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Case Report

Case Report: Psoriasis Pustulosa Generalisata in A Patient with Primary Hypothyroidism

Sri Adila Nurainiwati[1], Prajnamita Manindhya El Farah[2*], Isbandiyah[3], Jodii Arlan Kurnia[4,5]

[1] Dermatology and Venereology Department, General Hospital, Muhammadiyah University of Malang, East Java, Indonesia
[2] Faculty of Medicine, University of Muhammadiyah Malang, East Java, Indonesia
[3] Internal Medicine Department General Hospital Muhammadiyah University of Malang, East Java, Indonesia
[4] General Hospital of Abdhi Famili, Banyuwangi, East Java, Indonesia
[5] Magister Program in Hospital Management, Faculty of Medicine, Universitas Brawijaya, East Java, Indonesia

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CORRESPONDING AUTHOR*
Prajnamita Manindhya El Farah
prajnamitamaelfa@gmail.com
Faculty of Medicine, University of Muhammadiyah Malang, East Java, Indonesia

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ABSTRACT

Introduction: Psoriasis is a chronic relapsing pathological skin condition with a genetic basis. Generalized pustular psoriasis (GPP) is the most severe type with an incidence rate of 0.9%. Psoriasis can be associated with various other diseases, one of which is hypothyroidism.

Case Presentation: A 47-year-old woman presented with red patches, scaling, pustules on her face spreading throughout the body accompanied by pruritus, burning sensation, and fever. The patient had a 10-year history of recurrent erythematous scaly skin lesions and a known history of primary hypothyroidism treated with levothyroxine. Management given ceftriaxone IV 2x1g, diphenhydramine IV 2x10mg, ondansetron IV 3x4mg, paracetamol IV 1g PRN, methotrexate tablet 2x2.5mg, inbion tablet 1x1, euthyrox tablet 1x100mg.

Conclusion: GPP is a rare and potentially severe form of psoriasis that requires early recognition and comprehensive management. This case highlights the coexistence of GPP and primary hypothyroidism, suggesting the importance of evaluating thyroid dysfunction and other comorbidities in patients with severe psoriasis. Multidisciplinary management, systemic therapy, laboratory monitoring, and regular follow-up are essential to improve clinical outcomes and prevent complications.

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INTRODUCTION

Psoriasis is a chronic, recurrent pathological skin condition with a genetic basis [1]. associated with the immune system characterized by abnormal growth and differentiation of epidermal cells [2]. Psoriasis can affect the skin, nails, and/or joints and can occur in all age groups, from children and adolescents (under 18 years old) to adults between 40-50 years old [3]. Pustular psoriasis is a less common subtype of psoriasis that can manifest as acute, subacute, or chronic pustular eruptions [4].

Pustular psoriasis is divided into two forms: generalized pustular psoriasis (GPP) and localized pustular psoriasis [5]. encompasses acute GPP, annular pustular psoriasis, pustular psoriasis in infants and adolescents, pustular psoriasis during pregnancy (impetigo herpetiformis), and localized pustular psoriasis (other than hands and feet). For the localized pustular psoriasis type, it includes palmoplantar pustulosis (PPP) and Acropustulosis

(acrodermatitis continua of Hallopeau) [6]. GPP is the most severe and rare type, accounting for 0.9% of all psoriasis cases [7].

Psoriasis can be associated with various other diseases, one of which is thyroid disease. Hypothyroidism is a clinical syndrome due to thyroid hormone deficiency [8]. Clinically, there are three types: secondary hypothyroidism due to pituitary/hypothalamic damage; primary hypothyroidism due to thyroid gland damage; and other causes, such as pharmacological factors, iodine deficiency, iodine excess, or peripheral resistance. The most commonly found is primary hypothyroidism. Therefore, the diagnosis is generally made based on elevated TSH and decreased free T4.

Hypothyroidism is more dominant in women. In the elderly, its clinical presentation tends to be less clear and specific, often resembling symptoms of aging. According to Eapi et al, thyroid hormone imbalance can significantly affect various organs such as the heart, brain, digestive system, bones, muscles, skin, and other organs [9,10]. In a study conducted at Kaiser Permanente Medical Center in Los Angeles, it was found that psoriasis is associated with autoimmune diseases [11]. Here, a case will be reported discussing GPP in a patient with primary hypothyroidism. This case report is presented because GPP is a rarely encountered case, and this report will outline the approach to establishing a diagnosis and management for a patient with GPP and primary hypothyroidism, with the hope of adding insight and knowledge.

