

Table I. The occurrence of undercoding, correct coding, and overcoding before and after the implementation of dictation templates

	Coding		
	Appropriate coding	Undercoding	Overcoding
Before templates	59%	25%	16%
After templates	68%	23%	9%

code directly from the electronic documentation of the encounter.^{7,8}

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Wanted: A study with omalizumab to determine the role of IgE-mediated pathways in atopic dermatitis

To the Editor: We would like to comment on a Brief Report published in the *Journal* that concluded that the humanized, monoclonal anti-IgE antibody, omalizumab (Xolair), failed in the treatment of 3 patients with severe adult atopic dermatitis (AD).¹ Although clinical efficacy with anti-IgE therapy has been demonstrated in asthma (AS), allergic rhinitis, food allergy, and latex allergy, there have been no published randomized, double-blind, placebo-controlled studies in AD.¹⁻³ Omalizumab is approved by the Food and Drug Administration (FDA) for patients 12 years of age or older with moderate to severe persistent AS who have a positive skin test or in vitro reactivity to a perennial aeroallergen and whose symptoms are inadequately controlled with inhaled corticosteroids. AS and allergic rhinitis trials found that anti-IgE reduced not only circulating free IgE levels (IgE not bound to omalizumab), but also the expression of the high-affinity IgE receptor (FcεRI) on basophils, mast cells, and dendritic cells (Fig 1).^{2,4} In early phase II and III trials of omalizumab, it became clear that dosing must take into account both weight and pretreatment IgE levels (IU/mL),^{5,6} as clinical efficacy required achieving free IgE levels that were lower than 17 IU/mL.⁵

Herein lies the major flaw in the case series presented by Krathen and Hsu.¹ The treated patients with AD had IgE levels (5440; 23,000; or 24,400 IU/mL) that were 7- to 30-fold higher than the highest pretreatment serum IgE level that can adequately be dosed based on current FDA recommendations (ie, 700 IU/mL for weight ≤ 60 kg). Although patients were given a dose greater than FDA recommends (450 mg subcutaneously every other week), these patients received from 0.002 to 0.0005 mg/kg/IU every 4 weeks, which is significantly less than the recommended guidelines established to achieve clinical efficacy (0.016 mg/kg/IU).

Therefore, there was no hope to achieve sufficient reduction in free IgE levels necessary to reduce FcεRI expression and the lack of clinical efficacy in the chosen patients is expected (Fig 1).² This study highlights the importance of understanding the mechanism by which omalizumab works. Having said that, the authors report that a patient who, in addition to AD had severe AS, noted significant improvement in lung disease after 4 months of omalizumab therapy. Without providing objective findings of this improvement (eg, Forced Expiratory Volume at 1 minute, Forced Vital Capacity, or rescue medication use [beta agonists or inhaled

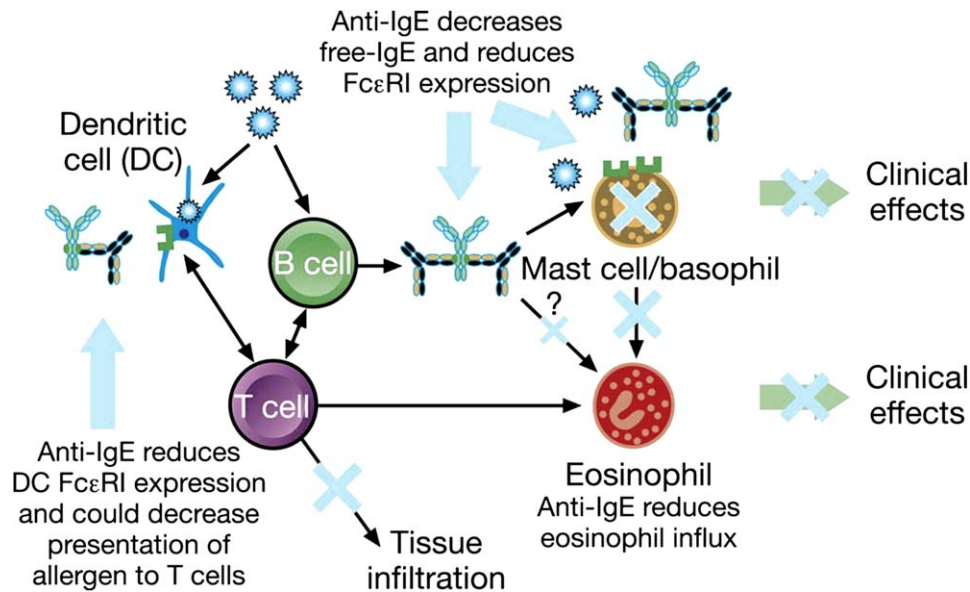


Fig 1. Clinical and biologic effects of omalizumab therapy. Reprinted from Holgate et al² with permission from the American Academy of Allergy, Asthma, and Immunology.

corticosteroid)) it is difficult to interpret this comment. To date, out-of-range dosing has led to minimal improvements over placebo in AS trials.⁶

Patients with AD and IgE levels within the dosing range (<700 IU/mL), in general, have milder disease and, therefore, are not a group that needs an expensive new biologic agent. Nevertheless, we suggest that omalizumab is a valuable tool to determine IgE's role in atopic skin disease, which is a highly controversial area. Establishing the role of IgE in this disease will encourage the application of new small molecular-weight antagonists directed against IgE signaling molecules that will be a more pharmacoeconomically viable treatment option than omalizumab for patients with the high levels of IgE characteristic of patients with severe AD. Therefore, we suggest that a well-designed and controlled study be performed testing omalizumab's efficacy in patients with mild to moderate AD who are within dosing range (based on weight and IgE levels), and that anecdotal case reports such as the one published by Krathen and Hsu¹ need to be interpreted with caution.

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Reply

To the Editor: We thank Drs Beck and Saini for their interest in our case series. They state that “there was no hope to achieve sufficient reduction in free IgE levels necessary to reduce FcεRI expression and the lack of clinical efficacy in the chosen subjects is expected.” There is no data on the dosages required to treat atopic dermatitis with this medication; it simply does not exist. To state that there was no hope even before the patients started the medication is a little unfair.