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Erythrodermic Psoriasis and Alpha-gal Syndrome

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Alpha-gal Syndrome, Allergic reactions, Skin disease.

Alpha-gal syndrome (AGS) is a newly recognized and classified disease. This is an acquired delayed IgE antibody-mediated tick-borne allergy to mammalian meat and products due to a reaction to the oligosaccharide galactose-alpha-1, 3-galactose (alpha-gal). Patients with AGS often manifest with a delayed allergy 2 to 6 hours after ingesting mammalian meat or animal products. Various manifestations of AGS are still being revealed. Here we submit a case of AGS presenting as an explosive onset of erythrodermic psoriasis with quick and complete resolution after elimination of the ingestion of mammalian meat and products derived from it.

AGS is a delayed-onset allergic reaction due to exposure to mammalian meat. This is due to the synthesis of IgE antibodies against alpha-gal which is a sugar present in the tissues of mammals except humans and other primates [1]. It is also called mammalian meat allergy, red meat allergy, tick-bite meat allergy and alpha-gal allergy. Patients develop sensitivity towards alpha-gal through the bite of Lone Star tick (*Amblyomma americanum*), but occasionally other species in America and other regions of the world [2]. Some well-known features of AGS are abdominal pain, nausea, vomiting, diarrhea, heartburn, urticaria, angioedema, anaphylaxis, hypotension, tachycardia, and fatigue [1-3]. The onset of allergic reaction can occur within 2-6 hours (in some cases as soon as 10 minutes) after exposure to alpha-gal found in not only mammalian meat but also dairy products, gelatins, biologic drugs, gelatin-containing vaccines, animal heart valves, and cosmetics [1-3]. Diagnosis of AGS is frequently based on the classic clinical history of exposure to the provocative antigenic source, delayed allergic symptoms, and a serum IgE level of >0.1 IU/mL to alpha-gal [2]. If the serum IgE is not elevated, then confirmatory skin prick testing and intradermal testing can be done to establish the diagnosis of what could be considered 'seronegative' AGS especially if the clinical picture is suggestive [2].

The current recommended treatment for AGS is abstaining from mammalian-meat consumption, and eliminating the use of mammalian products [2,4]. It is also recommended to use epinephrine for post-exposure prophylaxis to prevent anaphylactic reaction/shock [2-5].

Psoriasis is a chronic inflammatory papulosquamous skin disease which can be associated with various factors including infection (e.g. guttate psoriasis after Streptococcal infection), familial/genetic predisposition, or medications (including hydroxychloroquine, and tumor necrosis factor alpha inhibitors [TNFi]) [6,7]. It is

commonly associated with a positive family history, psoriatic arthritis, onychodystrophy, enthesitis, and dactylitis [7]. Symptoms classically include well-defined erythematous scaly plaques but can include desquamative plaques, and sterile pustules; laboratory anomalies include neutrophilia, leukocytosis, elevated acute phase reactants, and elevated transaminases [7]. Psoriasis is mainly a clinical diagnosis, although a skin biopsy can be utilized occasionally for confirmation of diagnosis [7]. Erythrodermic psoriasis is a rare form of psoriasis encompassing a large surface area of skin (75-90%) with a painful pruritic scaly rash on an erythematous background [8]. There are multiple triggers for erythrodermic psoriasis such as infection, medication-withdrawal, or emotional stress. In addition to pruritus, there can be systemic symptoms of fever, chills, arthralgia, and lymphadenopathy. Typical agents to treat erythrodermic psoriasis are conventional synthetic disease modifying anti-rheumatic drugs (DMARDs) like methotrexate, calcineurin inhibitors like cyclosporine, biologics like TNFi, and IL-17 antagonists, and targeted synthetic DMARDs like Janus-kinase inhibitors. Systemic steroids for erythrodermic psoriasis are controversial and not recommended due to frequent exacerbation after steroid cessation [8]. To the best of our knowledge there is no studied relationship between psoriasis and AGS, let alone AGS presenting as erythrodermic psoriasis. We hereby submit a situation of erythrodermic psoriasis being the presenting feature of AGS. We intend to expand on the current literature on what is known about AGS, specifically its variable presentations at the onset.

