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CLINICAL CORRESPONDENCE

Scalp-Sparing Facial Demodicosis Mimicking Acute Cutaneous GVHD after Allogeneic HSCT: A Clinicopathologic Confirmation and Rapid Therapeutic Response

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Received: 14 October 2025 | **Revised:** 16 October 2025 | **Accepted:** 18 October 2025

To the Editor,

We report a post-allogeneic hematopoietic stem-cell transplantation (allo-HSCT) patient presenting with scalp-sparing, sharply demarcated centrofacial erythema that was ultimately diagnosed as demodectic folliculitis.

A 48-year-old woman with relapsed acute myeloid leukemia underwent allo-HSCT. Per protocol, graft-versus-host disease prophylaxis was cyclosporine-based. In the early post-transplant period, the Hematology service requested dermatologic consultation; the patient was promptly evaluated by the Dermatology Department. Clinical examination documented sharply demarcated centrofacial erythema and mild edema with a stinging/burning sensation; there was no fever, mucosal involvement, or other systemic symptoms. The scalp was spared (Figure 1a).

UV-induced fluorescence dermatoscopy (UVFD) revealed dilated follicular openings with keratotic plugs, translucent filaments protruding from follicular ostia ("Demodex tails"), perifollicular erythema, and fine whitish scaling (Figure 2a). Accordingly, UVFD accentuated follicular plugs and Demodex tails, facilitating targeted sampling for microscopy and biopsy. A KOH-prepared skin scraping obtained from lesional skin with a 1 cm^2 template and examined by light microscopy demonstrated an elevated Demodex density of 18 mites/cm^2, consistent with demodicosis (Figure 2b); although diagnostic cut-offs (e.g., ≥5 mites/cm^2) are conventionally defined for standardized skin surface biopsy rather than scraping, they remain a practical interpretive reference in this setting [1]. Given the differential diagnosis of acute cutaneous GVHD, a punch biopsy was performed to exclude aGVHD; histopathology demonstrated folliculocentric inflammation with intrafollicular mite structures, with no histologic evidence of aGVHD (Figure 2c).

The eruption resolved within one week under a regimen of single-dose oral ivermectin 200 μg/kg, daytime topical metronidazole 1%, and nightly topical permethrin 5% cream (Figure 1b).

Facial eruptions in the early period after allo-HSCT often raise concern for acute graft-versus-host disease (aGVHD); nonetheless, viral exanthems, drug eruptions, and conditioning-related toxic erythema must also be considered in the differential diagnosis [2, 3]. Demodicosis—overgrowth of Demodex mites that normally inhabit pilosebaceous units—represents an under-recognized mimic in immunocompromised hosts and has been reported after HSCT [2–5]. In our patient, the triad of dermoscopic signs, a quantitative KOH skin scraping, and biopsy showing folliculocentric inflammation with intrafollicular mites established demodectic folliculitis. Antibiotic exposure and post-transplant immunosuppression likely facilitated mite overgrowth, and targeted therapy yielded rapid remission [2–5]. Although limited by single-case design and short follow-up, the concordant clinicopathologic findings and brisk response strongly support demodicosis as the cause.

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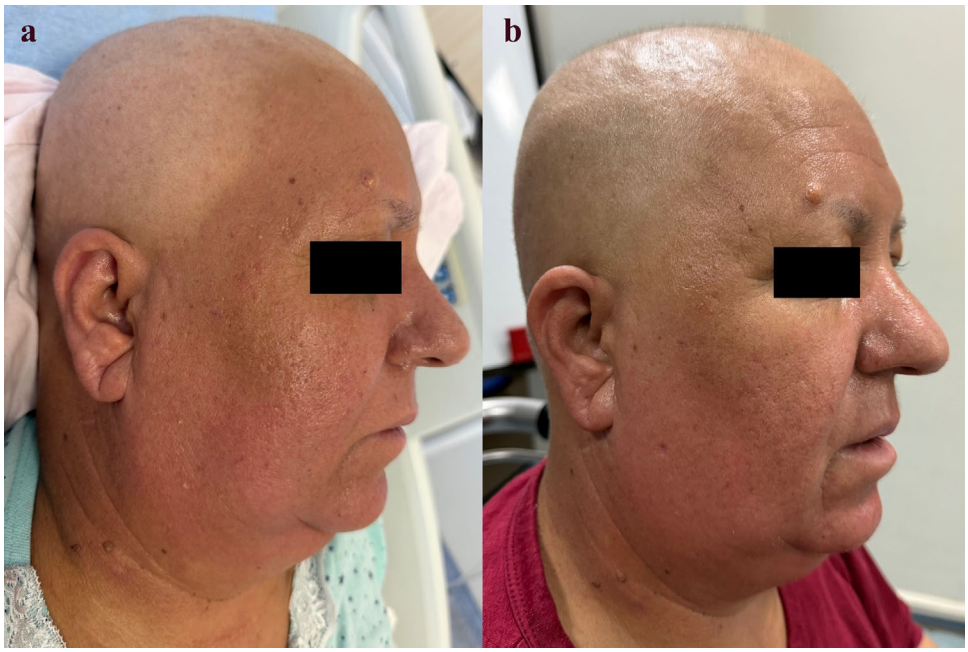


