CASE REPORT

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Bullous pemphigoid in 65 years old female: a case report

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ABSTRACT

Introduction: Bullous pemphigoid is an autoimmune disorder. The incidence of bullous pemphigoid has increased over time, current understanding regarding treatment and complication is an important issue considering the disease often occur in elderly resulting in high rates of morbidity to the patients.

Objective: Aim of the current case report is to describe the clinical relevance regarding symptom and treatment of bullous pemphigoid.

Case report: A 65 years old female patient, came with chief complaints of bullae in the abdominal region with itching and burning sensation in the ruptured bullae. Over time bullae spread in the lower and upper extremity. The patient was admitted for four days with therapy intravenous steroids, an oral antihistamine, and potent topical steroids. The patient was discharged from hospital in good condition.

Conclusion: Bullous pemphigoid is an inflammatory autoimmune skin disease and usually result in good prognosis with adequate management.

Keywords: Bullous pemphigoid, autoimmune, case report

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INTRODUCTION

Bullous pemphigoid (BP) is an autoimmune disease that causes the formation of tense bullae, commonly found in elderly.[1] This disease is caused by the presence of autoantibodies against structural proteins of the hemidesmosome and the presence of bullae in the subepidermal. The incidence of BP has increased over time in the regions of western Europe and North America.[2-4] The highest incidence was reported in the United Kingdom, with an incidence of 42.8 cases per 1 million population per year. BP generally occurs between 60-90 year age group.[3-5] The risk of experiencing BP increases to 300x when a person is over 90 years of age compared to person under the age of 60 years.[6] The current study reported a case of BP in 65 years old female.

CASE REPORT

A female patient, 65 years old, Indonesian citizen, Balinese, came to Dermatology and Venereology outpatient clinic of Klungkung General Hospital with chief complaints of bullae containing water, bullae were found in the abdomen region initially but over time spread to the upper and lower extremity. This complaint has been felt since one day before admission. The patient said that those bullae appear suddenly after the patient wakes up from sleep. This bulla is felt itchy, and there are also ruptured bullae that are felt very painful with a burning sensation. A history of fever before bullae formation was denied. History of medication use before admission also denied.

Patients said never experienced such a condition before. Patients deny having diabetes mellitus, hypertension, and other chronic diseases. Patients have no history of allergies, family history of atopic such as asthma, allergic dermatitis, and allergic rhinitis also denied by the patient. General exams and physical examination were within normal limit.

Most of the bullae are presented in the right arm, right shoulder, and right leg. Right shoulder lesion efflorescence is erythema, papules with largely rounded bullae, tense, containing clear colored fluid, well-defined margin, with size 3x4 cm (Figure 1). However, lesions in the right-hand region are the condition of bullae that have been aspirated (Figure 2). The right leg region (the dried bulla) with a brownish solitary crust, 2x2 cm in size, and well-defined border (Figure 3). Total skin lesion approximately 6% of body surface area.

The patient was diagnosed with bullous pemphigoid, the patient was admitted to the hospital for 4 days and treated with ringer lactate infusion, dexamethasone injection 5 mg every 12 hours, tetracycline 500 mg tablet every 6 hours

