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Perspective

Types and Pathogenesis of Pemphigus Vulgaris

Kenneth Jones*

Department of Dermatology, University of Malaga, Malaga, Spain

DESCRIPTION

Pemphigus vulgaris is an autoimmune disorder that can leads to development of blisters over the skin and also in lining of the mouth, nose, throat and genitals. The formed blisters can be easily burst and leads to development of infections by exposing the raw areas. The blisters are painful and those sores in the mouth are non-itchy and painful. The blisters are generally non-scarring until and unless any infections that are caused due to exposure of the site to the infectious organisms.

Types of pemphigus diseases

Pemphigus vulgaris: It involves in blistering on skin and mucous membrane which leads to erosion over the skin. The lesions formed by the pemphigus vulgaris are soft and can easily burst which leads to exposure of raw areas of the skin unhealed and painful. After skin blistering it can also lead to blisters in the mouth and scalp region of the patient.

Pemphigus foliaceus: It is superficial with cutaneous lesions. The mucous membranes are typically not affected. Pemphigus foliaceous usually affects the scalp, face, toros, and/or armpits. The skin lesions are usually small, scattered blisters that rapidly evolve into scaly, crusted erosions.

Drug-induced pemphigus: Drug-induced pemphigus is caused by a combination of biochemical interactions and aberrant stimulation of host B-cells producing intracellular IgG antibodies. These autoantibodies attack the desmoglein, causing the cells within the epidermis to separate, a process called acantholysis. It is most commonly caused by penicillinamide or captopril.

Fogo selvagem: It involves in characterization in the flaccid bullae and absence of mucous lesions. Antibodies released or produced against the desmosomes can be detected in the skin epidermis and in serum of the patient.

Paraneoplastic pemphigus: An autoimmune, multiorgan syndrome, associated with the neoplastic disease involved to be patient suffering from severe and critical mucosal involvement characterized by extensive, stubborn inflammation of the mucous linings.

Pathogenesis of pemphigus vulgaris

According to certain *in vitro* studies pemphigus vulgaris has been caused by pathogenic IgG autoantibodies which lead to acantholysis and bullae formation. The autoantibodies are directed towards calcium dependent intracellular adhesion proteins which are desmogleins (DSG3 and DSG1) which were responsible for anchoring keratin intermediate filaments to the cell membrane of epidermal cells. The DSG1 can be found in superficial layers of epidermis, whereas DSG3 can be predominantly found in non-keratinized epithelia of mucosal surfaces. Along with these DSG3 and DSG1 there are non-DSG autoantibodies includes the anti-E-cadherin antibodies and Keratinocyte acetylcholine receptors which were responsible intracellular signalling which causes shrinkage of cells and causes cell apoptosis.

Autoimmune mechanisms of pemphigus vulgaris: The actual mechanism of pemphigus vulgaris blister formation is not clear so various mechanisms have been proposed to explain the pathogenesis of the disease.

Steric hindrance of cell adhesion caused by autoantibodies: It states the autoantibodies binds to extracellular DSGs causes loss of intracellular adhesion and leads to pemphigus acantholysis. This mechanism has been considered to be insufficient as it could not explain the signalling that trigger keratinocyte loss.

Desmoglein compensation: This theory is explained by distribution of DSG in skin and mucosa. According to this theory the DSG3 and DSG1 gets distributed solely or both. When the autoantibodies attack these desmoglein, if both DSG3 and DSG1 are present one loss would be compensated by the other. If only one desmoglein is present it leads to pemphigus vulgaris. This mechanism has a limitation of occurrence of pemphigus vulgaris is related to suprabasal layer but as the autoantibodies are against DSG1 and DSG3, the pemphigus vulgaris should occur in entire epidermis. There are many instances, where no anti DSG antibodies and DSG1 titre are detected on patients with pemphigus vulgaris. There are certain other cadherins which could express keratinocytes that can compensate DSG1 and DSG3.

Correspondence to: Kenneth Jones, Department of Dermatology, University of Malaga, Malaga, Spain, E-mail: kennethjones36@yahoo.com

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CONCLUSION

Pemphigus vulgaris a rare autoimmune disorder that can leads to development of blisters over the skin and also in lining of the mouth, nose, throat, genitals. The blisters are painful and the sores in the mouth are non-itchy but painful. The ruptured blisters form as lesions. It affects less number of human beings and there is no appropriate etiology for the cause of pemphigus vulgaris but it leads to effect entire quality of life of the patient where the patient doesn't know the actual reason for the attack. The empirical treatment includes many drugs among them cyclophosphamide and mycophenolate mofetil are most widely used which prevents the blister formation and promotes healing of lesions.