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Commentary

An Overview on Bullous Pemphigoid

Elena Gilbert*

Department of Pharmacology, University of Kent, England, United Kingdom

ABOUT THE STUDY

Bullous pemphigoid is an autoimmune pruritic skin condition that affects persons over the age of 60 and causes blisters (bullae) in the area between the epidermal and dermal layers of the skin. Pemphigoid is a form of pemphigoid disease. With the production of anti-hemidesmosome antibodies, it is categorised as a type II hypersensitivity reaction.

Signs and symptoms

Early lesions may have a hives-like red raised rash, but they can also be dermatitic, targetoid, lichenoid, nodular, or even without a rash (essential pruritus). Tense bullae erupt gradually, most typically on the inner thighs and upper arms, but they can also affect the trunk and extremities. Any region of the surface of the skin might be affected. Only a small percentage of patients have oral lesions.

The condition can be acute, but it can also be chronic, lasting months to years with phases of aggravation and remission. Symptoms of several other skin disorders may be similar. Milia are more prevalent in epidermolysis bullosa acquisita because the antigenic targets are deeper. Linear IgA illness can be identified by a more ring-like structure with a central depression or centrally collapsed bullae. In contrast to pemphigus vulgaris, where the sign is positive, Nikolsky's sign is negative.

Causes

There are no apparent triggering causes in the majority of instances of bullous pemphigoid. Exposure to UV light and radiation treatment have both been suggested as possible triggering factors. Certain medicines, such as furosemide, nonsteroidal anti-inflammatory agents, DPP-4 inhibitors, captopril, penicillamine, and antibiotics, have been linked to the onset of pemphigoid.

Pathophysiology

The development of IgG autoantibodies targeting dystonin, also known as bullous pemphigoid antigen 1, and/or type XVII collagen, also known as bullous pemphigoid antigen 2, a component of hemidesmosomes, causes the bullae to form. Neuropathy is linked to a distinct kind of dystonin. Following antibody targeting, a cascade of immunomodulators causes a varied influx of immune cells to the afflicted location, including neutrophils, lymphocytes, and eosinophils. Uncertain events cause a separation at the dermoepidermal junction, causing bullae to extend.

Diagnosis

Pruritus and/or predominant cutaneous blisters, linear IgG and/or C3c deposits (in an n- serrated pattern) by Direct Immunofluorescence Microscopy (DIF) on a skin biopsy specimen, and positive epidermal side staining by indirect immunofluorescence microscopy on human Salt-Split Skin (IIF SSS) on a serum sample are required for diagnosis. The use of routine H and E staining or ELISA testing does not improve the accuracy of the first diagnosis.

Treatment

Topical steroids, such as clobetasol and halobetasol, have been shown in certain trials to be as effective as systemic, or pill, treatment while being somewhat safer. Systemic prednisone and potent steroid-free immunosuppressant medicines, such as methotrexate, azathioprine, or mycophenolate mofetil, may be suitable in difficult-to-manage or widespread instances.

Some of these treatments might have serious side effects such kidney and liver damage, increased infection susceptibility, and bone marrow suppression. Antibiotics like tetracycline or erythromycin can also help manage the condition, especially in people who are unable to take corticosteroids.

Rituximab, an anti-CD20 monoclonal antibody, has been proven to be helpful in treating some instances of pemphigoid that had previously been considered resistant. A meta-analysis of ten randomised controlled trials published in 2010 found that oral steroids and potent topical steroids are effective treatments for moderate bullous pemphigoid, though their use may be limited by side effects, and that lower doses of topical steroids

Correspondence to: Dr. Elena Gilbert, Department of Pharmacology, University of Kent, England, United Kingdom, E-mail: Elenagrt@ed.ac.uk

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are safe and effective for the treatment of moderate bullous pemphigoid.

Even with commonly used drugs like rituximab, IgA-mediated pemphigoid might be difficult to cure.