The scientific value of this case does not rely solely on the coexistence of GPP and primary hypothyroidism, because thyroid dysfunction has previously been reported as a comorbidity in patients with psoriasis. Rather, the academic importance of this case lies in the clinical complexity of managing acute GPP requiring hospitalization in a patient with active endocrine dysfunction and systemic abnormalities, including obesity, anemia, and hypoalbuminemia. This case highlights the importance of early recognition, multidisciplinary management, careful systemic treatment selection, laboratory monitoring, and cautious interpretation of thyroid dysfunction in severe pustular psoriasis.

CASE PRESENTATION

A 47-year-old woman presented to the Emergency Department of Muhammadiyah University Hospital in Malang (UMM Hospital) with complaints of red, scaly patches with pustules on her face and neck, spreading to the chest, back, both arms, and legs, accompanied by pruritus and a burning sensation for the past 3 weeks. The patches were painful and itchy. She also complained of intermittent fever for 2 weeks, stiffness, and decreased appetite. The patient had a history of erythematous plaques on her skin for 10 years that appeared intermittently. She occasionally took antihistamines to relieve pruritus and used clobetasol propionate 0.05% cream. However, she rarely followed up or had consistent treatment. There was no family history of similar conditions. She had been taking Euthyrox 1x100 for a year due to hypothyroidism.

Physical examination revealed a moderately ill-looking patient with compos mentis consciousness. Vital signs were blood pressure 110/70 mmHg, pulse rate 92 beats per minute, respiratory rate 20 breaths per minute, temperature 38.3°C, and oxygen saturation 96%. Body weight: 84 kg, height: 155 cm, BMI: 35 (obese). General status showed a normocephalic head, anemic appearance in both eyes, and no jaundice. Examination of the ears, nose, throat showed no abnormalities, and there was no lymph node enlargement in the neck. Examination of the chest found a single, regular heart sound (S1 and S2) without murmurs or gallops. Lung examination revealed vesicular breath sounds with no abnormal sounds. Abdominal examination showed normal bowel sounds, no distension, no hepatomegaly, and no splenomegaly. Both upper and lower extremities were warm, with no edema in the lower limbs.

Dermatological examination revealed multiple millet-sized to lentil-sized pustules with well-defined borders, forming a "lake of pus" with erythema, scaling, and crusts on the face, neck, chest, back, both arms, and legs. Nails were within normal limits. There was no oral or ocular involvement, and no joint inflammation was observed. Complete blood count revealed decreased hemoglobin (9 g/dL), hematocrit (28.6%), and increased leukocytes (13,200 uL)

while other hematological parameters were within normal limits. Kidney and liver function tests were normal. Immunoserological examination showed increased CRP (97.21 mg/L), elevated TSH (>6.00 uIU/mL), and decreased free T4 (<1.00 pmol/L). Based on history and physical examination, the working diagnosis for the patient was GPP and primary hypothyroidism.

Skin biopsy was performed to support the diagnosis. Histopathological examination showed psoriasiform epidermal hyperplasia with elongated rete ridges, spongiosis, intraepidermal pustular formation, and inflammatory cell infiltration. Perivascular inflammatory infiltrates were also observed in the dermis. These findings were compatible with pustular psoriasis when correlated with the clinical presentation of widespread pustules on erythematous plaques, systemic symptoms, leukocytosis, and elevated inflammatory markers (Figure 1).

On the first day, the patient complained of pain around the lesions. Initial management included hospitalization with intravenous NS fluid at 14 drops/minute, intravenous ceftriaxone 2x1g, intravenous diphenhydramine 2x10mg, intravenous ondansetron 3x4mg, and intravenous paracetamol 1g (as needed). Oral therapy included mefenamic acid tablets 3x500mg for pain relief. The patient was advised to maintain skin hygiene, avoid stress, get enough rest, drink plenty of fluids, and avoid scratching to prevent new lesions and infections. The patient was referred to the internal medicine department due to elevated TSH and decreased free T4 levels, indicating primary hypothyroidism, ongoing therapy for 7 months, and decreased hemoglobin to 9 g/dL.

