Case Report

Bullous Pemphigoid in a 53-Year-Old Man with Rapid Response to Corticosteroid Therapy: A Case Report

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ABSTRACT

Bullous pemphigoid (BP) is an autoimmune disease marked by tense subepidermal bullae. The incidence of BP is very low and remains rarely reported. In Indonesia, there are no published data regarding the incidence or prevalence of BP. We report a 53-year-old male patient who presented with fluid-filled blisters involving almost the entire body, accompanied by pruritus for one month. Several blisters had ruptured, causing pain and a burning sensation. Dermatological examination revealed generalized multiple tense bullae filled with clear fluid, some of which were flaccid, arising on partially erythematous and partially normal skin. Most lesions had ruptured, resulting in erythematous macules, hyperpigmented and hypopigmented macules, and multiple erosions. Histopathological examination supported the diagnosis by demonstrating subepidermal bullae formation. The patient was treated with a combination of topical and oral corticosteroids and showed rapid clinical improvement within two months. Bullous pemphigoid has a good prognosis when treated adequately.

Key Words: Autoimmune, Bullous Pemphigoid, Corticosteroid

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INTRODUCTION

Bullous pemphigoid (BP) is an autoimmune disease characterized by tense subepidermal bullae, primarily impacting the elderly population. The incidence of BP is low and remains rarely reported in the global. To date, there are no published data regarding the incidence or prevalence of BP in Indonesia. BP is caused by autoantibodies targeting hemidesmosomal antigens, resulting in structural damage at the supepidermal.[1–6] The diagnosis of BP is established based on clinical features, histopathological findings, and immunofluorescence examination.[4,7] Corticosteroids remain to be the primary option of BP treatment; however, their duration and dosage should be carefully limited due to potential adverse effects.[1,3–5] We report a case of bullous pemphigoid in a 53-year-old male patient who treated with a combination of topical and oral corticosteroids and showed rapid clinical improvement within two months.

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CASE REPORT

A 53-year-old man came to the Dermatology and Venereology Outpatient Clinic of dr. Chasbullah Abdulmadjid Regional General Hospital, Bekasi, with complaints of fluid-filled blisters affecting almost the entire body, accompanied by pruritus for one month. Two months ago, the patient initially experienced pruritus in the thigh region. One week later, pruritic blisters appeared at the same site and over time spread to almost the entire body, excluding the oral mucosa and genital region. Several blisters had ruptured, causing pain and a burning sensation. The patient denied any history of medication use, systemic diseases, or family history of the disease. He worked as a car washer with daily outdoor activities and prolonged exposure to sunlight.

General and physical examination were within normal limit. Patient's body weight was 53 kg. Dermatological examination revealed generalized skin lesions in the form of multiple discrete bullae containing clear fluid, with tense walls, some of which had become flaccid. The bases of the bullae were partially erythematous and partially normal skin. Lesions varied in size from lenticular to nummular. several bullae had ruptured, resulting in multiple erosions and erythematous macules, as well as hyperpigmented and hypopigmented macules with well-demarcated borders, the size is from 0.5×0.5cm to 10×5cm. Nikolsky and Asboe-Hansen signs were negative.

A skin biopsy was performed, and histopathological examination revealed hyperkeratotic epidermis with

subepidermal bullae formation containing eosinophils, neutrophils, and erythrocytes. The dermis showed fibrotic changes accompanied by chronic perivascular inflammatory cell infiltration.

