



Development of Demodicosis after Omalizumab Injection

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Dear Editor:

Omalizumab, humanized anti-immunoglobulin E (IgE) monoclonal antibody indicated for severe persistent allergic asthma and chronic idiopathic urticaria, has been known to have a few side effects, such as gastrointestinal helminth infections. *Demodex*, a parasite that inhabits the pilosebaceous unit, causes rosacea or folliculitis when it proliferates. We report two cases of demodicosis after omalizumab treatment.

A 47-year-old woman presented with pruritic erythema and papules, mostly on her forehead, glabella, and nose (Fig. 1A, C). The patient had recurrent idiopathic angioedema which had not been adequately controlled with antihistamines and systemic corticosteroid. Consequently, she received eight cycles of omalizumab injection for 1.5 years. This treatment was discontinued 6 months prior to the appearance of facial redness. Baseline serum total IgE was less than 100 IU/ml and deemed negative (12.8 IU/ml) after discontinuation of omalizumab.

Skin scraping test performed on five papules of her face showed 18 *Demodex* mites on microscopy. She was prescribed oral metronidazole 250 mg twice daily for 2 weeks, and symptoms improved (Fig. 1B, D).

A 32-year-old man presented with pruritic erythema and papules on his face that had been gradually worsening for a year (Fig. 2). He had been receiving omalizumab injections

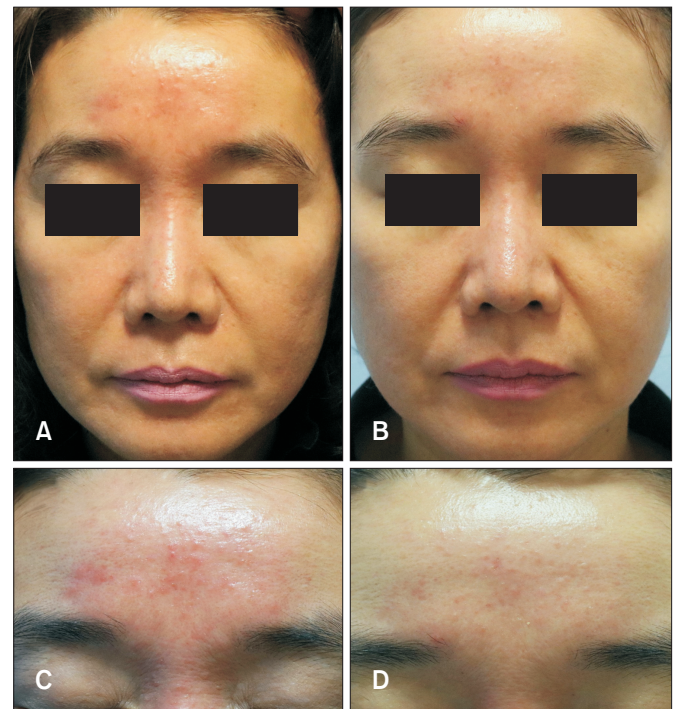


Fig. 1. Photographs of the entire face of the patient: (A, C) photographs taken on the initial visit, showing diffuse erythema on the forehead and glabella; (B, D) photographs taken after 2 weeks of metronidazole treatment. We received the patient's consent form about publishing all photographic materials.

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Fig. 2. Multiple erythematous papules and pustules on the bilateral cheeks were noted in the second patient.

for 2 years because of chronic idiopathic urticaria refractory to antihistamines and oral cyclosporine. Since the patient had been diagnosed with seborrheic dermatitis for many years, he thought that his facial skin lesion was due to an aggravation of seborrheic dermatitis. However, skin scraping test showed 17 *Demodex* mites from five papules. The lesions dramatically improved with topical metronidazole and topical ivermectin.

The mechanism by which IgE acts against parasitic infections is not precisely understood, but a significant increase in IgE levels during parasitic infections suggests a possible protective role of IgE¹. A study performed on patients at high risk of helminth infections showed that the incidence of helminth infection was 50% after omalizumab injection, compared to 41% in those who did not receive omalizumab injection². As methods for the detection of IgE specific to *Demodex* are unavailable, direct evaluation of their association is difficult. However, mast cell density has been reported to increase in the skin lesions of rosacea patients³; mast cells are known to mediate protective effects against the parasites in combination with IgE⁴.

In the first patient, symptoms of rosacea developed 6 months after the last omalizumab injection. The IgE levels remain persistently low after omalizumab treatment which was consistent with a previous study⁵. The association of omalizumab with the development of demodicosis was more prominent in the second case, in which the patient's symp-

toms worsened gradually when he was still receiving the omalizumab injections. The causality and temporality of omalizumab injection and the development of demodicosis in these cases strongly suggest that decrease in IgE levels following omalizumab treatment may be associated with demodicosis. Therefore, larger retrospective cohort studies are required to determine the relationship between omalizumab treatment and demodicosis.

CONFLICTS OF INTEREST

The authors have nothing to disclose.

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