Although pustules in GPP are typically sterile, intravenous ceftriaxone was administered empirically because the patient presented with fever, leukocytosis, markedly elevated C-reactive protein, painful widespread lesions, and impaired skin barrier function, raising clinical concern for possible secondary bacterial infection or systemic infection. Ceftriaxone was not intended as disease-specific therapy for GPP, but as empirical antimicrobial coverage while infection had not been fully excluded.

Bacterial culture of pustular fluid and blood culture were not performed before antibiotic administration. This is acknowledged as a limitation, because culture examination would have helped distinguish sterile pustular inflammation from secondary bacterial infection and would have supported more targeted antimicrobial use.

On the second day, the internal medicine department diagnosed primary hypothyroidism and normochromic normocytic anemia. Hence,



Figure 1. Clinical presentation on admission. (A-C). In the anterior trunk, posterior trunk, brachium, gluteal area, and femur, there aregeneralized lesions consisting of well-defined erythematous plaques with multiple pustules on them. Some pustules merge to form a 'lake of pus,' and there are visible plaques accompanied byrough scales



Figure 2 Clinical appearance during hospitalization. (D-F). On the anterior trunk, brachium, and femur regions, there are still erythematous plaques with well-defined borders, along with multiple pustules on them. Some plaques and scales seem to be reducing, while parts of the 'lake of pus' appear dryand peeling



Figure 3. Clinical improvement before discharge. (G-H). On the seventh day of treatment, there was an improvement inthe lesions on the anterior trunk, brachium, femur, erythematous plaques, and the appearance of scales significantly reduced by the seventh day of treatment.

oral therapy was modified to methotrexate tablets 2x2.5mg, inbion tablets 1x1, paracetamol tablets 3x500mg, and euthyrox tablets 1x100mg.

Methotrexate was selected as systemic therapy because the patient had extensive pustular lesions with systemic symptoms requiring hospitalization, while renal and liver function tests were within normal limits. Methotrexate was initiated cautiously at a low dose because the patient had anemia, hypoalbuminemia, systemic inflammation, and reduced general condition during admission. The dose was planned to be adjusted according to clinical response and laboratory monitoring. Monitoring during methotrexate therapy included complete blood count, liver function tests, renal function tests, clinical signs of infection, gastrointestinal symptoms, oral ulceration, and worsening anemia. Folic acid supplementation was recommended to reduce the risk of methotrexate-related hematologic and gastrointestinal adverse effects.

Cyclosporine and biologic agents were considered as alternative systemic therapies for severe GPP. However, methotrexate was chosen because it was available, affordable, familiar in local clinical practice, and considered appropriate after evaluation of renal and liver function. Biologic therapy was not initiated because of limited availability and cost considerations. Cyclosporine was not selected as the initial therapy

because the clinical team prioritized methotrexate with close monitoring in the context of the patient's comorbid anemia and hypoalbuminemia.

By the fifth day, the patient complained of fatigue, dizziness, and nausea. Physical examination showed blood pressure 130/70 mmHg, pulse rate 72 beats per minute, temperature 37.2°C, respiratory rate 20 breaths per minute, and general status was within normal limits. Dermatological examination revealed erythematous plaques with sharp borders, accompanied by multiple pustules. Some plaques had started to peel, and the skin appeared shiny. The "lake of pus" had reduced. Treatment included intravenous NS fluid at 20 drops per minute, intravenous paracetamol 1g, intravenous ondansetron 3x4mg, intravenous ceftriaxone 2x1g, intravenous diphenhydramine 2x1, and oral medications: methotrexate tablets 2x2.5mg, euthyrox tablets 1x100mg, VCO (virgin coconut oil) 2x1, paracetamol tablets 3x500 mg, and Inbion 1x1 (Figure 2).

Laboratory results showed decreased hemoglobin to 7.4 g/dL and decreased albumin to 2.4 g/dL. Additional therapy included transfusion of packed red cells (PRC) 2 bags and oral VIP albumin tablets 3x500 mg, resulting in an increase in hemoglobin to 8.9 g/dL (Figure 3).

