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CASE REPORT

The Uncommon Coexistence of Mid-Borderline Leprosy and Generalized Pustular Psoriasis

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Abstract: Leprosy is a chronic granulomatous disease caused by *Mycobacterium leprae* that primarily affects the skin and peripheral nerves, whereas generalized pustular psoriasis (GPP) is a rare and severe form of psoriasis characterized by widespread sterile pustules and systemic symptoms. Their coexistence is extremely rare due to distinct genetic, immunologic, and epidemiologic profiles. We report a case of a 28-year-old female presenting with both mid-borderline (BB) leprosy and severe GPP. She initially developed numb, erythematous patches on her extremities, followed by pustular eruptions on her right arm that became generalized. Physical examination revealed lagophthalmos, right claw hand, and glove-and-stocking anesthesia without nerve enlargement. Skin lesions included anesthetic macules and plaques on extremities, punched-out lesions on the back, and pustules with crusting and scaling on the face and extremities. A slit-skin smear showed a bacterial index of 1+, and Gram staining of pustules revealed polymorphonuclear cells without bacteria. Histopathology from punched-out lesions revealed granulomas with epithelioid cells and Langhans giant cells. Biopsy of pustules showed features consistent with GPP, including psoriasiform hyperplasia, Munro's abscesses, and Kogoj's spongiform pustules. The patient was diagnosed with BB leprosy with severe reversal reaction coexisting with GPP. She was treated with WHO-recommended multidrug therapy for multibacillary leprosy and systemic corticosteroids, leading to marked clinical improvement within 47 days. This case highlights the importance of recognizing rare coexisting conditions of leprosy and autoimmune diseases, emphasizing the need for a comprehensive diagnostic approach and prompt management to achieve favourable outcomes.

Keywords: generalized pustular psoriasis, leprosy, reversal reaction

Introduction

Leprosy, also known as Hansen's disease, is a chronic granulomatous infection caused by *Mycobacterium leprae* (*M. leprae*) or *Mycobacterium lepromatosis*, predominantly affecting the skin and peripheral nerves.[1] This disease manifests with a broad spectrum of clinical presentations, ranging from localized skin lesions to extensive peripheral neuropathy, depending on the host's immune response.[2,3] According to the World Health Organization (WHO), 172,717 new leprosy cases were reported worldwide in 2024, with the South-East Asia Region accounting for 72% of the global burden.[4] India, Brazil, and Indonesia remained the three most affected countries, collectively contributing 79.8% of all new cases.[4,5] Indonesia continues to rank among the highest-burden countries globally, with 14,698 new cases reported in 2024, compared with 14,376 cases in 2023.[4,6]

Leprosy is primarily classified into two categories by the WHO: paucibacillary (PB) and multibacillary (MB).[3] An alternative classification system proposed by Ridley and Jopling further categorizes leprosy into five distinct forms based on clinical, histological, and immunological criteria: tuberculoid (TT), borderline tuberculoid (BT), mid-borderline (BB), borderline lepromatous (BL), and lepromatous leprosy (LL).[1,3] The PB form typically encompasses TT and BT, while the MB form includes BB, BL, and LL, reflecting the diverse immunopathological spectrum of the disease.[3] Despite

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significant progress in leprosy management, rare coexistence with other dermatological or systemic conditions, such as psoriasis, poses diagnostic and therapeutic challenges.[7,8]

Generalized pustular psoriasis (GPP), is a severe and potentially life-threatening variant of psoriasis, clinically characterized by the rapid eruption of sterile pustules on an erythematous and inflamed skin.[9] GPP is often accompanied with systemic symptoms, including fever, malaise, and leukocytosis, which may progress to sepsis and organ failure if left untreated.[9–11] This T helper (Th)17-mediated autoimmune disorder is in stark contrast to the immunological mechanisms underlying leprosy, which predominantly involve Th1 (tuberculoid form) or Th2 (lepromatous form) responses.[12]

