Dermatitis herpetiformis (Duhring's disease)
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Dermatitis Herpetiformis

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Dermatitis herpetiform is an intensely pruritic, chronic recurrent dermatitis that has a slight male predilection.
Histopathology
The typical histologic features are best observed in erythematous skin adjacent to early blisters. In these zones, the reticulated papillary dermis exhibits the characteristic central cleft between the papillary dermis and the epidermis. Within 1 to 2 days, the rete ridges lose their attachment to the dermis, and 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 utmost importance.
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utmost value. The papillary dermis beneath the papillae may have a relatively intense inflammatory infiltrate.
In 1967, Cormane described the presence of granular deposits of IgA within the dermal papillae in both lesional and non-lesional skin. This finding is indicative of dermatitis herpetiformis. Biopsies should be taken from clinically normal skin immediately adjacent to erythema, because false-negative results may occur when blistered or inflamed skin is evaluated. The presence of IgA deposits in two appropriately selected biopsy sites is a strong indication that the patient does not have dermatitis herpetiformis.
Circulating IgA antibodies that react against reticulin, smooth muscle endomysium, the dietary antigen gluten are found in the gut as substrate, IIF has been used to detect antiendomysial antibodies, which are present in 52% to 100% of patients.

Pathogenesis

Three important findings must be considered in the pathogenesis of dermatitis herpetiformis.
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spruelike changes on jejunal biopsy. Patients with celiac disease develop IgA autoantibodies to tissue transglutaminase.
The IgA deposition results in activation of the complement system followed by chemotaxis of neutrophils.
Ultrastructural Study.

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