The patient's condition had started to improve, with only complaints of stiffness in the hands. The patient was discharged with continued therapy, including inbion tablets 1x1 and VIP albumin tablets 1x1. The patient was scheduled for albumin transfusion at the 1-week post- hospitalization follow-up. At discharge, the patient showed clinical improvement, with reduced erythema, pustules, and scaling. She was advised to continue outpatient follow-up with the dermatology and internal medicine departments for monitoring of skin lesions, thyroid function, anemia, albumin level, and methotrexate-related adverse effects. A follow-up visit was scheduled one week after discharge for reassessment and further management planning (Table 1 and Table 2).

DISCUSSION

This case is clinically relevant because it illustrates the diagnostic and therapeutic challenges of GPP in a patient with multiple comorbid conditions, including primary hypothyroidism, obesity, anemia, and hypoalbuminemia. Although hypothyroidism is a recognized comorbidity of psoriasis, its coexistence with acute GPP may complicate systemic assessment, inflammatory interpretation, and treatment planning. Therefore, this case contributes to the literature by emphasizing structured evaluation, multidisciplinary management, and careful monitoring rather than by claiming a direct causal relationship between hypothyroidism and GPP.

GPP was first described by Von Zumbusch in 1910. It is a rare variant of psoriasis characterized by acute eruptions of multiple sterile pustules on erythematous and edematous bases. Sometimes it can be complicated by excessive infection, hypoalbuminemia, and involvement of internal organs, posing life-threatening risks [11,12]. This variant can occur with or without a history of psoriasis vulgaris and usually recurs in episodes. The patient, a 47-year-old woman, aligns with research conducted in Brazil showing a higher prevalence in women, affecting all ages but particularly between 40-50 years old. This condition can affect all races. The prevalence of pustular psoriasis is higher in Asia, reported as 7.46 cases per 1 million population in Japan, while in the Caucasian population, it's reported as only 1.76 cases per 1 million population in France, with a male-to-female ratio of 1:1.2, both in Japan and France [12,13,14].

The dermatological examination revealed generalized lesions in the form of erythematous plaques with multiple milky to lentil-sized pustules, well-defined, which coalesced with scales and crusts on top. This corresponds to manifestations in GPP, marked by fever occurring several days before the appearance of sterile pustule eruptions measuring 2-3 mm on a red (erythematous) skin base, accompanied by pruritus. The pustular lesions can spread throughout the body and extremities, including palms and soles. Pustules may merge, forming a 'lake of pus,' indicating increased disease severity. The confluent pustular lesions easily rupture, leaving erosions, causing pain.

Systemic symptoms that may accompany this condition include fever with chills, continuous

Table 1. Clinical Timeline

Time Point	Clinical Findings	Investigation	Management	Outcome
10 years before admission	Recurrent erythematous scaly plaques	Not routinely evaluated	Intermittent topical corticosteroid and antihistamine use	Recurrent flares
1 year before admission	Diagnosed with primary hypothyroidism	Thyroid function examination	Levothyroxine 100 mcg once daily	Continued treatment
3 weeks before admission	Worsening erythematous plaques with pustules, pruritus, burning sensation, and pain	-	Inconsistent treatment	Lesions progressed
Admission day	Generalized pustules, fever, systemic symptoms	CBC, CRP, TSH, FT4, renal and liver function tests	Hospitalization, IV fluids, symptomatic therapy, empirical ceftriaxone	GPP suspected
Hospital day 2	Persistent generalized pustular lesions	Internal medicine evaluation	Methotrexate initiated, levothyroxine continued, anemia evaluated	Multidisciplinary management
Hospital day 5	Reduced pustules, peeling plaques, fatigue, dizziness	Hb 7.4 g/dL, albumin 2.4 g/dL	PRC transfusion, albumin supplementation, continued systemic therapy	Clinical improvement
Hospital day 7	Reduced erythema, pustules, and scaling	Follow-up clinical evaluation	Discharge planning and outpatient follow-up	Improved condition
1 week after discharge	Follow-up planned	Albumin evaluation planned	Continued outpatient care	Scheduled follow-up

pruritus, headaches, nausea, malaise, muscle weakness, and joint pain. GPP can also affect mucous membranes, causing fissures and erythema on the tongue [15]. The patient complained of systemic symptoms like fever, nausea, dizziness, body stiffness, and pruritus all over the body. No nail abnormalities, oral or ocular involvement, or joint inflammation were observed in this patient. Biopsy results showed intraepidermal blisters, spongiosis, and elongated rete ridges. Perivascular infiltrates and dermal papillae were also noted.

