was less likely related to omalizumab administration for the reasons discussed below. First, the patient did not develop AA during the first course of omalizumab. Second, AA improved despite continued omalizumab, which is consistent with previous reports. Third, AA exacerbation occurred 10 months after omalizumab was discontinued. Therefore, because AA and atopic dermatitis or CSU are closely related,[5] we believe some factors of the patient's atopic background may have contributed to AA development. In our patient, trichotillomania was considered the differential diagnosis because of the negative pull test and the presence of scratching marks on the lesions. However, we concluded that AA was the more likely diagnosis because of the well-defined shape of her hair loss lesions, the absence of a history of hair pulling and psychological problems, and the presence of exclamation-mark hairs.

We should recognize that patients with allergic diseases have risk factors for AA. When AA occurs in patients using antibody drugs, it should be carefully determined whether the symptoms are due to an adverse reaction to the drug and provide accurate information to the patient.

Acknowledgment

Written informed consent was obtained from the patient's parent and the patient for the publication of this report and images.

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Conflict of interest: None.

Funding source: None.

doi: 10.1111/ijd.16823

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Morphea: an unusual case affecting lip and alveolar bone

Dear Editor,

A 24-year-old woman presented to the dental clinic with a 2-year history of receding gums on her upper front teeth. She had no significant family or medical history. Oral examination revealed a linear hypopigmented lesion on the left upper lip mucosa, which extended through the vermilion and attached gingiva of the upper left central and lateral incisors (Figure 1a). Meanwhile, the affected teeth presented labial gingival recession and root exposure with increased mobility (Figure 1b). An x-ray revealed advanced alveolar bone loss (Figure 2). A biopsy of the labial mucosa demonstrated thickening collagen in the lamina propria with lymphomonocytic infiltration, confirming the diagnosis of morphea (Figure 1c). The laboratory examinations including complete blood counts, comprehensive metabolic panel, rheumatoid factors, antinuclear antibodies, and extractable nuclear antigens showed no abnormalities. After treatment with topical tacrolimus ointment for 3 months, the induration of the lesion gradually improved with no evidence of further progression at 1 year of follow-up (Figure 1d). Considering the desire for esthetic rehabilitation of periodontal defects, the patient was referred to the Department of Periodontics for further consultation and evaluation. Periodontal surgery including bone grafts and guided bone regeneration may be considered to restore the loss of alveolar bone following stabilization of the affected lesion.

Morphea, also known as localized scleroderma, is a rare chronic connective tissue disease primarily affecting the skin, subcutaneous tissue, bones, and even the central nervous system. It is characterized by excessive collagen deposition leading to thickening and sclerosis of the skin and underlying tissues.[1] To date, the etiopathogenesis of morphea remains unclear, and it might be associated with genetic predisposition and multiple environmental triggers including trauma, radiation, infections, and certain medications.[1] Clinically, morphea presents typically as well-circumscribed plaques with signs of sclerosis and atrophy. The sclerosing process particularly affects the skin, and cases of morphea with merely oral involvement are rarely reported.[2]

The clinical presentations of oral involvement in morphea are diverse, encompassing scar-like or pale indurated plaques in oral mucosa, localized gingival recession, delayed tooth eruption, atrophic root development, and even mandibular and/or maxillary resorption.[2,3] In this case, we reported a young female patient with morphea limited to the orofacial region with gingival recession along with resorption of anterior maxilla alveolar bone. A "groove" sign presented at the site of tendons and ligaments, and

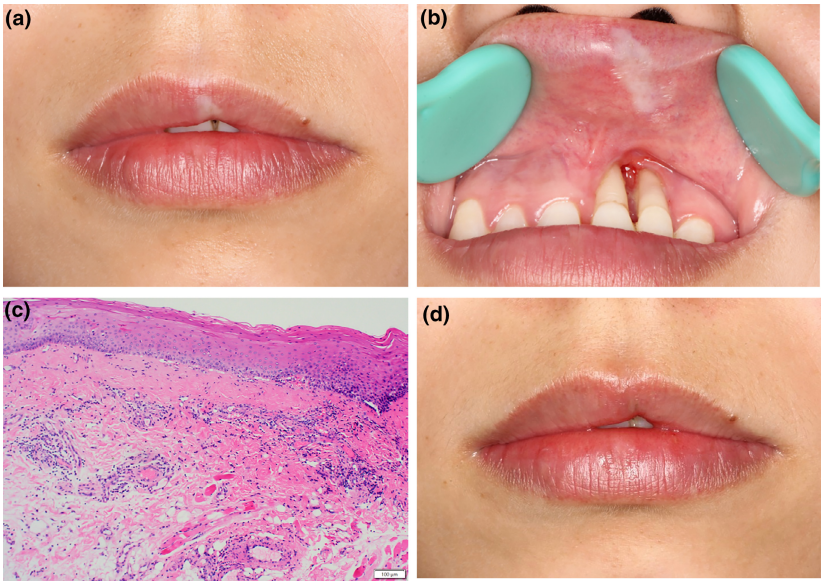

Figure 1 (a) Malformation of the upper lip, (b) labial gingival recession and root exposure, (c) histopathologic manifestations of the patient's biopsy showed epithelial thinning and flattening of the rete pegs, as well as thickening of the lamina propria collagen and the presence of perivascular lymphomonocytic infiltration in the lamina propria (hematoxylin and eosin, ×100), and (d) appearance of the upper lip after treatment with topical tacrolimus ointment