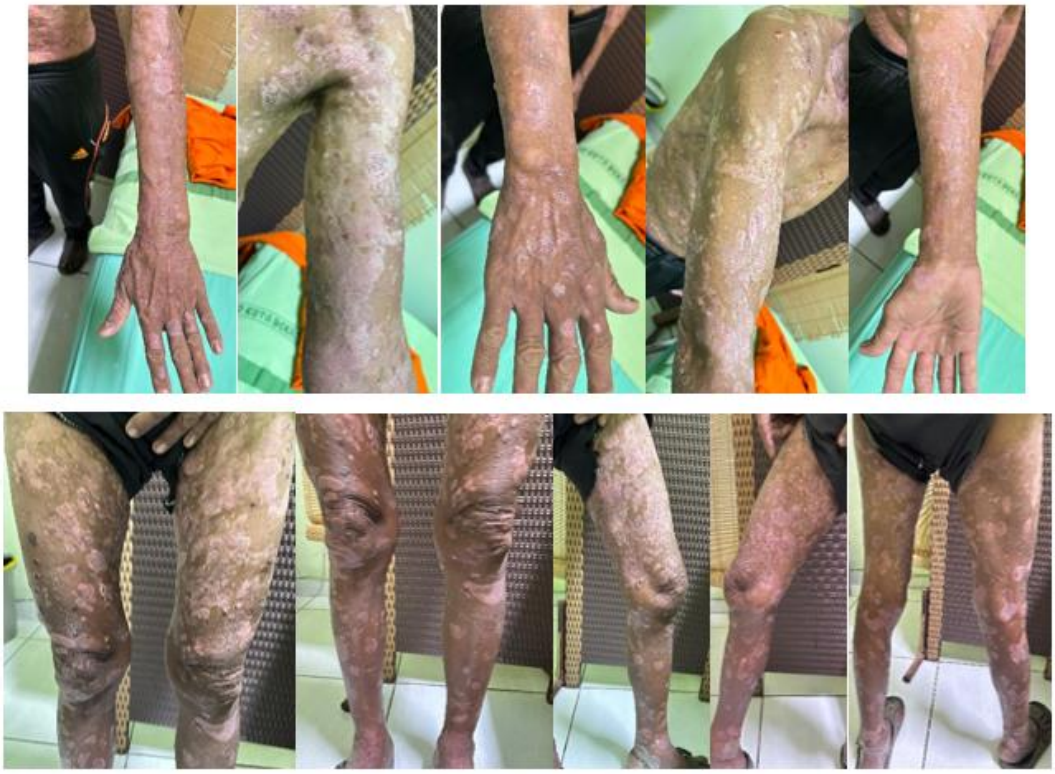


Figure No.1: Clinical presentation of the patient at the initial visit

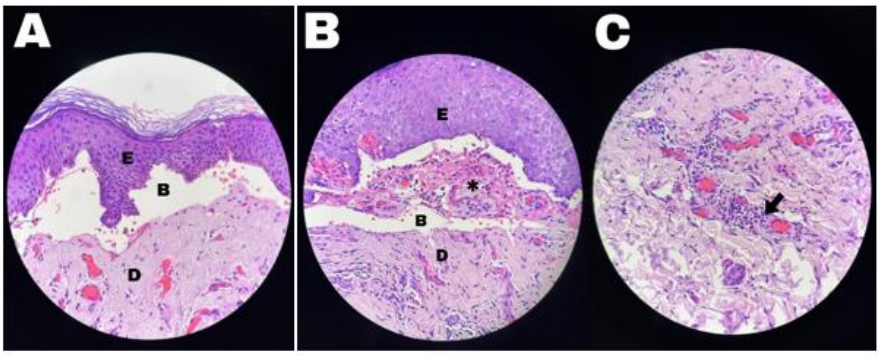
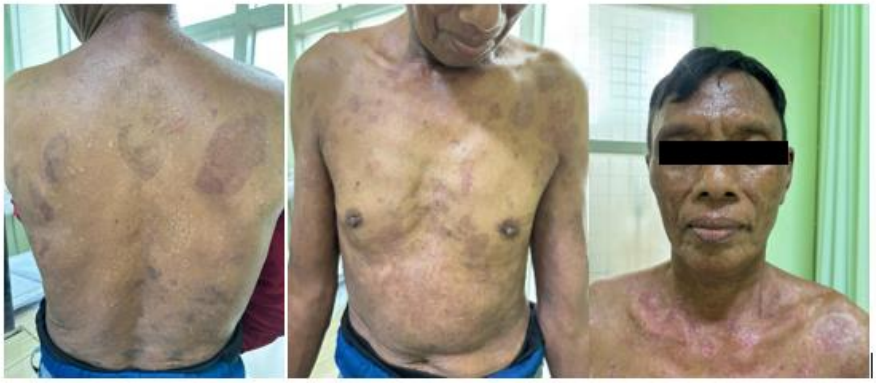


Figure No. 2: Histopathological examination. (A) dan (B) subepidermal bullae, (C) inflammatory cells infiltration E: epidermis, B: subepidermal bullae, D: dermis, *: inflammatory cells