A 69-year-old lady presented to the Internal Medicine clinic in Tahlequah, the capital of The Cherokee Nation, Oklahoma, with a new explosive onset extensive pruritic scaly flattened erythematous rash which started abruptly 1 week prior. Her past medical history included chronic peripheral vascular disease and hypothyroidism. She reported the rash initially started on the dorsum of both hands and then quickly spread to her arms, chest, neck, abdomen, face, scalp, back, legs, and feet, sparing the soles of her feet and palms of her hands. The rash covered about 90% of the patient's body surface area at the time of presentation.

She initially treated her condition with topical emollients and over-the-counter antihistamines without relief. There was concern for contact dermatitis for which she was treated with topical triamcinolone cream, conservative measures of cleaning her sheets and bedding at home, but this did not resolve her rash. She was provided permethrin and ivermectin due to concerns over scabies, but this also did not relieve her symptoms. After informal consultation with Rheumatology for a possible autoimmune etiology, a detailed immunologic panel was obtained and multiple punch biopsies of the skin rash were obtained. Serology showed a significantly elevated titer of IgE antibodies to alpha-gal at 3.27 IU/mL, this being more than 10% of the total IgE level (the cut-off being 2%). Skin biopsy showed foci of parakeratosis overlying an epidermis exhibiting psoriasiform hyperplasia and focal deficiency of the granular layer with occasional Langerhans cells. The papillary dermis contained an infiltrate of lymphocytes and histiocytes with an 'eosinophilic component' which included the granulating cells in abundance (Figures 1-3 below).

Figure 1

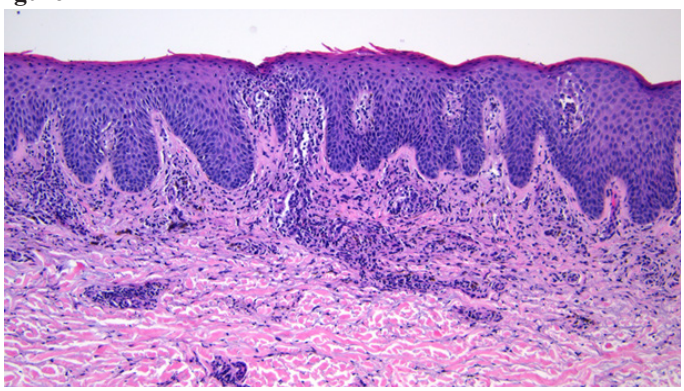


Figure 2

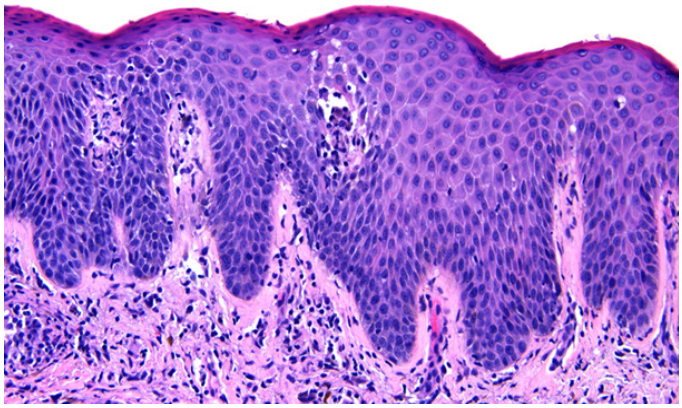
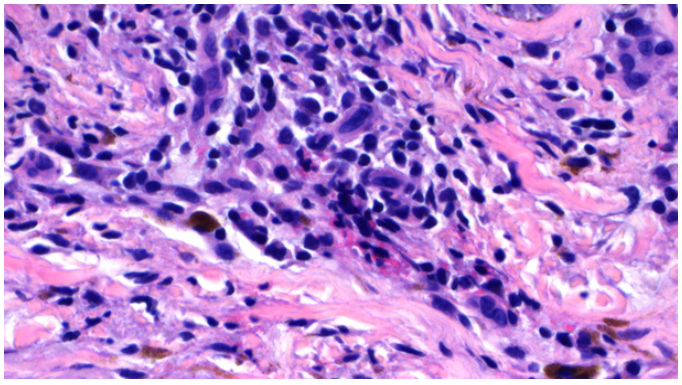


