

Nummular Eczema Successfully Treated with a Gluten-free Diet: First Description

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In 2016, an 11-year-old girl experienced an allergic rhinitis reaction. Months later she started to experience spontaneous pruriginous skin lesions on her lower limbs. She denied local trauma, pain, or drug use. She had various episodes of recurrence of these lesions, which were not related to infections or drug use.

Her physical examination showed skin lesions with an annular pattern as well as erythematous with mild scaling plaques on her left elbow, left the popliteal region, and lower third of the left leg. She was treated with local moisture creams, and a slight improvement was noted. Antihistamines were offered, but showed no effect.

Topic hydrocortisone had a marked improvement, but new lesions relapsed after the drug suspension.

Laboratory tests revealed normal eosinophil levels (280 cell/mcl/mm³). A mild increase in total IgE was observed (138 KU/L; nr < 123 KU/L). Serology for infectious diseases and antipretptolysin O were all negative. Antinuclear antibodies, anti-dsDNA, anti-Sm, anti-Ro/SS-A, anti-La/SS-B, anti-U1RNP, IgA, and IgG anti-deaminated gliadin, anti-endomysium, and anti-tissue transglutaminase were not found and complement levels were normal.

Although the patient denied symptoms and signs linked to gluten, and gluten-related autoantibodies were negative, we suggested that the patient try a gluten-free diet (GFD). After starting this diet, she experienced a complete and quick resolution of all skin lesions.

She stayed symptom-free until August 2020, when she ate pizza. A few days after eating the pizza, she had a relapse of the typical skin lesions on her left foot [Figures 1A and 1B]. A direct mycological test and culture for fungi were negative. A skin biopsy showed parakeratosis, spongiotic cells in the epidermis, acanthosis, vacuolar degeneration of the basal layers of the epidermis, and cellular infiltrates containing mononuclear cells in the upper dermis. We reinforced the need to keep a GFD, and after that, she had a marked improvement of the lesion.

Figure 1. Typical sharply defined, oval-shaped erythematous eczematous plaque over the left leg compatible with nummular eczema [A] and a more detailed vision [B]



The authors followed the World Medical Association Declaration of Helsinki. Informed consent was obtained from the patient for publication of her case.

Nummular eczema is a chronic inflammatory skin disease characterized by multiple and symmetrical sharply defined, oval or coin-shaped, erythematous eczematous pruriginous plaques. It appears commonly in men between 55 and 65 years of age, and in women 15 to 25 years old, it is a rare condition in pediatrics [1,2].

The diagnosis is clinical, and biopsy usually unnecessary. The differential diagnosis was performed with tinea corporis, psoriasis, contact dermatitis, lichen simplex, and stasis dermatitis [3]. This eczema's pathophysiology is unclear [4]. Treatment involves avoidance of precipitating factors, optimal skincare, and high or ultra-high potency topical corticosteroids.

To the best of our knowledge, we are the first to describe a patient with nummular eczema induced by gluten. This diagnosis and treatment seem correct as the patient stayed symptom free for 2 years when she followed a GFD. Furthermore, after eating pizza, she had a relapse, but the lesion cleared after reintroduction of the GFD.

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Capsule **$\gamma\delta$ T cells link immunity to nutrition**

Gamma delta ($\gamma\delta$) T cells are immune cells best known for host barrier defenses in epithelial tissues. **Sullivan** and co-authors discovered a previously unrecognized role for $\gamma\delta$ T cells in sensing nutrient uptake in the small intestine. The researchers analyzed mice fed a high-carbohydrate versus a high-protein diet and observed remodeling of the small intestinal epithelium in response to dietary carbohydrates. Nutrient availability triggered an epithelial-

immune cell circuit that was required for digestion and absorption of carbohydrates. Intestinal $\gamma\delta$ T cells regulated the expression of a carbohydrate transcriptional program by limiting interleukin-22 production from type 3 innate lymphoid cells. These findings may also provide insights into how $\gamma\delta$ T cells modulate metabolic disease.

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Eitan Israeli

Capsule**DUBbing new inflammasome inhibitors**

Inflammasome assembly and activation leading to mature interleukin-1 β (IL-1 β) release is dysregulated in a wide range of inflammatory diseases. Optimal activation of the NLRP3 inflammasome, a protein complex important for IL-1 β release, requires the activity of BRISC, a deubiquitinating enzyme (DUB) complex composed of four protein subunits including the metalloprotease BRCC3. **Ren** et al. demonstrated that the compound thiolutin, a zinc chelator that inhibits BRCC3, can potently inhibit NLRP3 deubiquitination and inflammasome activation.

Thiolutin was effective at inhibiting NLRP3 activation and preventing IL-1 β production in multiple mouse models of inflammatory disease, including a model of diet-induced nonalcoholic fatty liver disease. Holomycin, a derivative of thiolutin with reduced toxicity, was also effective at inhibiting NLRP3, paving the way for the development of agents that selectively target deubiquitination of NLRP3 to regulate its activity.

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Capsule**Costs of moving stem cells**

Adult stem cells travel long distances to a wound to repair the damaged tissue. The potential cost of migration has been revealed in in vitro studies of cancer cell lines, dendritic cells, and primary stem cells. If these cells have to squeeze into wounds, then this constriction may cause DNA damage. **Sahu** et al. showed that adult stem cells in *Schmidtea mediterranea*, a highly regenerative planarian

flatworm, accumulate DNA damage as they migrate. The flatworm's stem cells actively repair the migration-inflicted DNA damage en route. The authors propose that during migration, the stem cells go through a "migration-damage-repair-migration" cycle as they home into a wound.

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