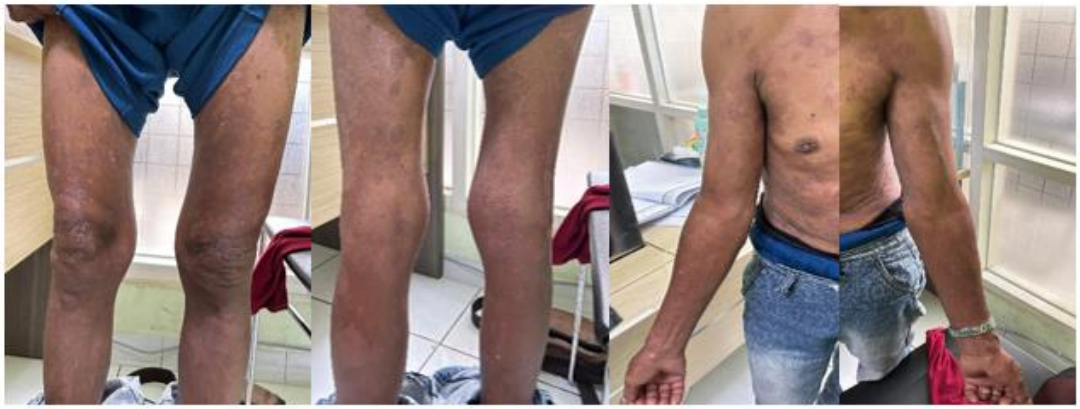


Figure No. 3: Clinical presentation of the patient after two months of therapy shows clinical improvement with no new bullae formation and post-bullous hyperpigmented and hypopigmented macules without scarring

At the initial visit, the patient was treated with oral methylprednisolone 32 mg daily for seven days. Clinical improvement was observed, there is no new bullae formation, and the methylprednisolone dose was subsequently tapered by 4 mg weekly. The treatment was combined with topical clobetasol propionate 0.05% cream applied twice daily, mupirocin cream for erosions, and cetirizine 10 mg once daily to reduce pruritus. After two months of therapy, the patient's condition stabilized with no new bullae, and all medications were discontinued.

DISCUSSION

Bullous pemphigoid often begins with a prodromal phase characterized by pruritus without bullae formation, which may last for days to months. The classic presentation of BP is tense, pruritic subepidermal bullae containing clear fluid. When bullae rupture, extensive erosions may develop but typically heal spontaneously without scarring. BP most commonly affects the flexural region, the lower abdomen, and the thighs, although other body regions may also be involved. Mucosal involvement is rare, but have a high mortality and morbidity rate.[1–3,8]

The incidence of BP escalates with age, exhibiting a significantly elevated risk in elderly populations.[5] It is thought to associated with immune system dysregulation called immunosenescence and age-associated changes in skin barrier integrity. Ultraviolet (UV) radiation, medications, and certain systemic diseases have also been implicated as potential triggering factors for BP. UV exposure may alter antigenic structures in the basement membrane zone, inducing autoantibody formation.[9,10] In this case, the patient was younger than the typical age group affected by BP. No identifiable triggering factors were found other than prolonged sunlight exposure, which was suspected to play a role as a precipitating factor.

Histopathological examination for the diagnosis of BP should be obtained from a new and intact small bullae, revealing subepidermal bullae with superficial dermal infiltration consisting of eosinophils, neutrophils, lymphocytes, monocytes, and macrophages.[1,2,4] In this patient, classic clinical features of BP were observed, and histopathological findings supported the diagnosis. Direct immunofluorescence examination was not performed due to limited facility availability.

Treatment of BP depends on the extent of skin involvement and the presence of comorbidities. Localized BP can often be successfully managed with high-potency topical corticosteroids alone, whereas extensive or generalized disease requires systemic corticosteroids such as prednisone. The recommended initial dose of prednisone is 0.75–1 mg/kg/day. Once clinical improvement is attained, gradually tapering off the prednisone dose by approximately 5 mg per week is recommended to minimize adverse effects associated with long-term high-dose steroid use. Immunosuppressive agents may also be considered as steroid-sparing therapies.[1,3–5] In this case, the patient received combination therapy with topical and systemic corticosteroids at the lowest dose and achieved rapid clinical improvement within two months without recurrence.

CONCLUSION

Bullous pemphigoid is a relatively rare autoimmune disease associated with high morbidity and mortality rate. However, when promptly and accurately diagnosed and treated with appropriate therapy, it can have a favorable prognosis.

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