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Persistent Facial and Chest Papular and Pustular Eruption in a Stem Cell Transplant Patient

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INTRODUCTION

Demodex folliculitis is a condition caused by inflammation of the pilosebaceous unit due to the *Demodex folliculorum* mite.¹ The mite is a normal inhabitant of the human hair follicle and though they have been found in all areas of human skin, they predominate on the face. The face, with a high density of active sebaceous glands is a desirable home for demodex mites who feed on exfoliated epidermal cells and sebaceous gland secretions. The eruption of demodex folliculitis can mimic many common skin pathologies such as rosacea, acne vulgaris, and bacterial folliculitis.² When demodex folliculitis occurs with immunocompromised patients, the clinical presentation can be atypical and severe.³ Herein, we report a case of severe demodex folliculitis and review of the literature.

CASE DESCRIPTION

The patient is a 68-y-old male with a history of myelodysplastic syndrome for which he received a matched, unrelated allogeneic hematopoietic stem cell transplant (HSCT). Before the transplant, the patient was treated with 6 cycles of azacitidine. His conditioning regimen consisted of fludarabine and melphalan as well as cyclophosphamide 1-time posttransplant. Following the transplant, he was started on mycophenolate mofetil and tacrolimus for prevention of graft-versus-host-disease (GVHD). On day 69, the patient

was transitioned to sirolimus therapy for GVHD prevention. His skin was within normal limits until day 78, with the development of an erythematous pustular rash on his upper trunk and face (Figure 1). The pustules were presumed to be bacterial folliculitis as bacterial culture demonstrated few coagulase-negative staphylococci, and he was prescribed minocycline hydrochloride 100 mg BID. While on minocycline, his rash progressed to extend further down the trunk, with worsening erythema and development of more pustules on the trunk (Figures 2 and 3). At this time, consideration of a sirolimus-induced pustular drug eruption was entertained. Sirolimus was held in hopes that the rash would resolve. While off sirolimus, on day 188, the patient was diagnosed with acute grade 1 gastrointestinal GVHD via endoscopy. Although there was clinical improvement in his rash, he was



FIGURE 1. Erythematous papulopustular rash on the face.

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FIGURE 2. Worsening rash with diffuse erythema spreading down the trunk.



FIGURE 3. Worsening rash with diffuse erythema spreading down the trunk.

started on methylprednisolone 2 mg/kg/d and intravenous tacrolimus 0.8 mg/d to treat his gastrointestinal GVHD.

However, a month later, while on a prednisone taper of 10 mg/d and tacrolimus 2 mg/d, he was admitted to his local hospital. During this admission, on day 222, he was diagnosed with cytomegalovirus colitis via esophagogastroduodenoscopy and flexible sigmoidoscopy. At this time, recurrence of the rash was noted, although the patient was no longer taking sirolimus. The rash was biopsied at the outside hospital and demonstrated findings that could be consistent with cutaneous GVHD (cGVHD), although the skin biopsy slides did not receive secondary dermatopathology review. With this leading diagnosis, the patient was treated with topical steroids.

After weeks of topical steroid use, the rash did not improve, so the patient presented back to our institution. At this time, his skin was thinned and bruised, and the rash was not clinically consistent with cGVHD. Thus, the patient was instructed to stop using topical steroids; with the persistence of pustules, he was again diagnosed with folliculitis and started on clindamycin lotion. Unfortunately, the rash continued to worsen on clindamycin. The patient was instructed to start topical desonide for presumptive contact dermatitis in reaction to the topical clindamycin, but the rash continued and extended beyond areas where the clindamycin was applied, such as the proximal upper arms and inner ear canal.

A reconsideration of the diagnosis was needed, thus prompting scrapings of pustules. The scrapings demonstrated numerous *Demodex folliculorum* mites (Figure 4), which lead to the diagnosis of demodex folliculitis. The patient was then prescribed 2 doses of oral ivermectin and topical permethrin. He noticed significant improvement just 1 wk after the second



FIGURE 4. Microscopic picture of the *Demodex folliculorum* mites from the scraping.

ivermectin dose; and 2 mo after the demodex folliculitis diagnosis, the patient had no active pustules.

DISCUSSION

Demodex mites are normal inhabitants of the human hair follicle and are predominately found in small numbers.⁴ Demodex folliculitis is caused by inflammation of the pilosebaceous gland due to overgrowth of the *Demodex folliculorum* mite. In immunocompromised patients, the mites can be

found in greater density and cause severe cutaneous eruptions. The increased severity in immunocompromised patients may be because of a combination of impaired immune defenses and aberrant immunologic responses.⁴ Although it is not rare to see severe facial eruptions from demodex folliculitis in immunocompromised patients, our case demonstrates 1 of the most diffuse eruptions noted in literature. Although it started on the face, our patient's lesions continued halfway down his trunk and onto both of his proximal arms. It is unclear as to what led to the more than usual dissemination of demodex folliculitis in our patient.

Demodex folliculitis presents as an erythematous papulopustular eruption that may mimic many other common skin pathologies. Demodex folliculitis is generally not associated with or triggered by GVHD but is often misdiagnosed as cGVHD in patients who have recently received an allogeneic HSCT as cGVHD is 1 of the most common causes of skin eruptions following an HSCT.³ This urges the importance of a biopsy, as treatment for cGVHD, often topical or systemic steroids, will not be beneficial in treating demodex folliculitis. A distinguishing clinical presentation that can aid in differentiating the 2 is the "cut off" sign. This is the clinical phenomenon in which demodex folliculitis involves the nose, cheeks, temple, and forehead and terminates at the hairline. This is most likely because of the differences in the pilosebaceous units of the scalp compared with the face and should prompt suspicion for demodex folliculitis if present.¹ This sign may be helpful when the diagnosis of a cutaneous eruption after a HSCT is still undetermined.

Treatment of demodex folliculitis is variable based on cases. Some cases report resolution with topical permethrin alone, whereas others report the need for topical sulfur or oral methotrexate.^{1,5,6} Our patient demonstrated rapid clearance of the rash 1 wk after the second dose of oral ivermectin,

which we hypothesize to be an efficient, effective treatment for severe, diffuse demodex folliculitis eruptions.

Often times, a papulopustular eruption on the face and trunk is thought to be folliculitis because of the appearance and location. Our patient had a severe facial papulopustular eruption that spread caudally, and because demodex folliculitis rarely develops on extremities or causes cutaneous eruptions this diffuse, it was not initially included as a potential diagnosis. This demonstrates a potential diagnostic pitfall for demodex folliculitis in an immunocompromised patient. The diagnosis should be highly considered in any patient presenting with a refractory papulopustular eruption and especially in those who are immunocompromised. It is imperative that dermatologists recognize demodex folliculitis and initiate prompt scraping of a pustule for diagnosis. Prompt scraping can lead to a quicker diagnosis and treatment onset, leading to faster resolution and improved treatment outcomes.

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