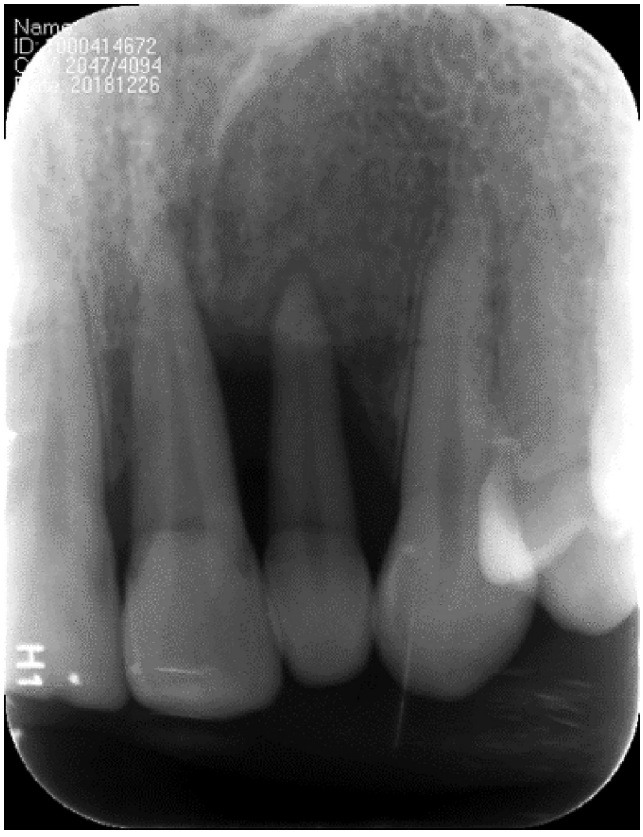

Figure 2 Localized interproximal alveolar bone resorption involving teeth

deep bone tissue loss developed in the atrophic phase. In clinical practice, differential diagnosis of morphea must be considered in addition to trauma and periodontal disease when the patient presents with severe but localized periodontal damage.

The diagnosis of morphea with oral involvement is made on clinical features and radiologic imaging with confirmatory histopathology, which is necessary to distinguish it from other entities including lichen sclerosus and scarring.[2,4] It is common for morphea patients to experience delays in diagnosis and treatment because healthcare providers fail to recognize this relatively rare disease. It was reported that 63% were given the diagnosis more than 6 months and 25.5% of patients were diagnosed more than 2 years after disease onset.[5] It was indicated that the evaluation of morphea differed depending more on the specialty of the treating physician than on disease characteristics, and solutions for this discrepancy may include narrowing the gap in knowledge and broadening awareness of morphea in more specialties.

In conclusion, we reported an unusual case with merely oral involvement of morphea. It is imperative for dermatologists and dentists to improve their ability to distinguish the broad spectrum of clinical presentations of morphea. In the treatment of morphea, strengthening the collaborative relationship between the dermatologist, dentist, and pathologist is preferable.

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Conflict of interest: None.

Funding source: None.

doi: 10.1111/ijd.16840

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Treatment of primary cutaneous blastomycosis by excision alone: a case report

Dear Editor,

Primary cutaneous blastomycosis can occur without systemic findings. Immunosuppression is a risk factor for both primary and disseminated blastomycosis. Due to polypharmacy, antifungal treatment may negatively impact a patients' health. Systemic azole therapy such as itraconazole is a common treatment for primary cutaneous blastomycosis.[1–4] Itraconazole, due to its inhibition of cytochrome P450 3A4, can cause changes in medication levels. This case has clinical significance for transplant patients on multiple immunosuppressants. It must be noted that posttransplant blastomycosis is uncommon relative to other fungal infections such as coccidioidomycosis or histoplasmosis.[5] We present the case of a patient with primary cutaneous blastomycosis who was treated without the use of antifungal agents due to concern for drug interaction.

A 67-year-old patient presented to the infectious disease clinic for the treatment of biopsy-proven cutaneous blastomycosis. They reported developing an enlarging right distal forearm papule 3 months prior to their clinic visit. They denied any fevers, chills, night sweats, respiratory symptoms, arthralgias, or other skin lesions. Past medical history was remarkable for a living donor kidney transplant performed 5 months prior to presentation. Their posttransplant medications included prednisone, tacrolimus, and mycophenolate. They reported trimming branches in the northern Mid-Atlantic United States (a known endemic blastomycosis area) and sustaining right forearm abrasions prior to the development of the papule. On physical examination, the patient presented with a red, indurated plaque on the right forearm that measured 2.2 × 2.5 cm (Figure 1). The patient's initial workup included negative fungal blood cultures and a negative *Blastomyces* serology and urine antigen. A computed tomography scan of the chest was within normal limits. Due to the lack of systemic findings and the concern for medication interactions, a localized treatment approach was discussed. Dermatology performed an excision with 5-mm surgical margins. Grocott-Gömöri methenamine silver (GMS) and periodic acid–Schiff–diastase (PAS-D) staining revealed fungal elements with occasional broad-based budding compatible with *Blastomyces* (Figure 2). Histological margins were measured from the neutrophilic and granulomatous dermal infiltrate. Based on this infiltrate, the margins were cleared by at least 1.9 mm. At 8-month follow-up, the patient remained asymptomatic with no signs of recurrence at the surgical site (Figure 1).