An Itchy, Round Rash on the Back of an Adolescent's Neck

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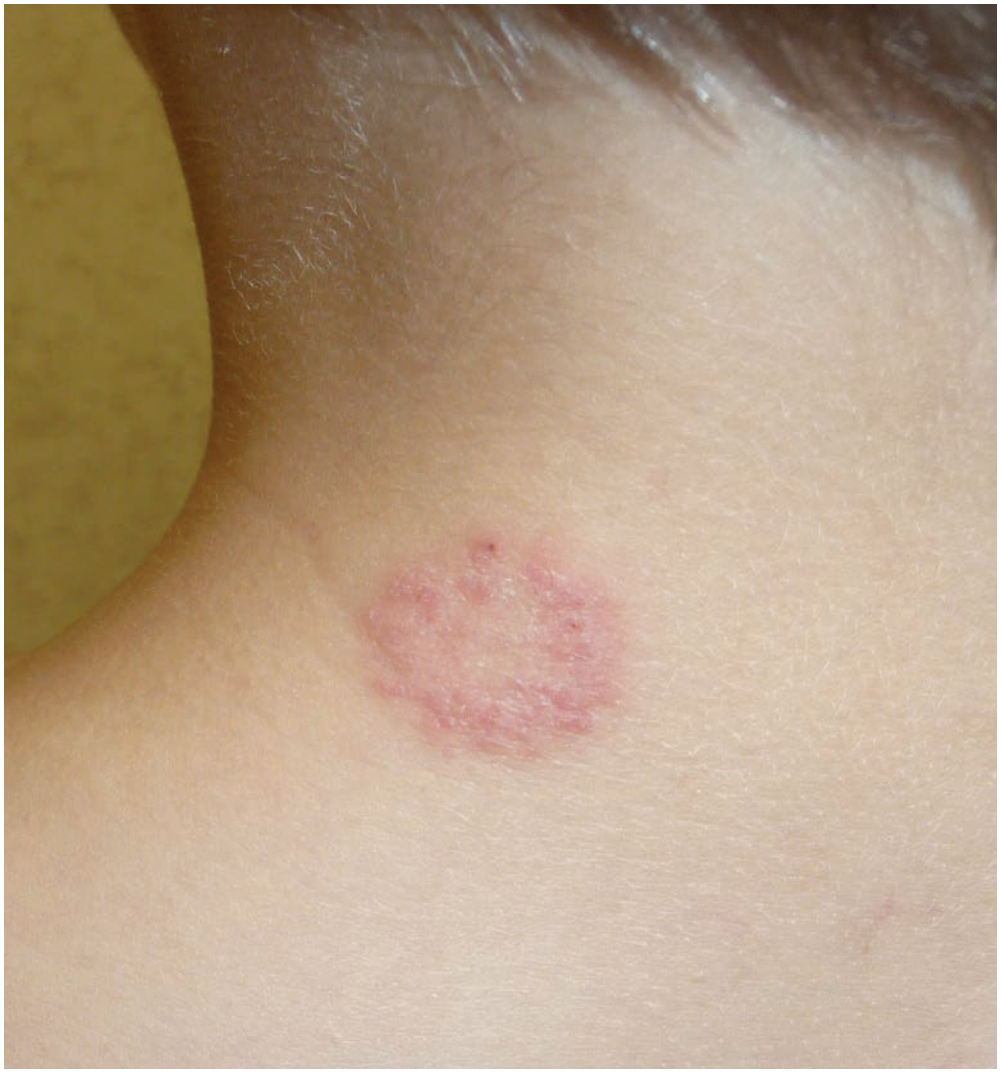
A 16-year-old boy presented with an erythematous round rash on the nape. The rash had been first noted 3 months ago, and it had been slowly enlarging. The lesion was slightly itchy.

Incidentally, the child had concomitant tinea pedis. There was no family history of similar skin lesions. He had a dog at home.

On physical examination, an annular, scaly plaque with a well-defined border and a raised leading edge was noted on the nape. There was some degree of central clearing. The rest of the examination findings were unremarkable.

What's your diagnosis?



Answer: Tinea corporis

Tinea corporis ("ringworm") refers to a superficial fungal infection of the skin other than on the scalp (tinea capitis), face (tinea faciei), bearded areas (tinea barbae), groin (tinea cruris), hands (tinea manuum), feet (tinea pedis), and nails (onychomycosis).[1] Tinea corporis most often is caused by any of the dermatophytes belonging to three genera: *Trichophyton*, *Microsporum*, and *Epidermophyton*.[1,2] Because tinea corporis is common and the differential diagnosis often is difficult, pediatricians must be familiar with this condition and its treatment.

