

Neonatal Kerion Celsi: Report of Three Cases

Margarita Larralde, M.D., Ph.D.,*,†, Begoña Gomar, M.D.,†, Paula Boggio, M.D.,†
María Eugenia Abad, M.D.,*,† and Betina Pagotto, M.D.†

*Dermatology Department, Hospital Alemán, Buenos Aires, Argentina,

†Pediatric Dermatology Department, Hospital Ramos Mejía, Buenos Aires, Argentina

Abstract: Tinea capitis is a fungal infection caused by dermatophytes, frequent in children but uncommon in the neonatal period. Kerion Celsi is the inflammatory manifestation of tinea capitis secondary to host immunologic responses and its occurrence in newborns is extremely infrequent. We describe three neonates with the diagnosis of kerion Celsi. The isolated dermatophytes were *Trichophyton mentagrophytes* var. *mentagrophytes* in two patients and *Trichophyton rubrum* in the third. Both patients with *T. mentagrophytes* referred an indirect contact with rabbits and were successfully treated with systemic antifungal (griseofulvin and fluconazole). The patient with *T. rubrum* had a father with a tinea manuum and both received just topical antimycotic treatment.

Kerion Celsi (KC) is an inflammatory presentation of tinea capitis (TC) which appears as a boggy plaque with alopecia, pustules and often, purulent drainage from its surface. It is uncommon in newborns and only a few cases occurring at this age have been reported in the literature (1–5).

We present three cases of KC in neonates, two caused by *Trichophyton mentagrophytes* and one by *Trichophyton rubrum*, which displayed good response to antifungal therapy.

CASES REPORT

Case 1

A 55-day-old boy, apparently healthy, presented with a well demarcated round-shaped hairless and boggy plaque on the scalp that was painful to palpation. Clinical manifestations began on the second week of

life. Mycological studies (direct examination of hairs in potassium hydroxide—KOH—and culture) were performed and *T. mentagrophytes* var. *mentagrophytes* was isolated. Treatment was started with oral griseofulvin 20 mg/kg/day. After 45 days of treatment, his scalp was clinically cured. His father worked as a rabbit breeder and related that some rabbits had patchy hair loss on the ears; thus, he was the presumed source of infection.

Case 2

A 20-day-old girl, seemingly healthy, appeared with a 1-week history of an erythematous, pustular and crusting lesion on scalp (Fig. 1). At the time of examination her father presented an erythematous, desquamative and ill-defined plaque on his left hand. Mycological studies (direct examination of hairs in KOH and culture) were

Address correspondence to Margarita Larralde, M.D., Ph.D., Acevedo 1070, Banfield, Buenos Aires, Argentina, or e-mail: margaritalarralde@fibertel.com.ar, maggie@advancedsl.com.ar.



Figure 1. Case 2: Erythematous, oozing and crusting plaque on parieto-occipital scalp.



Figure 2. Case 3: Round erythematous and pustular plaque on the parietal area of the scalp.

performed in the baby and her father and *T. rubrum* was isolated in both cases. Treatment with topic imidazole was initiated for both of them, with complete clinical recovery within 2 months of initiation.

Case 3

A 4-week-old boy, apparently healthy, presented with 1-week history of an erythematous plaque covered by several pustules on scalp. Physical examination revealed pus oozing from hair follicles. Clinical diagnosis of KC was made (Fig. 2). On mycological studies (direct examination of hairs in KOH and culture) *T. mentagrophytes* var. *mentagrophytes* was isolated and treatment with oral fluconazole 5 mg/kg/day was started. Four weeks of treatment resulted in complete resolution of the scalp lesion. Investigation of environmental conditions revealed the presence of a rabbit as a domestic pet in the patient's house. Parents denied direct contact of the baby with the animal.

DISCUSSION

Tinea capitis is a fungal infection caused by dermatophytes, frequent in children but uncommon in the neonatal period. KC is a markedly inflammatory manifestation of TC secondary to a vigorous host immune response, and its occurrence in newborns is extremely infrequent. The rare presentation of KC in neonates is probably explained by the immaturity of their immunologic system and also because dermatophytes require an incubation period of 1 to 3 weeks to produce clinical manifestations (6). Nevertheless, in some experimental studies it was demonstrated that *T. rubrum* could be reproduced in 5 days under occlusion and *T. mentagrophytes* in a shorter time (7,8). In our pediatric dermatology section, only three cases of neonatal KC that manifested around the second week of life have been seen in the last 10 years.

Kerion Celsi is caused most often by zoophilic dermatophytes (*Microsporum canis* and *T. mentagrophytes*), but also by anthropophilic (*T. rubrum*) and rarely by geophilic (*Microsporum gypseum*) species. In Argentina, the most frequent isolated agent is *M. canis*, which is transmitted by dogs and cats. As rabbits have become domestic pets, the frequency of *T. mentagrophytes* var. *mentagrophytes* has increased, with higher presentation of inflammatory lesions (5). In accordance with this is the fact that two of our three neonatal KC were produced by *T. mentagrophytes* var. *mentagrophytes* and that rabbits were observed in both patient's environment.

