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LETTER TO THE EDITOR

Demodicidosis as a cause of facial eruption developing early after allogeneic hematopoietic stem cell transplantation

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Graft-versus-host disease (GVHD) is the most common and clinically important cause of skin eruption after allogeneic hematopoietic stem cell transplantation (HSCT). Other causes include viral infection such as human herpesvirus 6, allergic reactions to drugs, and the toxicity of the conditioning regimens. Despite the rarity, infectious microorganisms may also cause this complication. We report our experience as regards two patients developing skin eruption limited to the face early after allogeneic HSCT, which was diagnosed as demodicidosis.

A 53-year-old woman with acute lymphoblastic leukemia (Patient 1) and a 44-year-old woman with chronic myelogenous leukemia (Patient 2) underwent bone marrow transplantation from an unrelated donor. Tacrolimus and short-term methotrexate were used as a prophylaxis for GVHD. Their post-transplant courses were complicated with acute skin GVHD, which was successfully treated with a topical steroid. Around day 110 post-transplant, both patients developed an itchy eruption limited to the forehead, cheeks, and jaw (Fig. 1). The eruptions were characterized by multiple papules and pustules with erythema. Microscopic examination found numerous Demodex mites in the pustules (Fig. 2), and the diagnosis of demodicidosis was made. Histological examination was not performed. Eruptions promptly resolved with topical sulfur, and completely resolved within 3–4 weeks. No recurrence was observed after the cessation of the treatment in Patient 2. However, 2 weeks after the cessation of the treatment, the demodicidosis recurred in Patient 1, and the same therapy was initiated, which was again effective. An additional 1-month treatment resulted in the complete resolution of the skin eruption without further recurrence.

Demodex mites inhabit human pilosebaceous ducts and are frequently found in healthy populations. Because of the distribution of pilosebaceous ducts, Demodex are exclusively found in the human face. Demodicidosis associated with immunocompromised hosts has been reported in patients with HIV infection and lymphoid malignancies, and in a recipient of allogeneic HSCT [1–7]. Thus, although still controversial, these reports strongly suggest that immunodeficiency may significantly contribute to its pathogenicity. The common feature of these reported immunocompromised settings is long-term impairment of cell-mediated immunity. Although there has only been one reported case of demodicidosis after allogeneic HSCT, long-term impairment of cell-mediated immunity could contribute to the susceptibility of recipients of HSCT and organ transplantation to demodicidosis.

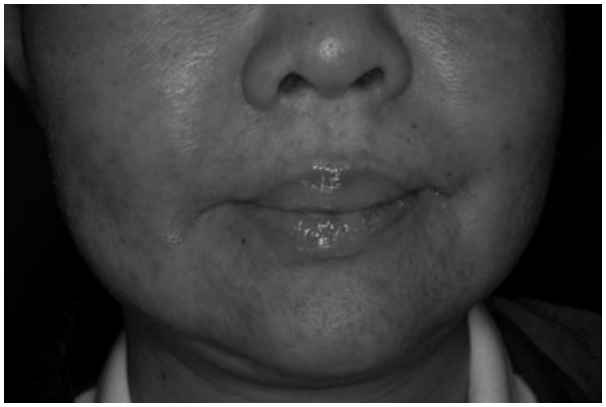

Figure 1 Skin eruptions that developed around day 110 after transplantation in Patient 1.

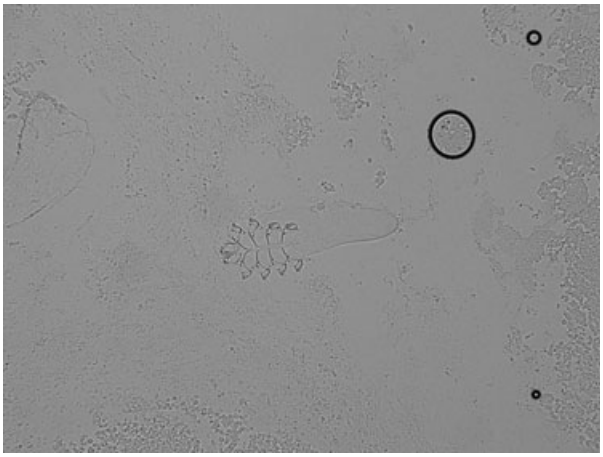

Figure 2 Demodex mites observed in the pustules in Patient 2.

Allogeneic HSCT and organ transplant recipients often develop skin eruptions because of various mechanisms, including GVHD, allergic reaction to drugs, and infection. Among the infectious causes of skin eruptions observed in HSCT recipients, examination for Demodex mites in skin samples should be included in the diagnostic measures, especially for eruptions localized on the face.

In conclusion, our experience emphasizes that transplant physicians should recognize demodicidosis as one of the etiologies of facial skin eruption after HSCT or organ transplantation. To understand its characteristics better, it is necessary to accumulate the cases of demodicidosis developing in the recipients of HSCT or organ transplantation.

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