The coexistence of leprosy and GPP is exceedingly uncommon, given their distinct in epidemiological, genetic, and immune mechanisms underlying the two diseases.[7,13] A large-scale study in 1992 conducted by Kumar et al[14] identified only 20 cases of concurrent leprosy and psoriasis among 145,661 leprosy patients, corresponding to an incidence of approximately 1.4 cases per 10,000 patients. The coexistence of these diseases not only complicates clinical diagnosis but also challenges therapeutic decision-making, as the symptoms of one condition may mask or mimic the other, leading to potential misdiagnosis.[15]

Here, we report a rare case of BB-type leprosy with severe reversal reaction coexisting with GPP in a 28-year-old female. This case aimed to contribute to the limited literature on this topic and emphasize the importance of managing patients with overlapping autoimmune and infectious conditions.

Case Presentation

A 28-year-old female, currently in the third month of multidrug therapy (MDT) for MB leprosy, presented to the Emergency Department of Hasan Sadikin Hospital Bandung with widespread pustules on erythematous macules covering almost her entire body. The lesions were accompanied by intermittent fever and mild pruritus, and had progressively worsened over the past week. The initial manifestation occurred 10 days prior, characterized by pustules on the right arm, which subsequently spread to the face, chest, abdomen, left arm, and both legs. Ruptured pustules formed yellowish crusts, some of which developed hemorrhagic crusts. The patient had been previously hospitalized at Tasik Medika Citratama (TMC) Hospital for five days, where she was diagnosed with GPP. During that admission, oral methotrexate (15 mg/week) was administered, leading to a reduction and thinning of pustular lesions and erythematous macules. However, the intermittent fever persisted despite treatment.

Three months before the current admission, the patient complained of numbness erythematous patches on the right forearm, left leg, and right foot. One month later, she consulted a dermatologist and was prescribed a compounded topical medication along with small white tablets taken twice daily. No significant improvement was observed. Over the following two weeks, the skin lesions progressively increased in number and distribution, involving the face, trunk, both arms, legs, and feet. The lesions became slightly raised and more erythematous, particularly on the face, where they appeared increasingly red and painful. Recurrence of lesions was observed, especially following episodes of sleep deprivation. Due to worsening symptoms, the patient visited Tasik Medika Citratama (TMC) Hospital, Tasikmalaya, West Java, Indonesia where she was diagnosed with MB-type leprosy. Notably, the patient had a positive family history of leprosy, as both her mother and grandfather had completed 12-month treatments at a public health center. She also reported difficulty closing her right eyelid and straightening the fingers of her right hand, as well as frequent slipping of her right sandal while walking for one month. Additionally, she had untreated dental caries for the past one year.

The physical examination showed lagophthalmos of the right eyelid and a claw hand deformity of the right hand involving all fingers, with hyperextension at the metacarpophalangeal joints and flexion at the proximal and distal interphalangeal joints, consistent with combined ulnar and median nerves involvement. There was no evidence of eyebrow loss, ear lobe infiltration, or nail changes. Both upper extremities (Figure 1A) and lower extremities (Figure 1B) showed erythematous plaques with scales and crusts, while anesthesia was confined to the distal extermities. Punched-out lesions showing clear inner and outer borders, overlying scales, and focal erosions were observed on the back (Figure 1C). Widespread pustular lesions on an erythematous background with scaling were present on the chest (Figure 2A), abdomen (Figure 2B), and upper thighs (Figure 2C). Additionally, glove-and-stocking anesthesia was noted bilaterally in both hands and feet. No nerve enlargement was observed in the great auricular, ulnar, common peroneal, or

