Atypical manifestations of graft-versus-host disease

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Background: Cutaneous graft-versus-host disease (GVHD) is classically described as morbilliform when acute and lichen planus–like or sclerotic when chronic. In addition to these well-known clinical forms, there are many other presentations of GVHD that are important to recognize. As the number of patients undergoing stem cell transplantation increases and the survival after transplantation improves, the prevalence of GVHD is expected to rise, and its various presentations will be increasingly encountered in clinical practice.

Objective: We sought to report unusual manifestations of skin GVHD and provide a summary of typical and atypical presentations of GVHD reported in the literature.

Methods: Patients with stem cell transplantation who developed unusual eruptions after transplantation had biopsy specimens taken to evaluate for histopathologic evidence of GVHD.

Results: Six patients presented with unusual cases of biopsy-proven GVHD, including follicular hyperkeratosis, thick-appearing white tongue, inverse pityriasis rosea–like, and eczema craquelé–like GVHD.

Limitations: This study is limited by case number.

Conclusions: Because of the high rate of cutaneous involvement with GVHD, the accessibility of the skin for diagnosis, and the morbidity associated with severe or long-standing skin involvement, it is important for dermatologists to recognize and accurately diagnose cutaneous GVHD in all its protean manifestations. (J Am Acad Dermatol 2015;72:690-5.)

Key words: eczema craquelé; follicular hyperkeratosis; graft-versus-host disease; pityriasis rosea; stem cell transplantation; white tongue.

Graft-versus-host disease (GVHD) is a common complication of allogeneic stem cell transplantation (SCT), with rates exceeding 50% depending on host and donor factors.[1] Generally, it presents either as an acute disease within days to weeks after allogeneic SCT, or as a chronic syndrome occurring months to years later. Acute GVHD (aGVHD) is typically characterized by an erythematous morbilliform eruption that may be associated with fever, diarrhea, and elevated serum liver enzymes. Chronic GVHD (cGVHD) is a more heterogeneous inflammatory and fibrotic process that may involve 1 or multiple organ systems. Skin manifestations are present in more than 90% of patients and are often the presenting sign of the disease.[2,3] The cutaneous manifestations of cGVHD are particularly protean and may be difficult to recognize even in patients with known cGVHD. Here we highlight this heterogeneity by reporting 4

Abbreviations used:

aGVHD:	acute graft-versus-host disease
cGVHD:	chronic graft-versus-host disease
GVHD:	graft-versus-host disease
SCT:	stem cell transplantation

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unusual presentations of GVHD in 6 patients, and by way of review we alert the dermatologist to common and uncommon cutaneous manifestations of GVHD.

CASES

Patient 1

A man in his 60s with a history of Jak2$^+$ myelofibrosis received an allogeneic SCT from a HLA-identical sibling with methotrexate and tacrolimus for GVHD prophylaxis. He presented 33 months after SCT with reports of a 1-year history of shiny, sclerotic skin of the back of hands and feet and pink follicular spicules on the flanks and buttocks (Fig 1, *A*). Histologic evaluation of a skin biopsy specimen from the upper aspect of the left buttock demonstrated parakeratotic follicular plugging with lymphocytic inflammation and apoptotic cells within the follicular epithelium, findings consistent with GVHD (Fig 1, *B*). Therapy with topical tacrolimus and ammonium lactate cream did not improve the follicular hyperkeratosis after 1 year.

CAPSULE SUMMARY

- Graft-versus-host disease is a common complication of stem cell transplantation, and its prevalence is rising with the increasing number of, and survival after, transplantations.
- Skin graft-versus-host disease may present in unusual ways, often mimicking other skin conditions.
- Dermatologists should have a high clinical suspicion for graft-versus-host disease in patients with stem cell transplantation who present with atypical rashes.

Patient 2

A similar eruption of dark keratotic follicular papules was noted in a woman in her 20s with a history of acute lymphoblastic leukemia 16 months after allogeneic SCT from an HLA-matched unrelated donor. Histologic examination revealed folliculotropic interface dermatitis consistent with GVHD.

Patient 3

A man in his 30s with a history of T-cell lymphoblastic leukemia was treated with an allogeneic SCT from an HLA-identical sibling and received cyclophosphamide for GVHD prophylaxis. His posttransplantation course was complicated by severe GVHD involving the skin, eyes, and liver, ultimately requiring liver transplantation. Approximately 2 years after SCT, he developed a thick white tongue with areas of ulceration on the left side of the tongue (Fig 2, *A*). Bacterial and fungal cultures, cytomegalovirus quantification, and herpes simplex virus polymerase chain reaction of the tongue were negative. Tongue biopsy specimen revealed parakeratotic, stratified squamous epithelium exhibiting sawtooth rete ridge formation and degeneration of the basal cell layer, with a dense bandlike infiltrate of lymphocytes immediately subjacent to the epithelium and scattered colloid bodies exhibiting satellitosis (Fig 2, *B*). Periodic acid–Schiff stains were negative for yeast. These histologic findings were suggestive of lichenoid tissue changes associated with GVHD. He was continued on his regimen of prednisone and tacrolimus in addition to viscous lidocaine, dexamethasone, and clotrimazole with no improvement noted after 3 months.

Patient 4

Similar features were seen in a man in his 50s with a history of biphenotypic acute leukemia after 9/10 HLA-matched unrelated donor allogeneic SCT complicated by severe and refractory sclerotic cGVHD who developed a shiny and thick-appearing porcelain white tongue such as that observed in patient 3.

Patient 5

A woman in her 50s with a history of non-Hodgkin lymphoma after autologous SCT complicated by secondary myelodysplastic syndrome was treated with an allogeneic SCT from a matched unrelated donor with tacrolimus and methotrexate GVHD prophylaxis. Approximately 17 months after allogeneic SCT, she presented with a rash that developed 2 months after tapering off all immunosuppressive medications. Symptoms of an upper respiratory tract infection accompanied the appearance of a single prominent, thin, scaly plaque in the left axilla, followed by the eruption of numerous similar but smaller lesions over the course of a few weeks. Physical examination was notable for thin, scaly, purple papules and plaques following skin tension lines on the neck, on the axillae, under the breasts, and on the abdomen/suprapubic area in a pattern consistent with inverse pityriasis rosea (Fig 3, *A* and *B*). A biopsy specimen of a lesion on the right side of her neck showed interface dermatitis with pigment incontinence, most consistent with GVHD (Fig 3, *C*). She was started on topical triamcinolone, with improvement of the rash after 1 week.

Patient 6

A man in his 50s with a history of scleroderma and myelodysplastic syndrome was treated with an allogeneic SCT from an HLA-matched sibling with

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