Acute cutaneous graft-versus-host reaction (GVHR) = اثار التغرس الجلدي الرفيع الرشيدي

**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 matemal-fetallymphocyte engraft
The disease can be divided into an acute and a chronic phase. Acute GVHD typically occurs between 7 and 21 days after transplantation and is characterized by skin, gastrointestinal, and hepatic involvement. The risk of chronic GVHD is 11 times greater if the patients had prior acute GVHD.
In the *acute phase*, the classic triad includes skin lesions, hepatic dysfunction, and diarrhea. The clinical severity is judged on the extent of these symptoms. In patients with progressive and fatal disease, cutaneous GVHD may be due to a synergistic effect from local irradiation.
In the *chronic phase*, an early lichenoid stage and a late sclerodermoid stage can be distinguished. Each stage can occur without the other. This is accompanied by a severe inflammatory process which gives rise to erosive, dry skin and can lead to complications like erythematosus-like eruption, cicatricial alopecia, chronic ulcerations, pyogenic granuloma, and angiomatous lesions.
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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 keratinocytes with individual cells 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 marked vacuolation, focal spongiosis, lymphocytic infiltration, and dyskeratosis at all levels of the epidermis. 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.
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In the late sclerodermoid phase, the epidermis is atrophic, with the keratinocytes being small, flattened, and often in a hyperkeratotic state. Septal hyalinization is observed.

**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 therapy results in a disparity of host and donor MHC, leading to the activation and proliferation of donor T cells. The greater the disparity between donor and recipient MHC, the greater the T-cell response. In identical pairs, the donor T cells are sensitized against self-antigens, which leads to a decrease in T-cell response. T cells represent 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 acute cutaneous graft-versus-host reaction (GVHR) are targeted to young rete ridge keratinocytes, follicular stem cells, and Langerhans cells. However, the exact 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 recipient's tissue is complex.
The necrotic keratinocytic cytoplasm is filled with numerous aggregated tonofilaments.
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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...
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
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