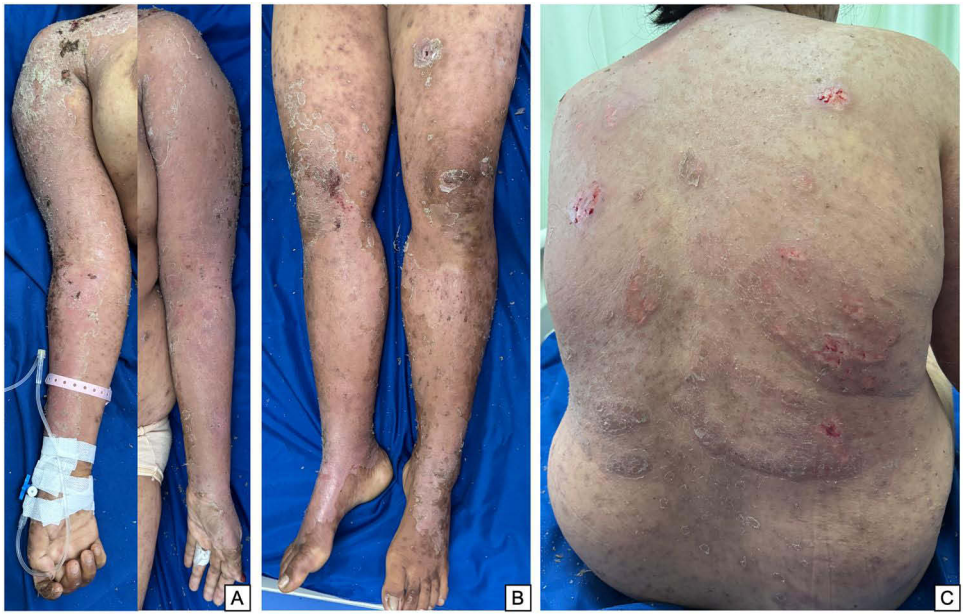

Figure 1 Physical examination on the first day of observation revealed erythematous plaques with scales and crusts on both upper extremities, accompanied by a claw hand deformity of the right hand (**A**). Both lower extremities were affected by erythematous plaques with overlying scales and crusts, with anesthesia confined to the distal extremities (**B**). Punched-out lesions showing clear inner and outer borders, overlying scales, and focal erosions were observed on the back (**C**).