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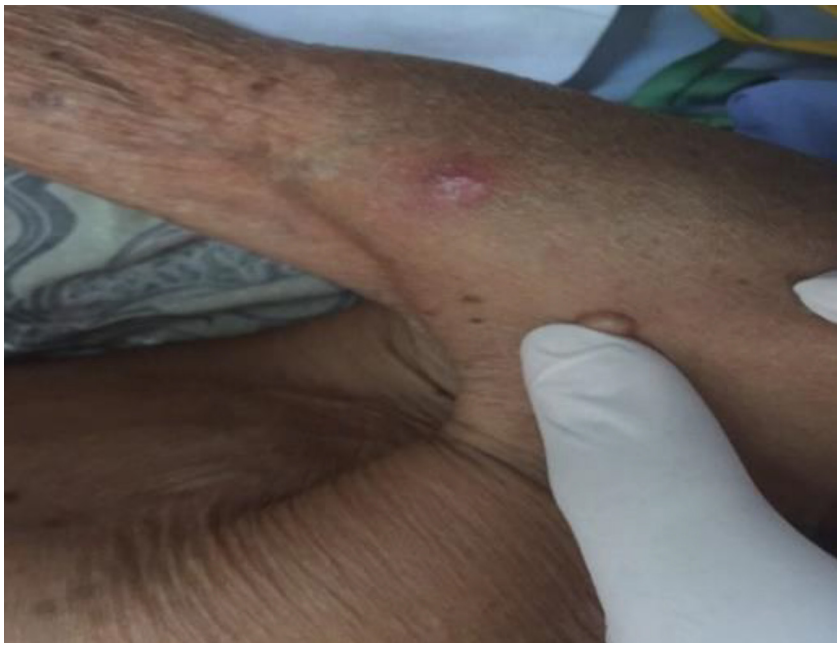

Figure 1 Tense bullae in the right shoulder region

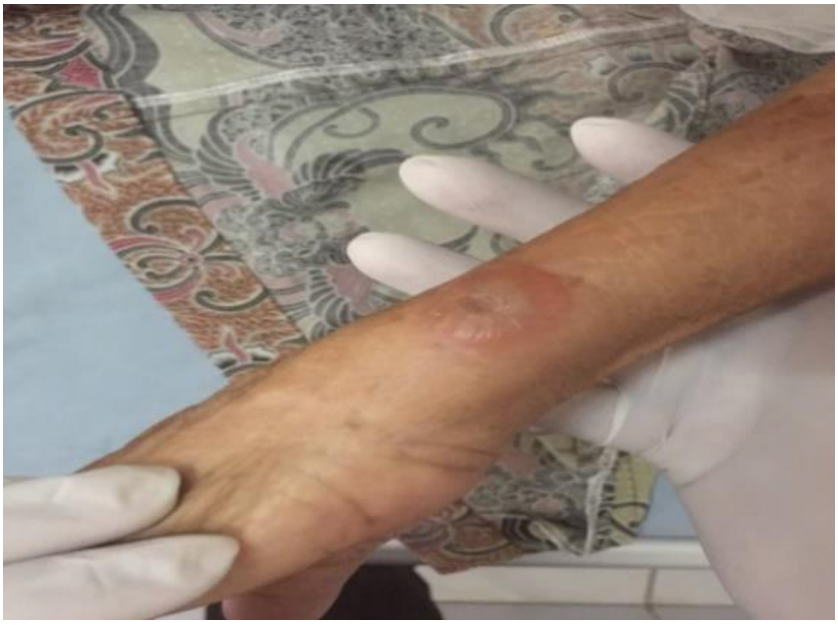

Figure 2 Aspirated bullae in the right arm region

for prevention of secondary infection in ruptured bullae, desoximetasone cream every 12 hours for topical treatment, and chlorpheniramine maleate tablet every 8 hours for itchiness medication. After four days of admission, the patient was discharged from hospital in good condition.

DISCUSSION

Bullous pemphigoid is a chronic autoimmune disease characterized by the presence of subepidermal bullae in the skin.[2,6] This disease usually affects older people over 60 years of age with bullous and less often involving the mucosa but has a high morbidity rate. There is no predilection for ethnicity, race, or sex that tends to develop BP disease. In BP, the immune system produces antibodies to the basal membrane of the skin, a thin layer of fiber tissue connects the outer layer of the skin (dermis) and the next layer of skin (epidermis).[7] These antibodies trigger inflammatory activity, which causes damage to the skin structure resulting in a subepidermal cleft that can fulfill with serous liquid to form bullae. A minority of cases may be triggered by drugs such as furosemide, sulphasalazine, penicillamine, and captopril.[8] A case study states that antipsychotic drugs and aldosterone antagonists are included in the precipitating factors of BP.[4]