Figure 3



The remaining rheumatologic panel including antinuclear antibodies with reflex to various extractable nuclear antibodies, and anti-neutrophil cytoplasmic antibodies, was unrevealing. With a diagnosis of AGS presenting as erythrodermic psoriasis, a shared informed decision was made to eliminate ingestion of/exposure to mammalian meat products including milk and dairy products. She was prescribed an epinephrine auto-injector but thankfully never required use.

She was reevaluated two weeks later in the clinic and was found to have a near-total resolution of her skin rash. On later evaluation, she endorsed an additional episode of rash with less severity, and later discovered her previous meal included a beef broth base. This episode was resolved within 3 days on a more mindful nutrition refraining from all mammalian products. This is a unique and interesting presentation of AGS with erythrodermic psoriasis, which demonstrated rapid recovery of disease with elimination of all mammalian-product exposure. The patient's inadvertent food-challenge resulted in the recurrence of her rash although less severe in nature. Alpha-gal syndrome is an emerging, tick bite-associated allergy triggered by the alpha-gal sugar found in mammals and mammal-derived ingredients, which are present in countless processed foods and other products. As physicians practicing here in Oklahoma, we have seen firsthand a marked increase in both the prevalence and severity of AGS allergic reactions among patients in recent years. Oklahoma, like much of the southern and Midwestern U.S., has experienced a rapid growth in lone star tick populations, significantly contributing to the rise of this dangerous allergy. The patient in this case did not know of recent tick bites but does live in rural eastern Oklahoma which is known to have more than 87 cases per million based on positive serologic testing in samples sent to commercial laboratories; in parts of Oklahoma, 3% or more of the population may now be affected by AGS [9].

The CDC estimates that up to 450,000 Americans may already be living with AGS, making it the '10th most common food allergy'. Alarmingly, up to 75% of those affected experience life-threatening anaphylactic reactions an even higher rate than individuals with peanut allergies. Unlike other allergens, mammal-derived ingredients are notoriously difficult to identify on food labels. Many byproducts have technical or unfamiliar names like glycerin or monoglycerides, magnesium stearate, or oleic acid. As a result, individuals with AGS and their families struggle daily to ensure the food they consume is safe, often without the clear guidance that labeling provides for other allergens.

Transparency and trust in our food system are essential for the well-being of the public. Without proper labeling, people with AGS are left vulnerable to severe health risks. The Alpha-gal inclusion act is advocating for the inclusion of alpha-gal on the list of major allergens.

This case shows a correlation between AGS and diffuse erythrodermic psoriasis, substantiated by the eosinophils in the skin biopsy. While there is no previously described connection between erythrodermic psoriasis and AGS, a plausible etiologic postulation is as follows. Psoriasis is a known immunologic inflammatory dermatologic reaction which is dependent on T cell activation and its associated interleukins to produce the characteristic rash [10]. Some known interleukins include IL-12, and IL-23 which result in T-cell activation which in turn produces IL-17, IFN-gamma, TNF, and IL-22 causing the characteristic psoriatic rash [7,10].

Erythrodermic psoriasis can be a presentation of AGS. We caution healthcare providers to be aware of AGS as an underlying association with erythrodermic psoriasis and to obtain a thorough history of their patient's potential exposure to tick/zoonotic infection, dietary habits and use of products containing derivatives of mammalian meat. We also advise practitioners to evaluate and treat AGS skillfully, and to ensure these patients are well supplied with emergent medications such as epinephrine-pen in the event of potential anaphylaxis. As more revelations appear about the myriad clinical presentations of AGS, we will continue to learn about the expanded spectrum of the disease to better educate our patients and prevent potentially fatal complications like anaphylactic reactions. To the best of our knowledge, this is the first observation of AGS presenting as erythrodermic psoriasis.

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