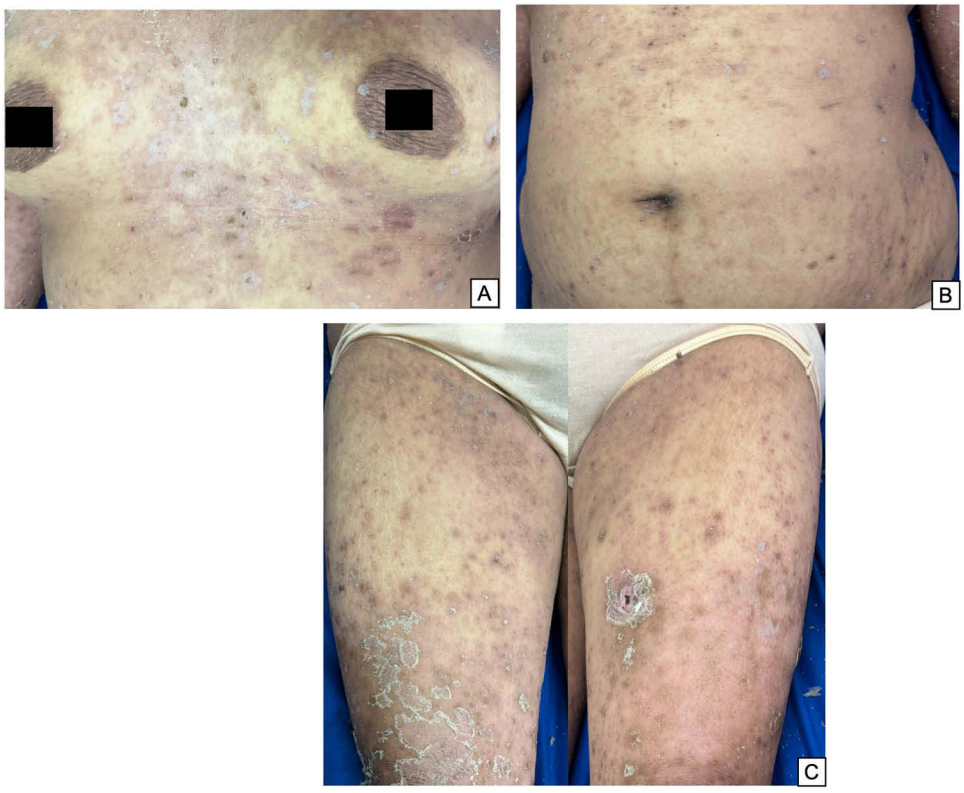

Figure 2 Widespread pustular lesions on an erythematous background with scaling involved the chest (**A**), abdomen (**B**), and upper thighs (**C**).

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posterior tibial nerves. Based on the presence of lagophthalmos and visible claw hand deformity, the patient was classified as having World Health Organization grade 2 disability.

Slit–skin smear examination revealed the presence of acid-fast bacilli (AFB) with an average bacterial index (BI) of 1 +. However, the morphological index (MI) could not be reliably assessed due to the low bacterial load. Gram staining of pustules demonstrated polymorphonuclear cells without bacteria. Histopathological examination was conducted on biopsy samples from both a punched-out lesion on the back and a pustular lesion on the right upper leg. Histopathological features from the punched-out lesion revealed granulomatous reactions with epithelioid cells, Langhans giant cells, and lymphocyte infiltration (Figure 3), suggestive of BT-type leprosy. Conversely, histopathological analysis of the pustular lesion from the right upper leg demonstrated psoriasiform reactions with mild acanthosis, spongiosis, Munro's microabscesses, and Kogoj's spongiform pustules (Figure 4), confirming the diagnosis of GPP. Although histopathological findings of the punched-out lesion suggested of BT-type leprosy, the presence of punched-out lesions and a BI of 1+ supported a clinical diagnosis of BB-type leprosy. Therefore, the patient was diagnosed with BB-type leprosy with a severe reversal reaction coexisting with GPP. She continued the MDT-MB regimen for 12 months and was treated with intravenous methylprednisolone at a dose of 125 mg/day (equivalent to 2 mg/kg body weight of prednisolone per day) to manage the GPP, followed by gradual tapering to prevent recurrence. By the 47th day of

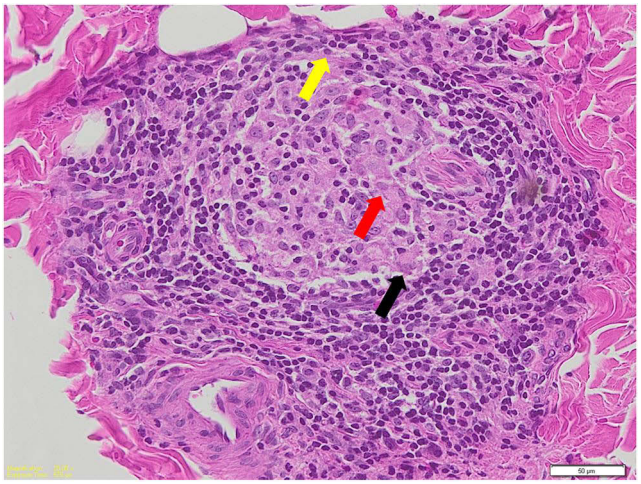

Figure 3 Histopathological findings of the punched-out lesion with hematoxylin-eosin stains (200 times magnification). There was granulomatous infiltration with epithelioid cells (red arrow), Langhans giant cells (black arrow), and lymphocytes (yellow arrow).

