

## TEACHING FILES (GRAND ROUNDS)

# ITCHING FOR AN ANSWER: MANAGING A RECURRENT EVOLVING ANNULAR RASH IN A TEEN GIRL

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### KEYWORDS

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### Clinical Problem:

A 13-year-old female with a history of atopy presented with a recurrent and worsening skin rash for 2 months (Figure 1). Around age 3 years, she developed a scaly, pink patch in the shape of a heart on her left dorsal foot. She was treated for tinea corporis with over-the-counter topical antifungal cream and hydrocortisone without response. A presumptive diagnosis of granuloma annulare was subsequently made and she was treated with desoximetasone 0.25% cream and halobetasol propionate 0.05% ointment for 6 months with no improvement. Parents stopped the topical steroids and the lesion resolved on its own several months later. It never itched or caused pain; it did not bother her. Over the next several years, the skin underneath the previous site became thicker and "lumpy." Examination at seven years old revealed a 1.5 cm subtle soft mobile subcutaneous nodule on the left dorsal foot. The lesion remained relatively unchanged until 2 months prior to presentation when she developed new slightly hyperpigmented "lumps" on her left foot. She described it as mildly pruritic. Although not painful, it was uncomfortable especially when wearing shoes and playing sports. Examination revealed multiple slightly violaceous soft mobile subcutaneous nodules extending down the left foot and persistence of the prior lesion.

*What are the causes of annular skin lesions in the pediatric population and how to treat them?*

### Discussion:

Annular lesions acquire their name from the Greek *annulus*, meaning "ring." These lesions characteristically manifest as circles of erythematous or otherwise discolored skin surrounding centers of normal or abnormal epidermis. Annular lesions in

**Figure 1.** Patient's foot at age 13 years with new lesions. Red arrows indicate original lesion borders that presented at age 3 years.



child populations carry a wide differential diagnosis that must be thoroughly considered by practitioners confronting such cases. The etiologies of annular lesions in children can be infectious, inflammatory, drug- or substance-induced, or idiopathic.<sup>1,2</sup> Infectious causes of annular lesions are common in the pediatric population. One important diagnosis for consideration is superficial dermatophytosis or tinea corporis. Colloquially termed ringworm, this infection arises from fungal colonization of the epidermis, with the most-implicated species being *Trichophyton rubrum*.<sup>3</sup> The disease spreads via direct skin-to-skin contact and can transmit from animal to human. The disease manifests as erythematous, intensely pruritic, scaling, circular, or oval patches or plaques that radiate outward, leaving central clearing behind raised

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advancing borders. Scrapings taken from these borders and prepared with potassium hydroxide demonstrate fungal hyphae to confirm the diagnosis. Tinea corporis usually responds to topical antifungal treatment like terbinafine<sup>4</sup> and with treatment readily resolves. Another infectious cause of pediatric annular lesions is erythema migrans (EM), a manifestation of Lyme disease. Caused by infection via *Borrelia burgdorferi* following a bite from colonized Ixodes ticks, the lesion heralds the first stage of Lyme disease and appears as a small papule surrounded by deep erythema. This rash blossoms into the classical “bull’s-eye” distribution with central irritation, an inner clear ring, and an outer erythematous border. Interestingly, younger children more often develop EM on their head or neck, whereas older children predominantly demonstrate infection on their extremities.<sup>5</sup> Discernment of infection can be made by history in which details like tick exposure and recent travel to endemic areas pinpoint the diagnosis. Treatment usually consists of two weeks of amoxicillin for pediatric patients younger than eight years of age, otherwise, treatment of choice is doxycycline. Other causes of pediatric cutaneous annular lesions, while not derived directly from infection, are thought to be associated with post-infectious states. Pityriasis rosea, shown to have possible causal linkage to reactivated human herpesviruses 6 and 7,<sup>6</sup> manifests as a ring-like “herald patch” that precedes the eruption of crops of ovoid lesions in a pathognomonic “Christmas Tree” distribution. Treatment focuses on symptomatic relief of associated pruritus, and the prognosis is favorable with rash resolution in two to three months. Inflammatory pediatric disease can lead to annular lesion formation. One disease of chronic inflammatory etiology is psoriasis, which has increased in prevalence among children in recent decades.<sup>7</sup> The disease manifests in pediatric populations most commonly as psoriasis vulgaris and is characterized by erythematous papules with an overlying white scale. On physical exam, upon scale removal, the underlying skin will bleed in pinpoint, known as the Auspitz sign. Psoriasis has no known cure but can be managed by topical steroids as first-line treatment. A disease with similar presentation but not as well-understood as psoriasis is nummular eczema (NE). NE, or discoid eczema, presents as solitary coin-shaped plaques ranging from 1 to 3 centimeters in size. Etiology of the disease is unknown, but some patients may have a concurrent history of atopy or excessively dry skin.<sup>8</sup> The plaques demonstrate pinpoint oozing on evaluation, sometimes requiring a microscopic evaluation to differentiate from fungal infection. Like psoriasis, the condition can be managed with topical steroids. Chemical- or environment-induced annular lesions usually present as urticaria or hives. The condition occurs when mast cells, in hyperacute immune reactions, induce edematous inflammation.<sup>9</sup> The inflammation manifests as circumferential wheals with central pallor and raised erythematous borders. The wheals can resolve and then appear in other locations, hence its description as a “migratory” rash. Diagnosis can be made clinically based on skin exam and history of recent exposure to a known or likely trigger. The condition resolves with removal

