Dermatitis herpetiformis (Duhring's disease)
Dermatitis herpetiformis (Duhring's disease) = دERMİTAİS HEPETİFORMİS (DUHRİNG'IN DİSİSESİ) = طاعون الدهون

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Dermatitis herpetiformis (Duhring's disease) is a chronic, inflammatory skin condition characterized by a pruritic, migratory papular eruption on the extensor surfaces of the extremities. It is associated with gluten sensitivity and is seen predominantly in the Caucasian population. The disease is characterized by the presence of IgA deposits in the skin, which are thought to be caused by an immune response to gluten peptides.

**Clinical Manifestations**
- Pruritic, papular eruptions on the extensor surfaces of the extremities.
- Migratory nature of the lesions, often with remissions and exacerbations.
- Frequently affects the knees, elbows, and buttocks.

**Pathology**
- Sub-epidermal vesicles and bullae formation.
- IgA deposits in the skin.
- Histologically, there is a lymphocytic infiltrate beneath the epidermis.

**Diagnostic Testing**
- Skin biopsy with direct immunofluorescence shows IgA deposits.
- Serologic tests showing increased IgA antibodies against gluten peptides.

**Treatment**
- Strict adherence to a gluten-free diet is the mainstay of therapy.
- Dapsone may be used as an alternative for patients who cannot tolerate a gluten-free diet.

**Complications**
- Increased risk of malignancy, particularly gastrointestinal lymphomas.
- Associated with celiac disease and gastrointestinal enteropathy.

**Association with Other Conditions**
- Patients with untreated celiac disease may have an increased frequency of malabsorption syndromes, such as pernicious anemia, steatorrhea, and osteoporosis.
- Associated with autoimmune conditions, such as insulin-dependent diabetes mellitus, lupus, and coeliac disease.

**Genetic Factors**
- Strong association between susceptibility genes and the development of dermatitis herpetiformis and celiac disease.

**Immunogenetic Considerations**
- IgA1 deposits in skin are of mucosal origin.
- Serum IgA may directly bind to epidermal tissue.
- Immunoglobulin A (IgA) 24-26% is known that patients with both GSE and DH have antibodies to Tgase-epidermal Tgases.

**Further Investigation**
- First-degree relatives of both DH and GSE patients are often affected.
- Genetic studies suggest a strong association between susceptibility genes and DH and GSE.

**References**
- Cormane et al.
- Meer et al.
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