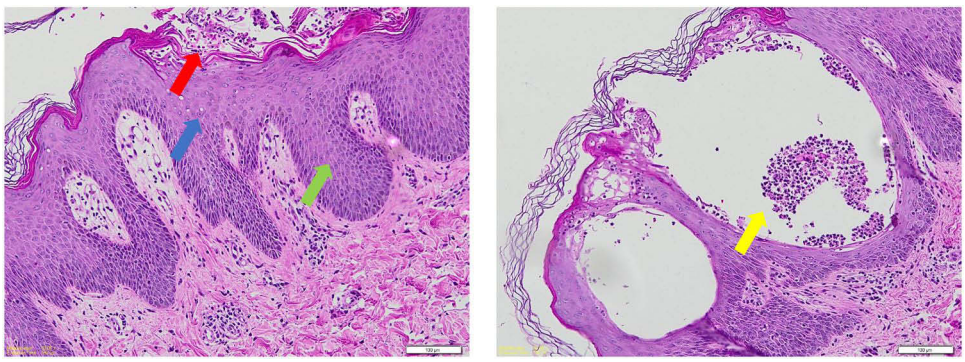

Figure 4 Histopathological findings of the pustular lesion with hematoxylin-eosin stains (100 times magnification). There was psoriasiform reaction with mild acanthosis (green arrow), mild spongiosis (blue arrow), Munro's microabscesses (red arrow), and Kogoj's spongiform pustules (yellow arrow).

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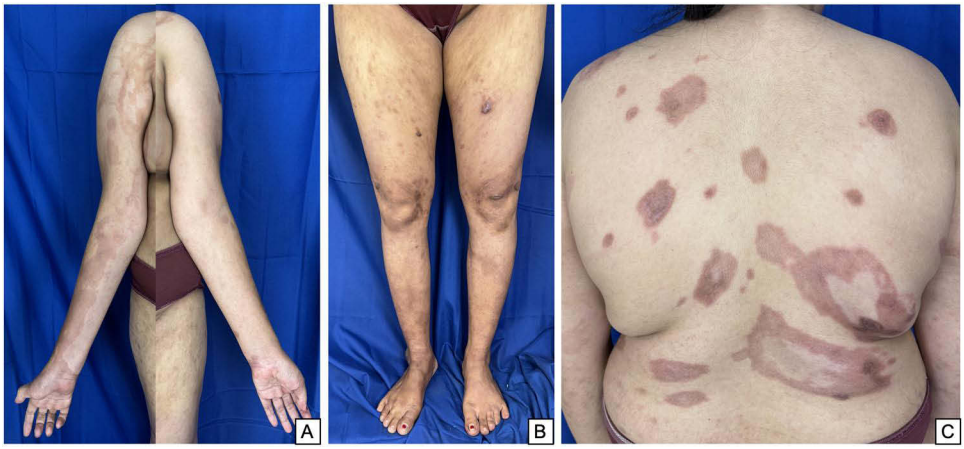

Figure 5 Clinical findings on day 47 demonstrating marked improvement, with the lesions evolving into hyperpigmented macules without new eruptions on both arms (**A**), legs (**B**), and back (**C**).

observation, significant improvement was observed, with transformation of the erythematous macules and plaques into hyperpigmented macules and no new pustular lesions on both arms (Figure 5A), back (Figure 5B), and legs (Figure 5C).

Discussion and Conclusion

Leprosy remains one of the most significant neglected tropical diseases despite global efforts to control its spread through MDT.[16,17] Approximately 200,000 new cases continue to be reported annually in over 120 endemic countries.[16] The diagnosis of leprosy is established based on the presence of at least one of three cardinal signs: 1) hypopigmented or erythematous skin lesions with sensory loss, 2) thickened peripheral nerves with function impairment, or 3) the detection of AFB in slit–skin smears.[1,3] Leprosy exhibits a spectrum of clinical manifestations that largely depend on the host's immune response.[1,13]

Borderline leprosy is an immunologically unstable form of the disease, characterized by skin infiltration that varies in number from a few to multiple lesions, affecting one or more areas of the body.[1] In BB-type leprosy, typical lesions present as annular or punched-out lesions with a clear inner and outer border.[18] Examination of AFB in BB-type leprosy shows a moderate number of bacilli.[3] The patient in this case report was diagnosed with BB-type leprosy based on the presence of punched-out lesions, anesthetic erythematous macules and plaques, and the detection of AFB with an average BI of 1+.

