and proteins should be corrected immediately to avoid electrolyte balance disturbances.[1,2] Leg elevation or diuretic medication can be used to treat peripheral oedema caused by fluid shifts to the extravascular space.[3] Sedative antihistamines are administered to alleviate the patient's itching and anxiety. Emollients or moisturizers repair the extensive skin barrier defect caused by desquamation. Antibiotics may be given if a secondary infection is suspected, along with skin smears examination and blood cultures to rule out sepsis.[2–5]

Various methods of EP-specific therapy, including conventional and biologic agents, are currently being developed.[2,5] Numerous conventional or biological medications can be prescribed to treat EP after each drug's benefits and adverse effects are reviewed.[2] Generally, conventional agents such as retinoids, methotrexate, cyclosporine, and mycophenolate mofetil can be given. Biological agents produce an effective response in EP cases. TNF-inhibitors (etanercept, adalimumab, infliximab, and golimumab), IL-12/IL-23 inhibitors (ustekinumab, guselkumab, risankizumab), IL-17 inhibitors (secukinumab, ixekizumab, brodalumab), and other biologic treatments (alefacept, efalizumab, and apremilast) are preferred.[2,5,9]

Systemic corticosteroids are conventional medications whose benefit in systemic psoriasis

therapy is still debatable. Exacerbations may occur following the cessation or reduction of systemic corticosteroid dose used to treat psoriasis. Discontinuation of systemic corticosteroids in patients with psoriasis can result in erythroderma or severe flares of widespread pustular lesions.[1,2,10,14] In cases of acute EP, systemic corticosteroids may be used in combination with conventional immunosuppressive drugs or other biologic agents. If no other conventional medication or biologic agents are available or the patient had contraindications to the treatment, systemic corticosteroid administration may be considered.[2,5,9,10,14]

The reported patients underwent comprehensive management, including vital sign monitoring, fluid, protein, electrolyte correction, and antibiotic medication to prevent subsequent infection. Desoximetasone and moisturizer are applied topically to produce vasoconstriction, anti-inflammatory, and promote re-epithelialization. The patient received systemic corticosteroids i.e. IV methylprednisolone 125 mg in divided doses of 62.5 mg twice a day, tapered to a maintenance dose of 16 mg/ day as the lesion diminished. Systemic corticosteroids are not associated with exacerbations or flares in patients since tapering is done carefully and gradually. Before administering corticosteroids, laboratory and radiological screenings were performed to minimize potential side effects. Systemic corticosteroids were chosen as the primary therapy in this patient because this case was acute, and other conventional agents or biologics were not available in our health facility. The patient acquired hospital treatment and showed remarkable clinical improvement. Extensive erythema diminished, leaving hyperpigmented areas and desquamated skin tissue re-epithelializing. Waspodo *et al*.[10] observed significant clinical improvement within six days of treatment with carefully tapered IV dexamethasone 10 mg b.i.d in a 51-year-old female with EP. Safitri *et al*.[15] had also described a case of EP in a 23-year-old male with concomitant iatrogenic Cushing's syndrome who improved clinically on methotrexate 15 mg/week and prednisone 20 mg/day for three weeks.

Conclusion

Erythroderma psoriasis is a severe form of psoriasis that can be life-threatening. It can be induced by various intrinsic and extrinsic conditions. The diagnosis of EP should be based on histopathology results. The management is multidisciplinary and comprehensive, comprising both general and specific treatment. Systemic corticosteroid with careful tapering has been found enhancing clinical outcomes in EP patients.

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