ETIOLOGY

Tinea corporis most often is caused by *Trichophyton rubrum*, *Trichophyton tonsurans*, or *Microsporum canis*.[2,3] Other causative organisms include *Epidermophyton floccosum*, *Microsporum audouinii*, *Trichophyton interdigitale* (previously known as *Trichophyton mentagrophytes*), and *Trichophyton verrucosum*.[3]

EPIDEMIOLOGY

The estimated lifetime risk of acquiring tinea infection is 10% to 20%.[3] The peak incidence is after puberty.[1] The sex ratio is equal.[1] Humans may become infected through close contact with infected persons or animals (particularly house pets) or contaminated fomites or soil.[1,4,5] Transmission among family members is the most common route; children often become infected by spores shed by a household contact.[5,6] Autoinfection by dermatophytes elsewhere in the body also may occur.[7] The transmission of tinea corporis is facilitated by a warm and moist environment, the wearing of occlusive clothing, and the sharing of towels and clothing.[1,7] Immunodeficiency, diabetes mellitus, genetic predisposition, xerosis, and ichthyosis are other predisposing factors.[5,8]

PATHOGENESIS

The causative organism can produce enzymes such as proteases that digest keratin and keratinases that penetrate keratinized tissue.[2,9] The hyphae then invade the stratum corneum and keratin and spread centrifugally outward.[9] Infection usually is cutaneous and restricted to the nonliving cornified layers, because the fungus is not able to penetrate the deeper tissue of healthy immunocompetent host.[2] Scaling results from increased epidermal replacement following inflammation.[9]

CLINICAL MANIFESTATIONS

Typically, tinea corporis presents as a sharply circumscribed, well-demarcated, annular, erythematous plaque with a raised leading edge and scaling.[7,10] The border can be papular, vesicular, or pustular.[6] The lesion spreads centrifugally and clears centrally to form the characteristic lesion commonly known as ringworm.[7,11] In general, lesions caused by anthropophilic species tend to be less erythematous and inflammatory than those caused by zoophilic species. Tinea corporis tends to be asymmetrically distributed. When multiple lesions are present, they may become coalescent. Pruritus is common.[7,12]

Tinea corporis also may present in a non-ringworm fashion. In tinea corporis gladiatorum, the lesion presents as well-defined, erythematous, scaling plaques.[3] Other forms include bullous tinea corporis, characterized by vesicles, bullae, or pustules that may be herpetiform; tinea imbricata, characterized by widespread, scaly, polycystic lesions; and tinea profunda or subcutaneous abscess.[12]

DIAGNOSIS

The diagnosis often is clinical, especially if the lesion is typical.[3] However, the diagnosis can be difficult in patients with prior use of medications such as corticosteroids or calcineurin inhibitors. Tinea incognito refers to a dermatophytosis that has lost its typical morphologic features because of the use of corticosteroids or calcineurin inhibitors.[13] If necessary, the diagnosis can be confirmed by potassium hydroxide (KOH) wet-mount examination of skin scrapings of the active border of the lesion.[4] A drop of 10% to 20% KOH, with or without dimethyl sulfoxide, is added to the scrapings.[1] The specimen is then gently heated to accelerate the destruction of the squamous cells if no dimethyl sulfoxide is added.[1,11] The KOH dissolves the epithelial tissue, leaving behind easily visualized septate hyphae.[1,11]

Although fungal culture is the gold standard to diagnose dermatophytosis, culture rarely is needed unless the diagnosis is in doubt or the infection is severe, widespread, or resistant to treatment.[4,9] Culture testing is expensive and takes 7 to 14 days for results.[6] The most common culture medium is Sabouraud peptone-glucose agar.[11] Wood lamp examination is not useful, since the lesion of tinea corporis usually does not fluoresce with a Wood lamp.