Histopathologically, GPP is characterized by neutrophilic accumulation within the epidermis, which may form spongiform pustules of Kogoj. Other supportive features include psoriasiform epidermal hyperplasia, elongated rete ridges, parakeratosis, Munro microabscesses, thinning of the suprapapillary plates, dilated capillaries in the papillary dermis, and superficial perivascular inflammatory infiltrates. In this patient, the biopsy findings of psoriasiform epidermal changes, spongiosis, intraepidermal pustular formation, and dermal inflammatory infiltrates supported the diagnosis of GPP in the appropriate clinical context.

Based on history, physical examination, and supporting tests, the patient was diagnosed with GPP. This aligns with the diagnostic criteria for GPP, including systemic symptoms like fever and malaise, multiple aseptic pustules present on erythematous skin throughout the body or extensively, some of which merge to form a 'lake of pus,' leukocytosis with neutrophilia, increased erythrocyte sedimentation rate, and elevated CRP levels [16].

Table 2. Laboratory Trend Table

Parameter	Admission	Hospital Day 5	After Intervention / Before Discharge	Interpretation
Hemoglobin	9.0 g/dL	7.4 g/dL	8.9 g/dL after PRC transfusion	Anemia worsened and improved after transfusion
Hematocrit	28.6%	Not available	Not available	Consistent with anemia
Leukocyte count	13,200/µL	Not available	Not available	Leukocytosis suggesting systemic inflammation or possible infection
C-reactive protein	97.21 mg/L	Not repeated / not available	Not available	Markedly elevated inflammatory marker
Albumin	Not available	2.4 g/dL	Not available	Hypoalbuminemia requiring supplementation
TSH	>6.00 µIU/mL	Not repeated	Not available	Consistent with hypothyroid status
Free T4	<1.00 pmol/L	Not repeated	Not available	Consistent with primary hypothyroidism

The main characteristic of GPP is recurrent episodes of sterile pustule formation due to massive infiltration by inflammatory cells like neutrophils and mononuclear cells in the epidermis. GPP can appear with pre-existing plaque psoriasis. However, GPP has also been proven to occur independently [17]. GPP is characterized by widespread fiery erythema [16]. with sterile pustule eruptions measuring 2-3 mm. Lesions can spread throughout the body, including the extremities. Pustules may merge, forming a 'lake of pus,' indicating increased disease severity. Confluent pustular lesions rupture easily, leaving erosions and causing pain. Systemic symptoms may include fever with chills, continuous pruritus, headaches, nausea, malaise, anorexia, muscle weakness, and joint pain. GPP can also affect mucous membranes, causing fissures and erythema on the tongue [15].

The immunopathogenetic mechanism underlying GPP is highly complex. Histopathologically, GPP is characterized by extensive epidermal infiltration by neutrophils and mononuclear cells, leading to clinically visible sterile pustule formation. Excessive expression or loss-of-function mutations in its antagonist, IL-36RN, are believed to play a central role in the pathogenesis of pustular psoriasis. Mutations in the IL-36RN gene result in unstable IL-36RA proteins with reduced affinity to their receptors. IL-36RA molecules inhibit the effects of some cytokines related to IL-36, including IL-1 cytokine family members (IL-36α, IL-36β, IL-36γ), highly expressed in the skin. Without functional IL-36RA, IL-36 is unimpeded in increasing the levels of proinflammatory mediators. IL-36 receptors are found in several types of skin cells, and when activated, cause inflammation on the skin and symptoms of GPP [18]

The development and severity of psoriasis exacerbations involve interactions between genetic and environmental factors [19]. There are several triggers for Generalized Pustular Psoriasis, including pregnancy, medications, sunlight exposure, alcohol, smoking, viral and bacterial infections, secondary hypocalcemia due to hypothyroidism, emotional stress, trauma, climate, and idiopathic factors [2,20,21]. Patients with pustular psoriasis generally may have pre-existing plaque psoriasis or develop it after a pustular episode. Repeated infections or sudden discontinuation of systemic or occasionally ultrapotent topical corticosteroids can also trigger psoriasis [19]

From the patient's history, it's suspected that triggering factors for the psoriasis include a history of previous skin conditions where the patient didn't regularly seek medical attention or comply with treatment, allowing for repeated infections. Environmental factors, emotional stress, and obesity might have contributed to the progression of the experienced psoriasis. Primary hypothyroidism may represent a relevant comorbidity in this patient, but its role in the onset

or exacerbation of GPP cannot be determined from this case alone.