FIGURE 1 | **(a)** Pre-treatment: sharply demarcated, scalp-sparing centrofacial erythema with mild edema. **(b)** One week after therapy (single-dose oral ivermectin 200 μg/kg, daytime topical metronidazole 1%, nightly topical permethrin 5%): marked regression of erythema and swelling.

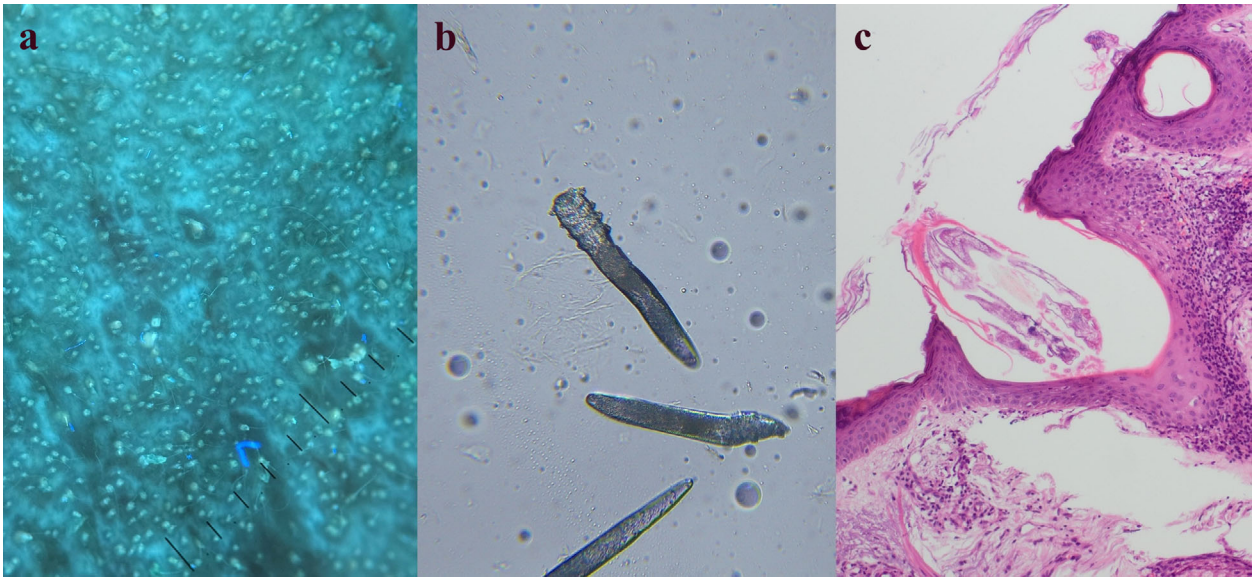


FIGURE 2 | **(a)** UV-induced fluorescence dermoscopy (UVFD): numerous dilated follicular openings with keratotic plugs and translucent protruding filaments ("*Demodex* tails") on an erythematous background. **(b)** KOH-prepared skin scraping obtained with a 1 cm^2 template showing abundant *Demodex*; quantified density 18 mites/cm^2, consistent with demodicosis. **(c)** Punch biopsy, H&E: (×20) folliculocentric lymphohistiocytic inflammation with intrafollicular *Demodex* organisms. Findings are consistent with demodectic folliculitis, with no histologic features of acute cutaneous GVHD.

A pragmatic algorithm—UVFD/dermoscopy followed by quantitative bedside mite counting—should guide evaluation, with biopsy reserved for residual uncertainty (e.g., borderline/discordant counts, atypical morphology, or to exclude aGVHD) and directed to a dermoscopically active folliculocentric lesion. Such triage minimizes misclassification as aGVHD, prevents unnecessary intensification of immunosuppression, and expedites targeted antiparasitic therapy.

Conflicts of Interest

The authors declare no conflicts of interest.

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