DIFFERENTIAL DIAGNOSIS

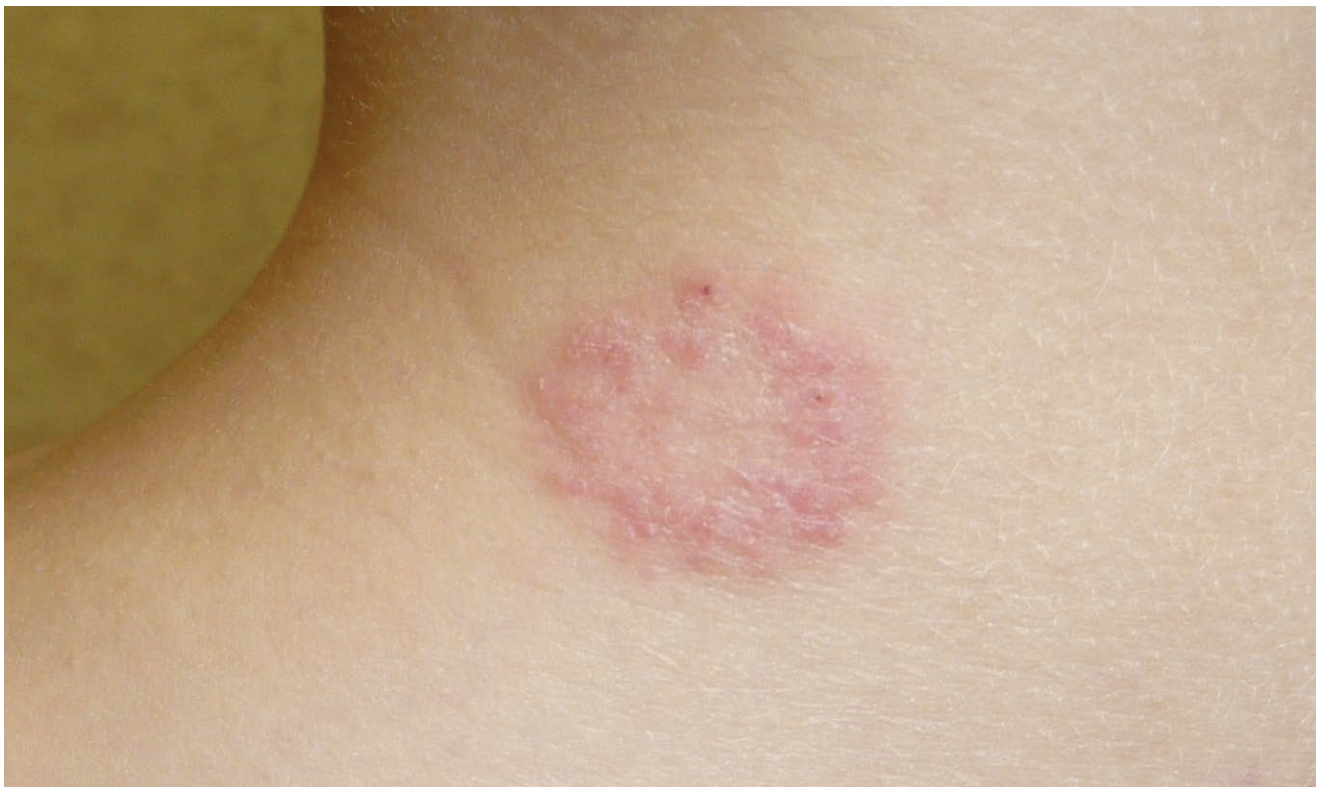
Differential diagnosis includes the herald patch of pityriasis rosea, *Candida* infection, nummular eczema, psoriasis, contact dermatitis, seborrheic dermatitis, tinea versicolor, granuloma annulare, fixed drug eruption, discoid lupus erythematosus, urticaria, erythema migrans, erythema marginatum, erythema annulare centrifugum, and impetigo.[6-9,12]

COMPLICATIONS

Occasionally, the lesion may become chronic. Postinflammatory hypopigmentation and hyperpigmentation may occur.[11]

TREATMENT

Superficial or localized tinea corporis usually responds to topical antifungal therapy, given twice daily for 2 to 4 weeks.



Commonly used topical antifungal agents include butenafine, ciclopirox, econazole, ketoconazole, miconazole, naftifine, terbinafine, and tolnaftate.[10] In a mixed-treatment comparison (head-to-head trials and trials with a common comparator) meta-analysis involving 14 topical antifungal treatments, there was no significant difference among the antifungals.[14] Butenafine, naftifine, and terbinafine might be the best strategies for maintaining cured status.[14] Topical antifungal agents are well tolerated, and adverse reactions are uncommon, except for rare instances of contact dermatitis. Because fungi thrive best in moist, warm environments, patients should be advised to wear loose-fitting clothes.[11]

Systemic treatment is indicated if the lesion is extensive, chronic, or recurrent, or if the patient is immunocompromised.[7] Oral antifungal agents used for the treatment of tinea corporis include itraconazole, fluconazole, ketoconazole, terbinafine, and butenafine.[9] Combined therapy with topical and oral antifungals may increase the cure rate.

PROGNOSIS

The prognosis is good with appropriate treatment. If the diagnosis is in doubt or if treatment is failing, consider referral to a dermatologist. ■

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