Dermatitis herpetiformis (Duhring's disease)
Dermatitis Herpetiformis
Dermatitis herpetiform is an intensely pruritic, chronic recurrent dermatitis that has a slight male predilection. The disease is characterized by the appearance of small, intensely pruritic papules and vesicles, typically occurring on the extensor surfaces of the elbows, knees, and buttocks. Dermatitis herpetiform is often associated with coeliac disease, and an increased but rare risk of lymphoma. Dermatitis herpetiform is in association with SLE has also been reported.
Histopathology
The typical histologic features are best observed in erythematous skin adjacent to early blisters. In these zones, the epidermis is thinned, with the rete ridges losing their attachment to the dermis. Within 1 to 2 days, the blisters then become unilocular and clinically apparent. At this time, the characteristic papillary microabscesses may be observed at the blister periphery. For this reason, the inclusion of perivesicular skin in the biopsy specimen is of
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utmost value. The papillary dermis beneath the papillae may have a relatively intense inflammatory infiltrate. Apoptotic keratinocytes may be noted above the papillary microabscesses.
In 1967, Cormane described the presence of granular deposits of IgA within the dermal papillae in both lesional and normal skin. This finding has since become a diagnostic hallmark of dermatitis herpetiformis. Performing direct immunofluorescence (DIF) on biopsy specimens is essential for confirming the diagnosis. While DIF is highly sensitive, the results may be false-negative in patients with recent blistering or inflammation. In such cases, repeating the DIF on biopsies taken from clinically normal skin immediately adjacent to areas of erythema helps to rule out dermatitis herpetiformis. If DIF remains negative, a second biopsied area from an appropriately selected site is strongly suggestive that the patient does not have dermatitis herpetiformis.
Pathogenesis

Three important findings must be considered in the pathogenesis of dermatitis herpetiformis.

Circulating IgA antibodies that react against reticulin, smooth muscle endomysium, the dietary antigen gluten...
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spruelike changes on jejunal biopsy. Patients with celiac disease develop IgA autoantibodies to tissue transglutaminase.
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The IgA deposition results in activation of the complement system followed by chemotaxis of neutrophils.
Ultrastructural Study

The changes in dermatitis herpetiformis resemble those observed in the inflammatory bullae of bullous pemphigoid.
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