Supplementary Online Content

Kridin K, Bergman R. Association of Bullous Pemphigoid With Dipeptidyl-Peptidase 4 Inhibitors in Patients With Diabetes: Estimating the Risk of the New Agents and Characterizing the Patients. *JAMA Dermatol*. Published online August 8, 2018. doi:10.1001/jamadermatol.2018.2352

eAppendix. Inclusion criteria and therapy strategy

eReferences.

This supplementary material has been provided by the authors to give readers additional information about their work

eAppendix. Inclusion criteria and therapy strategy

BP was diagnosed as a blistering disease of the skin with suggestive clinical features[1,2] with typical histopathology of BP, including a subepidermal blister with inflammatory-cell infiltrate in the superficial dermis, often containing eosinophils, alongside at least one of the following immunopathological features: (1) linear deposits of IgG and/or C3 along the basement membrane zone by direct immunofluorescence (IF); (2) circulating IgG autoantibodies binding to the basement membrane as demonstrated by the use of monkey esophagus and a standard indirect (IF) technique; (3) the presence of circulating IgG antibodies against the immunodominant domain of BP180 (NC-16A) using enzyme-linked immunosorbent assay (ELISA)[3]. Direct IF microscopy was performed after the perilesional biopsy was incubated with 1 M NaCl solution in cases that differentiation between BP and anti-laminin 332 mucous membrane pemphigoid (MMP), anti-p200 pemphigoid, or epidermolysis bullosa acquisita (EBA) was required (in BP, immunoreactant depositions are detected along the roof of the artificial split). Type IV collagen immunostaining on paraffin sections was performed in cases where EBA was clinically suspected. Cases with predominant mucosal involvement were defined as MMP and excluded from the current study[4].

The mainstay of therapy for BP patients in our center is an initial dosage of 0.75-1.0 mg/kg/day oral prednisone. Adjuvants such as doxycycline, azathioprine, mycophenolate mofetil, or methotrexate were added when prednisone monotherapy failed to control the disease or in cases with relapse. Topical potent corticosteroids were reserved for patients with mild localized disease. When in complete remission, all treatments were gradually tapered and withdrawn. Treatment was resumed when relapse occurred and then it was usually continued indefinitely at the lowest possible dose.

The diagnosis of diabetes mellitus was defined according to the acceptable criteria defined by the American Diabetes Association[5].

eReferences

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5. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care*. 2011;34(Supplement_1):S62-S69. doi:10.2337/dc11-S062.