

Clinical Communications

Food allergy—exacerbated atypical hidradenitis suppurativa

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Clinical Implications

- A few studies link hidradenitis suppurativa to immune pathways dysregulation, and some evidence suggests that diet plays a role in disease activity and severity. We report for the first time a case of food allergy—exacerbated atypical hidradenitis suppurativa.

We present a unique case of atypical hidradenitis suppurativa (HS) involving the posterior neck and exacerbated by egg allergy.

A 23-year-old male patient was referred to our allergy and clinical immunology unit with complaints of recurring papules and pustules on the posterior neck, the current episode occurring after the consumption of an egg white (EW) meringue dessert at a wedding a few days before his visit. A thorough history revealed recurring episodes of burning, local warmth, hyperhidrosis, and pain-localized posterior neck accompanied by abdominal pain and sometimes generalized pruritus following the consumption of uncooked or undercooked eggs. He had tolerated egg and egg-containing products until the age of 14 years. Pruritus and abdominal pain were not provoked by other foods; nor had they appeared spontaneously at times when egg or egg-containing products had not been eaten.

The patients' medical history was remarkable for seasonal allergic rhinitis, morbid obesity, and acquired acanthosis nigricans from the age of 15 years, and a laparoscopic sleeve gastrectomy preformed at the age of 19 years. The patient does not have hypertension, and has a fasting glucose level within normal limits.

The medical records showed several visits to dermatologists with a diagnosis of "furunculosis," when they described skin abscesses with draining sinus and postinflammatory double-ended comedones. The patient experienced at least 3 to 4 recurrences of the disease each year since the age of 14 years, and he reported that each time the appearance of skin lesions was consistently associated with ingestion of eggs and egg-containing products. Each time the initial skin inflammatory lesions progressed to more chronic lesions and new lesions were added. The subcutaneous nodules drained sporadically and healed with rope-like and linear scars. Prescribed treatments included mupirocin ointment, and courses of both clindamycin and amoxicillin/clavulanic acid. The previous repetitive hematoxyline-eosin biopsies from the skin lesions showed epidermal ulceration and dermal scarring. During exacerbations, punch biopsies for bacterial and fungal cultures and fungal PCRs were performed several times. All the investigations were negative for any causative microorganisms.

On physical examination, the patient exhibited cutaneous changes in acanthosis nigricans on the neck, the axillae, and the

groins. He had a large abscess with purulent and bloody discharge on his right posterior neck (Figure 1) and an additional small inflammatory nodule on the left posterior neck.

The routine laboratory test results were normal, and total plasma IgE level was 127 kU/L (normal, 2-78 kU/L). A skin prick test was performed with a large panel of commercial food allergen extracts (ALK-Abelló, Horsholm, Denmark). The patient showed 7 mm mean wheal size/15 mm erythema size reaction to EW and 12 mm mean wheal size/28 mm erythema size reaction to whole egg extract (negative control 0; positive control 4/10 mm).

The egg protein-specific IgE class antibodies measured by ImmunoCap — Phadia were positive for EW (23.9 kU/L), ovalbumin (9.7 kU/L), and ovomucoid (15.2 kU/L).

Egg allergy was confirmed by double-blind placebo-controlled challenge. The double-blind placebo-controlled challenge to raw EW was performed. Starting dose was 0.12 g EW protein. Consecutive doses were 0.24, 0.48, 0.96, and 1.9 EW protein (cumulative dose 3.4 g). Chocolate drink was used for masking. After consumption of 1.9 g EW protein, the patient experienced abdominal pain, nausea, nasal secretion, generalized pruritus, and mild urticarial rash, which were absent during the placebo series.

Although the posterior neck is an unusual site for HS, the diagnosis of atypical HS was made by 2 board-certified dermatologists, on the basis of typical lesion morphology and scarring, and the chronic and recurrent nature of the disease.¹

HS is a clinically defined disease, with no pathognomonic tests, which contributes to the significant diagnostic and therapeutic delay in many patients.² Clinical guidelines recommend that the diagnosis of HS rely on typical lesions, recurrence, and a predilection for typical intertriginous sites.^{1,2} However, despite the atypical distribution, the absence of pathogenic microbes on culture, lack of resolution with topical and systemic antibiotics, and associated conditions such as obesity and acanthosis nigricans all support the diagnosis of atypical HS. Although rare, previous cases of atypical HS involving solely the neck and without involvement of other typical areas have been described.^{3,4}

An elimination diet for eggs was recommended with no additional topical or systemic therapy. Our patient has undergone a nutritional counseling to ensure he has been avoiding allergenic foods while consuming a healthy, balanced diet. Within a few weeks, resolution of most symptoms occurred, with disease activity index reduced from moderate (1 abscess and 1 inflammatory nodule, according to physician global assessment scale) to minimal (only a single small noninflammatory nodule, according to physician global assessment scale) (Figure 1). Currently, the patient has been symptom free for 6 months, following an egg-free diet.

A growing body of evidence suggests that diet plays a role in HS activity and severity. The dietary interventions explored in the studies were a dairy-free diet, brewer's yeast exclusion, and the Mediterranean diet.⁵ Improvement in HS following a dairy restriction diet for 12 months was interpreted as caused by decrease in metabolic effects of dietary rather than by an immunologic mechanism.⁶ According to the nutritionist, there was no caloric decrease or any significant changes in



FIGURE 1. Clinical photograph of HS at presentation and after 3 months of egg-elimination diet.

consumption of carbohydrates, proteins, and dietary fats, to have substantial metabolic effects due to avoidance of eggs.

To date, there are no data available on a possible connection between food allergy and HS. Serum levels of IgE are increased in about one-third of patients with moderate to severe HS.⁷ Nevertheless, there is limited basic research on the contribution of IgE, T_H2 immunity, and mast cells in the pathophysiology of HS. An increased number of tryptase-positive mast cells was found in early and chronic lesions and in normal-appearing perilesional skin of patients with HS.⁸ Chemokine CCL-26 (eotaxin-3) is a newly identified inflammatory marker in HS that is upregulated in the circulation of patients with HS.⁹

We theorize that during coincidence of food allergy and HS, food-specific IgE may possibly trigger degranulation of mast cells in lesional and perilesional skin, causing exacerbations of HS.

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