

A Brief Description of Bullous Pemphigoid

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DESCRIPTION

Bullous pemphigoid is a rare skin condition that generally affects the people of middle age life and beyond. It causes a variety of skin findings, from itchy, hive-like welts to large, fluid-filled blisters which may get infected. It is an associate autoimmune disease, which means it is caused once the body's immune system malfunctions. The system is supposed to defend the body against bacterium, viruses, and diseases, however instead produces antibodies against healthy tissue, cells and organs. Some patients with BP produce other reaction diseases such diabetes and atrophic arthritis. Numerous different factors are reported to play a role in triggering BP. These includes medication (furosemide, penicillin's), mechanical trauma, and physical traumas (burns from radiation, sun or heat). Bullous is the medical term for a large blister (a thin-walled sac full of clear fluid). Typically the skin in BP is incredibly restless and huge, red welts and hives might seem before or throughout the formation of blisters. The blisters are widespread and frequently seem on the areas of the body that flex or move (flexural areas). Concerning 15%-20% of individuals with BP additionally develop blisters within the mouth or down the throat within the muscle system. Bullous pemphigoid and disease of the skin are the most frequent Autoimmune Bullous Diseases (AIBD) worldwide.

Treatment and causes

Both disease are related to a life threatening potential and impose a hyperbolic burden of mortality. The management of AIBD is difficult and often necessitates the administration of high-dose general corticosteroids and immunological disorder agents. Treatment of these diseases represents a great challenge in light of the COVID-19 pandemic, given issues concerning the vulnerability of pharmacologically immunological disorder patients. Bullous pemphigoid is associate autoimmune disease [1], which means it happens once the body's own immune system attacks the layer of tissue below the top layer of skin. The causes for this attack don't seem to be known, however bullous pemphigoid will generally be triggered by certain medications, as well as D-penicillamine. Bullous pemphigoid is the results of

associate attack on the basement membrane of the epidermis by immunoglobulin G +/- Immunoglobulin E (antibodies) and activated T lymphocytes (white blood cells) [2]. The target is the protein BP180 (also referred to as Type XVII collagen), or less often BP230 (a plakin). These proteins are among the NC16A domain of scleroprotein XVII. They are related to the hemidesmosomes, structures that make sure the dermal keratinocyte cells persist with the dermis to create a water-proof seal. The binding of the autoantibodies to the proteins and unleash of cytokines from the T cells result in complement activation, enlisting of neutrophils (acute inflammatory cells) and therefore the release of proteolytic enzymes. These destroy the hemidesmosomes and cause the formation of subepidermal blisters. The association of medicine diseases with bullous pemphigoid is assumed to relate to the presence of scleroprotein XVII within the central system and skin hemidesmosomes. Bullous pemphigoid can present with skin sensation, hive-like welts, and multiple blisters, referred to as bullae. These are most commonly seen on the: Arms, Legs, Abdomen, Groin, and Mouth.

The blisters might break open and become an open sore or ulceration. The skin round the blisters will seem either normal or red. The blisters are typically situated along the creases in the skin. You should seek medical attention from your healthcare supplier if you are thinking that you may have bullous pemphigoid. Treatment focuses on symptom relief and infection prevention. Tetracycline and minocycline antibiotics are very useful for mild to moderate illness. They do not act on bacteria, but they act directly on the immune system. They can be used in combination with a powerful topical steroid cream for faster relief.

Oral steroids (prednisone, prednisolone) are the best treatment for severe cases. Regular visits are required due to the need to adjust doses frequently and monitor side effects. A fairly high dose is required at first, and once the blisters stop, they slowly decrease over months or years. Dermatologists try to keep the dose as low as possible because steroids have some unwanted side effects. If this is too early the blisters will reappear [3]. Immunosuppressants (Imuran, Cellcept, Methotrexate, Cyclophosphamide, and Neoral) are often used in combination

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with oral steroids to allow low doses. Severe cases are best treated in hospitals to allow professional wound management and intravenous injection of the most effective treatment.

Bullous pemphigoid is a self-limiting condition that in most cases will eventually disappear completely and treatment can be discontinued. Treatment usually lasts for several years, but generally after a few months it is possible to reduce the dose of the drug to moderately low levels. Bullous pemphigoid also often has patterns of remission and recurrence. It remains asymptomatic for 5 to 6 years and may then suddenly redden.

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