According to a study conducted in America, there's a significant link between psoriasis and hypothyroidism. The skin is the site of synthesis and metabolism of several neuropeptides, including components of the Hypothalamus-Pituitary-Adrenal (HPA) and Hypothalamus-Pituitary-Thyroid (HPT) axes and is also a source of vitamin D. Thyroid hormone receptors are expressed in human skin and are crucial in stimulating keratinocyte proliferation and epidermal differentiation, acting as endogenous inhibitors of skin inflammation, hypothesized to play a role in psoriasis pathogenesis [22].

However, the coexistence of GPP and primary hypothyroidism in this patient should be interpreted cautiously. Previous studies have reported an association between psoriasis and thyroid dysfunction, possibly related to shared immune-mediated mechanisms, inflammatory cytokine activity, and autoimmune susceptibility. Thyroid hormones may also influence keratinocyte proliferation, epidermal differentiation, and skin barrier homeostasis. However, the exact relationship between thyroid dysfunction and GPP remains unclear.

In this case, primary hypothyroidism should be considered a relevant comorbidity rather than a direct cause of GPP. The patient's thyroid dysfunction, obesity, anemia, hypoalbuminemia, and systemic inflammation may have contributed to the overall clinical complexity. Nevertheless, a causal relationship between hypothyroidism and GPP cannot be established from a single case report. Further observational and mechanistic studies are needed to clarify whether thyroid dysfunction influences the onset, recurrence, or severity of pustular psoriasis.

Based on research, TH-1 cells play a role in the immunopathogenesis of psoriasis and autoimmune thyroid diseases [23]. While the exact underlying pathology remains unclear, environmental, genetic, immunological, and hormonal factors potentially influence each other [24]. Patients with thyroid dysfunction show significantly higher Psoriasis Area Severity Index (PASI) scores and increased serum C-reactive protein (CRP) levels. The prevalence of thyroid dysfunction in GPP patients is notably high at 45% [25]. Apart from hypothyroidism, the patient also complains of anemia. Anemia isn't commonly reported as a comorbidity in psoriasis [26]. But it can worsen in the presence of psoriasis. Excessive mitosis of keratinocyte cells occurs in psoriasis. During mitosis, cells use folate as a coenzyme in the methylation process during DNA synthesis, leading to a tendency for folate deficiency in psoriasis.

One treatment for psoriasis involves Methotrexate (MTX), which inhibits the enzyme dihydrofolate reductase, preventing folate from converting to its active form. As a result, the amount of folate available for DNA synthesis within the cell decreases, potentially disrupting the mitosis process in psoriasis, leading to reduced folate levels and causing folate deficiency anemia [27].

Management of GPP ideally involves bed rest and hospitalization to prevent fluid and protein loss, stabilize body temperature, improve skin barrier function, avoid possible complications since psoriasis patients have increased morbidity and mortality related to cardiovascular disorders, and use topical or systemic therapies. Topical treatments include corticosteroids (clobetasol, halobetasol, etc.), retinoids, immunomodulators, salicylic acid, coal tar, ditranol (anthralin) 0.2-0.8%, and emollients, while systemic treatments include methotrexate.

Generally, topical treatment is effective and relatively safe compared to systemic treatment. Methotrexate is a folate analog that competitively inhibits dihydrofolate reductase enzyme. Its main mechanism involves inhibiting thymidylate and purine synthesis, leading to decreased DNA and RNA synthesis. Disruption in nucleic acid synthesis results in the inactivation of T cells and keratinocytes, believed to have antiproliferative and immunomodulatory effects. Methotrexate is often prescribed worldwide for psoriasis, especially in developing countries. Based on evidence, MTX is a safe systemic treatment choice for moderate-to-severe psoriasis. According to the National Institute for Health and Care Excellence (NICE), patients with psoriasis who meet systemic therapy criteria should use methotrexate as first-line therapy due to its good efficacy and safety [19].