Generalized pustular psoriasis is an acute and severe variant of psoriasis, characterized by widespread sterile pustules and systemic symptoms.[19] Clinically, GPP presents with a rapid onset of sterile pustules on an erythematous skin, often accompanied by systemic symptoms, such as fever, malaise, and fatigue.[11,19] If left untreated, GPP can be life-threatening, with severe complications including sepsis, renal failure, and cardiovascular dysfunction.[11] The pathogenesis of GPP involves dysregulated interleukin (IL)-36 signaling, often due to loss-of-function mutations in the IL-36 receptor antagonist (IL-36Ra).[11,19] This dysregulation promotes neutrophil recruitment and sustained keratinocyte activation, leading to intense inflammation and pustule formation.[9,11] In this case, the diagnosis of GPP was established based on the presence of widespread sterile pustules along with systemic symptoms, further confirmed through histopathological examination.

The coexistence of leprosy with other systemic or dermatological conditions, such as psoriasis, is an exceptionally rare phenomenon.[7,8,13] Despite their potential coexistence, these two diseases arise from distinct pathogenic mechanisms. Leprosy, particularly the tuberculoid form, is driven by a robust Th1-mediated immune response, characterized by the production of IL-2, interferon-gamma (IFN)- γ, and IL-12 by CD4+ T cells. This response promotes macrophage activation, epithelioid cell transformation, and granuloma formation, which are critical for controlling the bacterial burden of *M. leprae*.[12] In contrast, the lepromatous form is driven by a predominant Th2 response, marked by increased production of IL-4, IL-5, and IL-10, along with high levels of antibody production. These mechanisms suppress

macrophage function, weaken cell-mediated immunity, and promote bacillary proliferation.[1,12] In this case report, the patient was diagnosed with BB-type leprosy, which exhibits a mixed Th1/Th2 response,[20] rendering it immunologically unstable and prone to shifting toward either TT or LL form.

Psoriasis is a chronic inflammatory disease characterized by an enhanced innate and adaptive immune response, including Th1 and Th17 responses, as well as elevated levels of tumor necrosis factor (TNF)- α and IL-17.[1,21] These immune mechanisms contribute to controlling the invasion and proliferation of *M. leprae*, suggesting that the hyperactive immune state observed in psoriasis may provide defense against leprosy.[21] Consistently, psoriasis patients have been shown to express higher levels of Th17-related markers, indicating a reduced susceptibility to the LL form. Interestingly, TT exhibits elevated IL-36 expression and Th17 activity compared to LL, suggesting a partial immunological overlap with psoriasis.[13] However, despite the shared immunological pathways, especially involving Th1 and Th17 responses, the clinical manifestations of these two diseases remain significantly distinct. Nevertheless, the interaction between psoriasis and leprosy remains a complex and poorly understood phenomenon. One hypothesis proposes that the dominant immune response in psoriasis, particularly the heightened Th17 activity and increased production of pro-inflammatory cytokines, may inhibit the development of lepromatous pole of leprosy, which is associated with a Th2-skewed and immunosuppressive profile.[13,22] Conversely, in tuberculoid leprosy, the strong Th1-mediated response might synergize with psoriasis-related immune pathways, creating a more resilient immune state against *M. leprae* infection.[22] Although it has not been explored, the immune system interaction between psoriasis and mid-borderline leprosy may involve both TT and LL forms.

