Graft Versus Host Disease
GVHD occurs in situations in which donor immunocompetent T cells transferred into allogenic hosts are
infrequently, unirradiated blood products, solid organ transplants, and maternal-fetallymphocyte engraft

The disease can be divided into an acute and a chronic phase. Acute GVHD typically occurs between 7
In the **acute phase**, the classic triad includes skin lesions, hepatic dysfunction...
In the chronic phase, an early lichenoid stage and a late sclerodermoid stage can be distinguished. Each stage can occur without the other. Symptoms may include an erythematous-like eruption, cicatricial alopecia, chronic ulcerations, pyogenic granuloma, and angiomatous lesions.
histopathology

the early changes in the acute phase consist of focal basal vacuolation and sparse superficial perivascular lymphocytic infiltrate with exocytosis of individual cells into the epidermis and follicular epithelium. The acute phase has been divided into four histopathologic grades. In grade 1 disease, there is focal or diffuse vacuolization of the basal keratinocytes, with some accompanied by two or more epidermal lymphocytes, a phenomenon known as satellite cell necrosis. The necrotic keratinocytes contain a pyknotic nucleus and eosinophilic cytoplasm. Grade 3 lesions are characterized by more pronounced vacuolation, focal spongiosis, lymphocytic infiltration, and dyskeratosis at all levels of epidermis. The epidermis may be severely thickened. In rare cases, basal vacuolization and dyskeratosis of the follicular epithelium may be the only changes.
In the *chronic phase*, the early lichenoid stage may still show evidence of satellite cell necrosis within the epidermis.
In the late sclerodermoid phase, the epidermis is atrophic, with the keratinocytes being small, flattened, and pyknotic. There is a dense perivascular lymphocytic infiltrate in septal hyalinization. The adnexal structures are destroyed. Subepidermal bullae were present in one reported case.

**IF Testing.** Epithelial basement membrane zone granular IgM and complement deposition is present in 39% of patients with the acute form and in 86% of patients with the chronic form of GVHD. In addition, IgM and C3 have been found in the walls of dermal vessels.

**Pathogenesis.**
Acute and chronic forms of the disease have a different pathogenesis. In acute GVHD, it is believed that preparative conditioning regimen injury and mismatch between donor and recipient MHC results in introduction of donor T cells that recognize host antigens. The greater the disparity between donor and recipient MHC, the greater the T-cell response. Both CD4+ and CD8+ T cells have been reported as being involved in the response, with CD8+ T cells representing a minority of infiltrates. B cells are not found.
The inflammatory cytokines (ILs, GM-CSF, TNF-a IFN-y) produced by activated T cells and by tissue damage during the... mechanisms by which the skin, liver, and gastrointestinal tract are targeted are not clear.

Less is understood about the pathophysiology of chronic GVHD. The role of donor T cells against the...
The necrotic keratinocytic cytoplasm is filled with numerous aggregated tonofilaments.
Differential Diagnosis.
The acute phase of GVHD is similar to EM, with scattered necrotic keratinocytes and the formation of subepidermal clefts.
The eruption of lymphocyte recovery occurs predominantly in patients after receiving cytoreductive therapy (without bone marrow transplant) for acute Graft versus Host Disease. This process leads to a relatively heavy lymphocytic infiltrate with nuclear pleomorphism and hyperchromasia.
Distinguishing between the lichenoid lesions of GVHD and lichen planus is often impossible.

However, late sclerotic lesions can be differentiated from scleroderma by the marked atrophy of
the epidermis. Active synthesis of collagen takes place largely in the upper third of the dermis;

in scleroderma, collagen is synthesized mainly in the lower dermis and in the subcutaneous tissu