The protein structure of the epidermal basement membrane that functions as the primary autoantigen in subepidermal bullous autoimmune skin disease. The main autoantigens in BP patients are BP 230 antigen (BP 230) and BP 180 antigen. BP autoantibodies accumulate in tissues and bind antigens to the basement membrane. Patients with BP experience autoreactive T cell responses to BP 180 and BP 230 antigens, and this may be important to stimulate lymphocyte B cells to produce pathogenic autoantibodies. After binding of the autoantibodies to the target antigen, subepidermal bullae formation occurs through a series of events involving complement activation, recruitment of inflammatory cells (especially neutrophils and eosinophils), and the release of various chemokines and proteases, such as matrix metalloproteinases-9 (MMP-9) and neutrophil elastase.[8-10] In this case, the fluid examination and epidermal membrane examination was not performed due to low facility and cost to perform the examination. Consideration for the diagnosis was based on the clinical manifestation.

Treatment of BP consists of immune suppressant agents because of the underlying mechanism of the disease involving reactivity of immune response throughout BP antigens. Treatment of BP is very dependent on the extent of disease such as local bullae which can often be treated successfully only with topical corticosteroids. Wider diseases are usually treated with oral prednisone. Potential topical steroids, such as 0.05% clobetasol propionate, are used twice a day, are also effective in moderate and severe bullous pemphigoid and may be safer than prednisone. In elderly patients, complications of systemic glucocorticoid therapy (such as osteoporosis, diabetes, and immunosuppression) may be very severe. Therefore, it is important to try

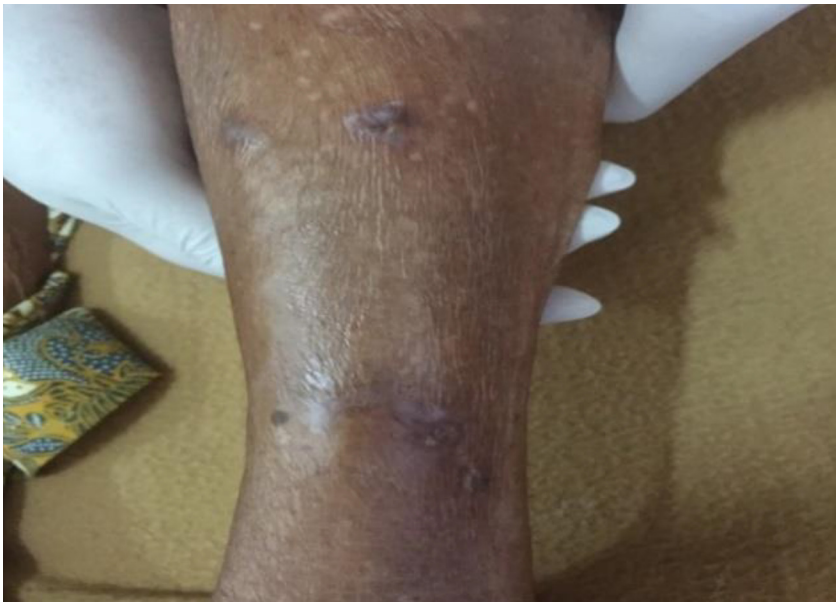

Figure 3 Ruptured bullae turn into crust

to minimize the total dose and duration of therapy with oral glucocorticoids.[2] Recommended dose of prednisone is 0.75-1.0 mg/kg/day with a tapering dose after three days of oral administration to control this disease. Administration of immunosuppressive agents such as azathioprine and methotrexate (and less often cyclophosphamide) is often used together as a potential steroid-sparing effect of prednisone.[2]

CONCLUSION

A 65 years old female patient diagnosed with bullous pemphigoid and treated with dexamethasone injection, prevention for secondary infection with tetracycline tablet, desoximetasone cream, and chlorpheniramine maleate for itchiness give the good outcome in the low facility hospital.

CONFLICT OF INTERESTS

There is no personal or institutional interest regarding this article.

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None.

AUTHOR CONTRIBUTION

All of authors are equally contributed to the study.

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