Another possible explanation for the rare coexistence of leprosy and psoriasis lies in their distinct yet overlapping genetic factors. Genetic susceptibility plays a major role in both diseases, although the associated alleles often exert opposing effects.[8,22] Psoriasis is strongly linked to human leukocyte antigen (HLA)-Cw*06, which is found in more than 50% of psoriasis patients and is associated with increased resistance to bacterial infections, including mycobacterial infections like leprosy.[8,13,22] The inverse relationship between HLA-Cw*06 and leprosy suggests that the genetic predisposition favoring psoriasis may confer a protective effect against leprosy.[22] Intriguingly, certain HLA variants also exhibit dual, antagonistic roles. For instance, HLA-DRB1*04 has been shown to increase susceptibility to psoriasis while offering protection against leprosy.[8,22] This highlights the complexity of the immunogenetic landscape shared by these conditions. Furthermore, Lu et al[21] conducted a comprehensive gene expression analysis to explore the molecular mechanisms underlying the mutual exclusion between psoriasis and leprosy. Their study identified several genes which exhibit mutually exclusive expression patterns in psoriasis and leprosy.

Additionally, the role of neuropeptides provides another plausible explanation for the rare coexistence of leprosy and psoriasis.[8,23] Neuropeptides such as substance P play crucial roles in the inflammatory and proliferative processes driving psoriatic lesion development.[7,23] In leprosy, nerve damage caused by *M. leprae* leads to a significant reduction in these neuropeptides, potentially suppressing inflammatory responses and thereby creating an environment that inhibits the formation of psoriatic lesions. This mechanism may further explain the rarity of coexistence of these two diseases.[8,23] In this case report, the patient had BB-type leprosy coexisting with GPP, an unusual phenomenon whose underlying mechanisms remain incompletely understood. Further research is needed to elucidate the potential immunological and neuropeptide interactions contributing to this coexistence.

Only a limited number of cases documenting the coexistence of leprosy and psoriasis have been reported in the literature. Bhobe et al[7] reported a case of BL with type 1 leprosy reaction coexisting with psoriasis vulgaris, presenting with erythematous scaly plaques on the extensor surfaces and hypopigmented patches corresponding to leprosy lesions. Similarly, Li et al[24] reported a patient with BL and plaque psoriasis, in which *M. leprae* bacilli were detected in both psoriatic and normal-appearing skin. Furthermore, Sheikh et al[25] reported a case of long-standing psoriasis subsequently diagnosed with both LL and BL, confirmed through nerve and skin biopsies. Despite these reports, the precise pathogenesis underlying the coexistence of psoriasis and leprosy remains poorly understood, highlighting the complex interplay between their immunological pathways. Notably, to the best of our knowledge, no previous cases of leprosy coexisting with GPP have been reported, as observed in our case.

Leprosy reactions are acute inflammatory episodes triggered by immune responses to *M. leprae* antigens,[26,27] which are classified into two types: type 1 leprosy reaction (reversal reaction), a type IV hypersensitivity reaction,

and type 2 reaction (erythema nodosum leprosum/ENL), a type III hypersensitivity reaction.[26] The borderline type of leprosy increases the risk of developing reversal reaction,[27,28] which presents with burning sensations and rapid color changes in skin lesions, leading to erythematous, swollen, and tender plaques.[1,26] A reversal reaction involving facial skin lesions is considered one of the criteria for severe reversal reactions.[26] Risk factors for reversal reactions include focal infections and psychological stress.[29] In this case, the patient was diagnosed with a severe reversal reaction due to the presence of increasingly erythematous and raised facial lesions, which were triggered by sleep deprivation.

Histopathological examination plays a crucial role in supporting the diagnosis of GPP, particularly in cases with atypical clinical presentation.[9] Similarly, in leprosy, histopathology serves as a valuable tool for identifying cases with atypical or unclear clinical features,[12] although it is not the gold standard for diagnosis.[30] In BB-type leprosy, histopathological findings typically include the formation of a granuloma composed primarily of epithelioid cells and macrophages, with lymphocytes scattered around the granuloma.[12] In GPP, extensive neutrophilic infiltration within the epidermis is observed, along with characteristic findings such as Kogoj's spongiform pustules, papillary edema, spongiosis, and parakeratosis.[9,19] While pustular leprosy reactions also exhibit a mixed inflammatory infiltrate, the neutrophilic infiltration is less extensive, localized to the dermis, and associated with granulomatous formation and AFB, distinguishing it from GPP.[27] In this case, although histopathological findings from the punched-out lesion was suggestive of BT-type leprosy, the presence of punched-out lesions and a BI of 1+ supported a clinical diagnosis of mid-borderline leprosy. Therefore, the patient was diagnosed with mid-borderline leprosy and GPP.

