

Erythema Nodosum Probably Induced by Kerion Celsi

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A 6-year-old girl visited us with a 4-week history of inflammatory, pustular, tender patches and plaques on the scalp, and a 3-day history of multiple, erythematous, indurated, tender, subcutaneous nodules on both pretibial areas. A swab was taken from the scalp lesion and colonies of *Trichophyton mentagrophytes* grew on culture. The histopathological findings of the leg nodule were consistent with erythema nodosum. The patient was treated with oral itraconazole and deflazacort, combined with topical potassium permanganate solution. Erythema nodosum regressed two weeks later and the kerion of the scalp regressed six weeks after starting the treatment, leaving residual scarring alopecia. The patient was diagnosed as erythema nodosum probably induced by kerion celsi, that has not been reported in the Korean literature. (Ann Dermatol 16(2) 64~66, 2004)

Key Words: Erythema nodosum, Kerion celsi, *Trichophyton mentagrophytes*

INTRODUCTION

Erythema nodosum (EN) is the most common form of panniculitis and typically presents as an acute eruption of erythematous, tender, subcutaneous nodules over the pretibial areas bilaterally. EN is regarded as a delayed hypersensitivity response to a variety of antigenic stimuli including infection, neoplasia, inflammatory disease, and chemical agents. The association of kerion celsi (KC) and EN has been rarely reported, and we could find only ten cases of EN associated with KC in the literature¹⁻⁶. However, it has not been reported in Korean literature.

CASE REPORT

A 6-year-old girl visited us with a 4-week history

of scalp lesion and a 3-day history of both legs lesion. She had presented with erythematous, scaly, pruritic patches on the scalp four weeks before and had been treated with topical antifungal agent at private clinic, however two weeks later the scalp lesion had aggravated to become pustule. In addition erythematous painful nodules had developed on the both legs three days earlier. The girl complained of itching and pain of the scalp lesion. The patient's history was unremarkable and her grandmother denied any contact with domestic animals. Physical examination revealed inflammatory, pustular, tender patches and plaques on the fronto-occipital scalp (Fig. 1), and multiple, erythematous, indurated, tender, subcutaneous nodules on the pretibial areas of both legs (Fig. 2).

Routine laboratory tests were within the normal values, except for mild leukocytosis and an increase in the erythrocyte sedimentation rate. A swab was taken from the scalp lesion and the culture showed a growth of white, soft, cottonlike colonies identified as *Trichophyton mentagrophytes* on Sabouraud's dextrose agar with antibiotics. The histopathological findings of the leg nodule were an inflammatory infiltrate of the thickened septa of subcutaneous fat that extended to the periseptal areas of the fat lobules, and a superficial and deep dermal perivascular inflammatory infiltrate in the overlying dermis,

Received January 8, 2004

Accepted for publication April 26, 2004

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Fig. 1. Inflammatory, pustular patches and plaques on the scalp.



Fig. 2. Multiple, erythematous, indurated nodules on the pretibial areas of both legs.

which were consistent with erythema nodosum (EN) (Fig. 3).

The patient was diagnosed as EN induced by kerion celsi and was treated with oral itraconazole 5 mg/kg/day and deflazacort 0.7 mg/kg/day, combined with topical 1:10,000 potassium permanganate solution. The lesion of EN regressed two weeks later and oral deflazacort tapered off over the next three weeks. The kerion of the scalp regressed six weeks after starting the treatment, and healed leaving

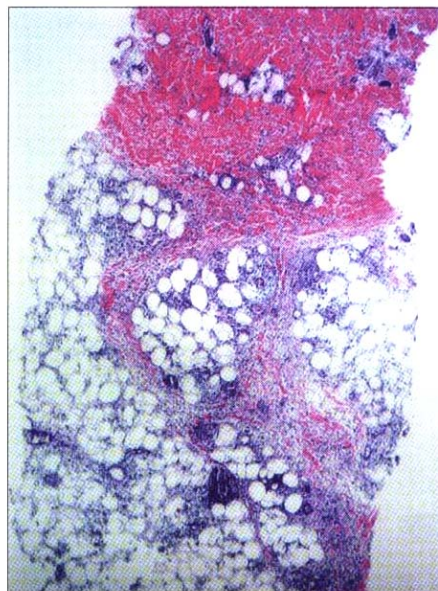


Fig. 3. The biopsied tissue of the leg nodule revealed thickened septa with an inflammatory infiltrate that extended to the periseptal areas of the fat lobules, and a perivascular inflammatory infiltrate in the overlying dermis (H&E, $\times 40$).

residual scarring alopecia and areas of nevus comedonicus on follow-up examination a month later.

DISCUSSION

It is known that 35 - 55% of cases of erythema nodosum (EN) are idiopathic, however a wide variety of triggering factors have been associated with EN. Streptococcal infections, especially upper respiratory infections, are the most common cause, and tuberculosis, Mycoplasma, sarcoidosis, inflammatory bowel disease, and drugs are commonly reported causes.

In fungal infections, EN has usually been associated with systemic mycoses like coccidioidomycosis, histoplasmosis, and sporotrichosis. The association of kerion celsi (KC) and EN has rarely been reported; we could find only ten cases of EN associated with KC in literature¹⁻⁶. The previously reported patients were either children or adolescents, aged 5 to 18 years. The most common organism is *Trichophyton mentagrophytes* (*T. mentagrophytes*) isolated in eight patients, and *T. gypsum* and *T. sulfureum* were isolated in another two patients. Our described

patient was also a child and *T. mentagrophytes* was identified. EN developed on average 13 days after KC had appeared in the previous ten cases, but EN developed 25 days after the onset of scalp lesion in our case. However, in fact, it developed 11 days after the aggravation of scalp lesion. We agree to the opinion of Calista et al. that the excessive cellular immune response to *Trichophyton* antigens or self-antigens induced by the *Trichophyton* could cause the severe clinical appearance and could induce EN⁶.

Our patient had not been given any oral medication before visiting us and we could not find any sign of other infection or systemic disease in the patient. In addition, the lesion of EN regressed in accordance with improvement of KC. We think our patient's EN was probably induced by KC, and it was more evident unless oral deflazacort had been taken. This is an unusual and interesting case that has not been reported in Korean literature. We propose KC be considered one of the precipitating factors of EN.

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