Apart from methotrexate, the patient was given virgin coconut oil (VCO) orally. Virgin Coconut Oil (VCO) was used only as supportive adjunctive care and was not considered a disease-modifying therapy for Generalized Pustular Psoriasis (GPP). Although VCO may have moisturizing and skin barrier-supporting properties, current evidence is insufficient to recommend it as primary therapy for GPP. Therefore, clinical improvement in this case should mainly be attributed to hospitalization, systemic therapy, supportive care, correction of systemic abnormalities, and multidisciplinary management rather than to VCO administration. VCO contains 92% medium-chain fatty acids, such as lauric acid, which can be directly absorbed through the intestinal wall, a faster process that bypasses hydrolysis and enzymatic processes. It is then supplied to the bloodstream and sent to the liver for metabolism. In the liver, VCO is processed into energy that enhances the function of all endocrine glands, organs, and body tissues. VCO can be used as an antimicrobial, immune system booster, antibiotic, to maintain heart and blood vessel health, to prevent osteoporosis, to manage diabetes, to boost stamina, and to preserve skin and hair health [28].

This case highlights the need for hospital-based dermatology services to establish a clear clinical pathway for the early recognition, hospitalization, and multidisciplinary management of GPP. Because GPP is a rare but potentially life-threatening inflammatory skin disease, early diagnosis, rapid systemic treatment, laboratory monitoring, and evaluation of systemic complications are essential to prevent morbidity. Recent international consensus also emphasizes the importance of standardized diagnostic criteria and appropriate clinical assessment in patients with suspected GPP [29]. Collaboration between dermatology, internal medicine, clinical pathology, pharmacy, and nutrition units is important, especially in patients with comorbid conditions such as primary hypothyroidism, obesity, anemia, and hypoalbuminemia.

This case also emphasizes the importance of medication safety, continuity of care, and standardized clinical documentation. Systemic treatment such as methotrexate requires accurate dosing, laboratory monitoring, and patient education to prevent adverse effects. Hospitals should strengthen discharge planning, follow-up systems, and patient counseling regarding treatment adherence, recognition of flare symptoms, avoidance of triggering factors, and the importance of regular control for both psoriasis and thyroid disease. In addition, rare cases such as GPP should be documented using standardized case report guidelines to improve the quality, transparency, and clinical usefulness of published reports [30].

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying clinical images. All identifying information has been removed to protect patient confidentiality.

CONCLUSION

GPP is a rare but severe inflammatory skin disease that may present with widespread pustular eruptions and systemic manifestations. This case describes a 47-year-old woman with GPP accompanied by primary hypothyroidism, obesity, anemia, and hypoalbuminemia. The diagnosis was established based on clinical manifestations, systemic symptoms, laboratory findings, and histopathological features. Hospitalization, supportive therapy, systemic treatment with methotrexate, management of hypothyroidism, and multidisciplinary care resulted in clinical improvement.

The coexistence of GPP and primary hypothyroidism in this case highlights the importance of evaluating comorbid conditions in patients with severe psoriasis. Although thyroid dysfunction may contribute to immune dysregulation and inflammatory activity, a causal relationship cannot be established from a single case report. Early recognition, appropriate systemic therapy, laboratory monitoring, patient education, and structured follow-up are essential to prevent complications, reduce recurrence, and improve patient outcomes.

This case report has several limitations. First, bacterial culture of pustular lesions and blood culture were not performed before empirical antibiotic administration, limiting the ability to

microbiologically confirm or exclude secondary infection. Second, thyroid antibody examination was not available, so autoimmune thyroiditis could not be confirmed. Third, serial measurements of CRP, TSH, and free T4 were not completely documented during hospitalization. Fourth, long-term follow-up data regarding recurrence, methotrexate dose adjustment, and thyroid function control were limited. These limitations should be considered when interpreting the relationship between GPP and primary hypothyroidism in this patient.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this case report. The authors have no financial, personal, or professional relationships that could inappropriately influence the content, interpretation, or conclusions of this manuscript.

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