The main source of the fungi responsible for KC is from humans or animals, though dermatophytes may spread via fomites (hairbrushes, combs, hats, and contaminated grooming instruments) (6). In newborns, inter-human spread is the most common mode of transmission of anthropophilic dermatophytes, but has also been considered when zoophilic species are involved. In both cases 1 and 3, the zoophilic dermatophyte involved was indirectly transmitted by humans who had close contact with the babies.

Kerion Celsi clinically presents as a boggy plaque with alopecia, pustules and often, purulent drainage from its surface. It is usually solitary but multiple lesions may be found. Reactive lymphadenopathy, especially cervical or suboccipital, is a very common associated feature. All the newborns presented herein had unique lesions with the typical clinical aspect of KC and absence of lymphadenopathies.

Confirmation of KC diagnosis is desirable, and the gold standard diagnostic method is fungal culture (9). This was performed in all our patients.

The principal differential diagnoses of KC include impetigo and bacterial or sterile folliculitis or abscesses.

Despite the fact that it is very uncommon in newborns, KC should be considered within differential diagnosis of neonatal scalp inflammatory lesions. Careful examination of people in close contact with infants as well as pets may help in the clinical suspicion.

Systemic treatment is the first election for TC and KC. Griseofulvin is the drug of choice at doses of 20 mg/kg/day for 6 to 12 weeks. Other options are terbinafine, fluconazole, and itraconazole (10–13). Topical therapy may be tried in newborns and a favorable response may be explained because in the neonatal period a higher percentage of hairs are in telogen phase, and dermatophytes mainly affect the hairs in anagen phase (10,14). We had a good clinical response with systemic treatment in cases 1 and 3 and with topical therapy in the case 2. However, each patient must be evaluated carefully to decide the best individual therapy.

We report on three neonatal cases of KC and emphasize that although rare, a high index of suspicion is crucial when facing inflammatory scalp lesions in newborns, to establish an early diagnosis and initiate an appropriate treatment.

REFERENCES

1. Khare AK, Singh G, Pandey SS. Kerion tinea faciei and tinea corporis in an infant. *Ind J Dermatol Venereol Leprol* 1984;50:271–272.
2. Weston WL, Morelli JG. Neonatal tinea capitis. *Pediatr Infect Dis* 1998;17:257–258.
3. Torrelo A, Zambrano A. Querion de Celso en un recién nacido. *Actas Dermosifiliogr* 1998;89:133–140.
4. Aste N, Pinna AL, Pau M et al. Kerion Celsi in a newborn due to *Microsporum canis*. *Mycoses* 2004;47:236–237.
5. Larralde M, González VM, Label M et al. Variación clínica y epidemiológica de dermatoficias zoófilas. *Arch Argent Pediatr* 2001;99:205–209.
6. Paller AS, Mancini AJ. Skin disorders due to fungi. In: Paller AS, Mancini AJ, eds. *Hurwitz clinical pediatric dermatology*. USA: Elsevier Saunders, 2006:449–478.
7. Reinhardt J, Allan A, Gunnison D. Experimental human *Trichophyton mentagrophytes* infections. *J Invest Dermatol* 1974;63:419–422.
8. Sloper JC. A study of experimental human infections due to *Trichopyton rubrum*, *Trichophyton mentagrophytes*, and *Epidermophyton floccosum*, with particular reference to the self-limitation of the resultant lesions. *J Invest Dermatol* 1955;25:21–28.
9. Gilaberte Y, Rezusta A, Gil J et al. Tinea capitis in infants in their first year of life. *Br J Dermatol* 2004;151:886–890.
10. Friedlander SF, Rueda M, Chen BK et al. Fungal, protozoal and helminthic infections. In: Schachner LA, Hansen RC, eds. *Pediatric dermatology*. Philadelphia: Mosby, 2003:1093–1106.
11. Romano C, Gianni C, Papini M. Tinea capitis in infants less than 1 year of age. *Pediatr Dermatol* 2001;18:465–468.
12. Lipozencic J, Skerlev M, Orofino-Costa R et al. A randomized, double-blind, parallel-group, duration-finding study of oral terbinafine and open-label, high-dose griseofulvin in children with tinea capitis due to *Microsporum* species. *Br J Dermatol* 2002;146:816–823.
13. Dastghaib I, Azizzadeh M, Jafari P. Therapeutic options for the treatment of tinea capitis: griseofulvina versus fluconazole. *J Dermatolog Treat* 2005;6:43–46.
14. Gilaberte Y, Sáenz-Santamaría MC, Coscojuela C et al. Tinea capitis en lactantes. *Piel* 2000;18:21–29.