Multidrug therapy, introduced by the WHO in 1981, remains the standard treatment for leprosy. It consists of rifampicin, dapsone, and clofazimine for 6 months in PB and 12 months in MB leprosy.[2,31,32] During the reaction state, MDT remains essential. The primary goal of treating reversal reaction is to restore normal nerve function and prevent further damage from inflammation.[27] The WHO recommends an initial dose of prednisone ranging from 0.5 to 1.0 mg per kilogram of body weight per day, equivalent to 30–40 mg daily for most adults. The dosage is then gradually tapered by 5–10 mg every two weeks.[26] For GPP, systemic therapy such as acitretin (first line) or cyclosporine (second line) is used to control lesions and inflammation.[33] Methotrexate (MTX) serves as an alternative; however, its use is limited by a slow onset of action and potential adverse effects.[19,33] Systemic corticosteroids are reserved for cases where other treatments are contraindicated or unavailable, with reported doses of methylprednisolone ranging from 5 to 160 mg/day,[34] then gradual tapering to prevent recurrence.[33,35] In this case report, MTX was contraindicated due to suspected anemia, acitretin was unavailable, and cyclosporine was avoided due to its immunosuppressive effects, which could exacerbate leprosy. Consequently, systemic corticosteroids were used to manage both GPP and the leprosy reaction, with methylprednisolone administered at 125 mg/day (equivalent to prednisolone 2 mg/kg/day) to achieve clinical improvement, followed by gradual tapering to prevent relapse. The dose of corticosteroids for the treatment of reversal reactions in this patient corresponds to the dose for the treatment of PPG. This case report has several limitations, including the absence of additional immunological or molecular investigations, such as cytokine profiling or genetic analysis, to further elucidate the pathophysiological relationship between generalized pustular psoriasis and multibacillary leprosy.

Understanding the molecular and immunopathological relationship between leprosy and psoriasis is essential for identifying the underlying mechanisms and potential therapeutic targets. Further research, including molecular data and clinical trials, is needed to elucidate the immunological interplay between psoriasis and leprosy.

In conclusion, the coexistence of mid-borderline leprosy and GPP is extremely uncommon and poses significant diagnostic challenges. This case underscores the necessity of diagnosing rare coexisting disorders of leprosy and autoimmune diseases, as well as the need for a complete diagnostic approach and prompt management for obtaining favourable outcomes.

Abbreviations

AFB, Acid-fast bacilli; BB, Borderline-borderline (mid-borderline) leprosy; BI, Bacterial index; BL, Borderline lepromatous; BT, Borderline tuberculoid; ENL, Erythema nodosum leprosum; GPP, Generalized pustular psoriasis; HLA, Human leukocyte antigen; IL, Interleukin; IL-36Ra, Interleukin-36 receptor antagonist; LL, Lepromatous leprosy; MB,

Multibacillary; MDT, Multidrug therapy; MI, Morphological index; MTX, Methotrexate; PB, Paucibacillary; Th, T helper; TNF-α, Tumor necrosis factor alpha; TT, Tuberculoid; WHO, World Health Organization.

Ethics Statement

The ethical approval for this case report was obtained from the Research Ethics Committee of Dr. Hasan Sadikin General Hospital Bandung with the registry number DP.04.03/D.XIV.6.5/407/2024. The patient provided informed consent to participate in the study, in accordance with the Declaration of Helsinki.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and the accompanying images. Approval has been obtained from Dr. Hasan Sadikin General Hospital to publish the case details.

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Disclosure

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