of the trigger and short course of antihistamines. One other condition to consider for pediatric annular lesions is cutaneous lupus erythematosus (CLE). CLE arises from deposition of immunoglobulins and complement in the dermal-epidermal junction, manifesting as coin-like scaly erythematous plaques.<sup>10</sup> Lesion biopsy demonstrating lymphocytic infiltrate and hydropic changes in the skin confirms the diagnosis.<sup>11</sup> Treatment focuses on preventing progression to systemic lupus erythematosus using topical steroids and calcineurin inhibitors.<sup>10</sup> Our patient reached a presumptive diagnosis of GA after failed initial treatment for fungal infection. Compared to tinea and other diseases on this patient’s differential, the prevalence of GA in the general population is low, between 0.1% and 0.4%. Greater than two-thirds of cases occur in patients younger than 30 years old, with the disease afflicting females more often than males.<sup>12</sup> In pediatrics localized GA is common; few cases exist in the literature documenting generalized GA in childhood. Clinically, GA manifests as annular, non-scaly, violaceous to skin-colored plaques with ropy, often incomplete borders. The lesions can be otherwise asymptomatic or associated with mild to moderate pruritus. Localized lesions declare as singular plaques affecting the hands and feet, with rare cases of volar surface involvement reported.<sup>13</sup> Conversely, generalized presentations appear as multiple coalesced lesions on the trunk or the extremities. The pathogenesis of GA remains unclear. Several theories point to collagen degradation because of delayed-type hypersensitivity reactions from erroneous Th-1 lymphocyte stimulation.<sup>14</sup> Histological exam shows focal collagen degeneration surrounded by interstitial or palisading patterns of histiocytes and lymphocytes. Often the clinical exam is sufficient for diagnosis; rarely biopsy is performed to confirm the diagnosis. The treatment of choice is topical steroids, but in severe generalized cases inclusion of cyclosporine and even antimalarial medications have been shown to manage symptoms.<sup>15</sup> Our patient fits the diagnosis of generalized GA based on prior history and disease progression. In searching for similar pediatric GA cases, one study reported generalized GA in an eighteen-month-old female.<sup>16</sup> In that case, the diagnosis was based on clinical and histological findings. What makes our patient’s case generalized is the chronicity and morphology of her disease. The first-foot lesion appeared at three years old, resolved for several years, and then reappeared but remained stable for several years before additional lesions appeared and coalesced to form the present rash. Localized GA will resolve after a few weeks to months with some recurrence. However, the persistence of disease for nearly a decade, coupled with the clinical presentation, befits a generalized GA diagnosis. Our patient was treated with 0.1% triamcinolone ointment. At the time of this report, the patient had not noticed much change in the lesion but admits to inconsistently applying the steroid ointment.

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