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CORRESPONDENCE

Inflammatory tinea capitis due to *Microsporum canis* transmitted from asymptomatic domestic cats

Dear Editor,

Tinea capitis is a cutaneous fungal infection of the scalp that is common among children. It is mainly caused by *Trichophyton tonsurans* and *Microsporum* (*M.*) *canis*.[1] *M. canis* is a dermatophyte fungus of which cats and dogs are natural hosts, which is easily transmitted to humans.

A four-year-old girl with no immunodeficiency was referred to our hospital with a four-month history of erythematous lesions on her scalp. The lesions had gradually enlarged despite the use of antibiotics, oral, and topical corticosteroids. A physical examination revealed multiple erythematous lesions with hair loss, crusts, and pustules (Figure 1A). Potassium hydroxide examinations of the hair root and scales showed no fungal elements. A histological examination showed granulomatous inflammation (Figure 1B), histiocytes and a few multinucleated giant cells were present (Figure 1C). Periodic acid-Schiff and Grocott's staining showed no fungal elements.

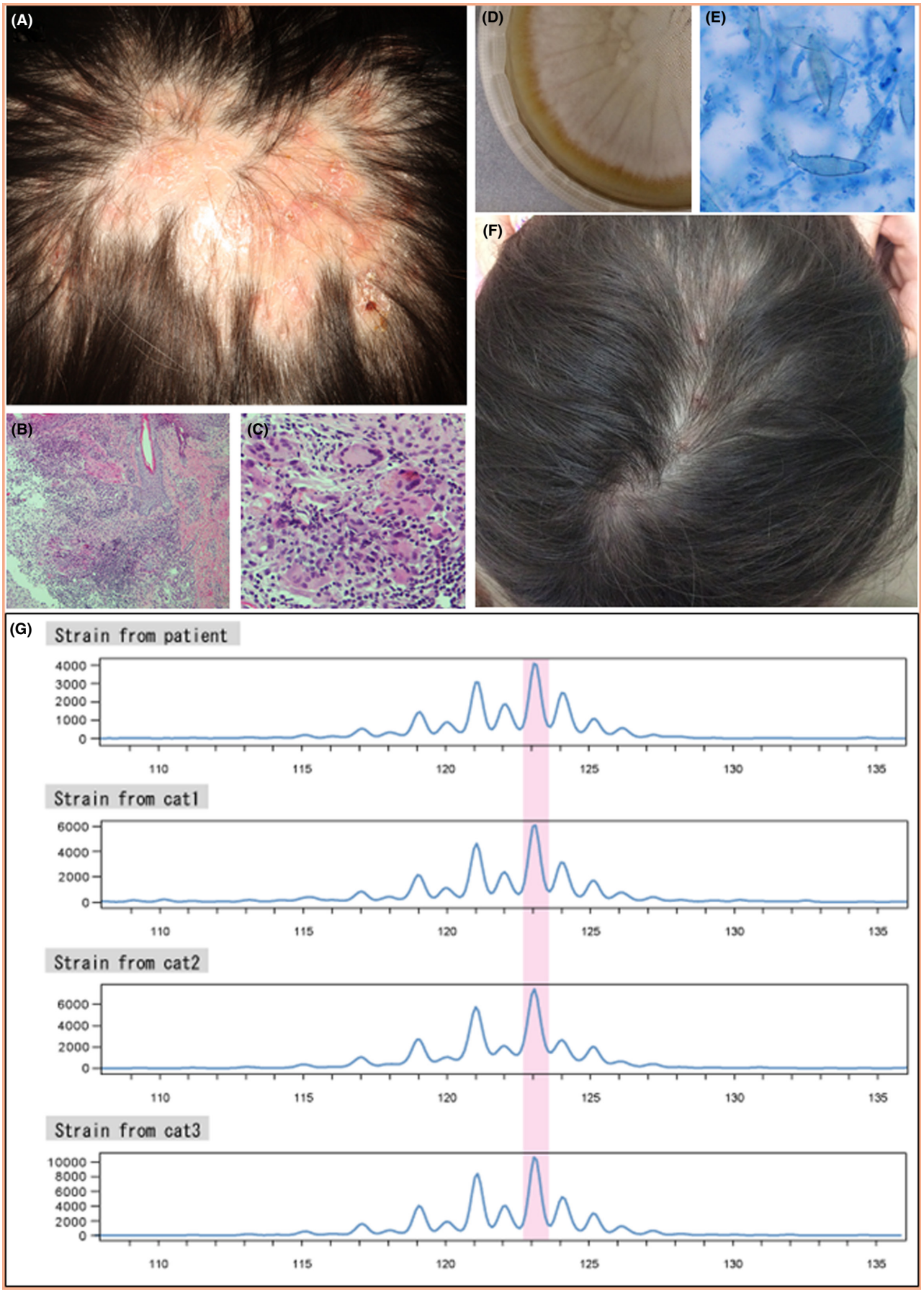
Culturing of scalp scales obtained by tape stripping on Sabouraud Dextrose Agar at 25°C showed a cream color with a dense cottony surface (Figure 1D). The scraped specimen from the culture showed hyphae and spindle-shaped macroconidia (Figure 1E). The sequence of the internal transcribed spacer (ITS) region in the ribosomal RNA gene of the strain amplified with the ITS1 and ITS4 primer set[2] was 100% homologous (698/698 bp) with that of the type strain *M. canis* CBS 217.69 (accession number MH859294.1) (data not shown). After 12 weeks of oral treatment with terbinafine (5 mg/kg/day), the hair regrew completely, although crusted erythema was observed (Figure 1F).

Fungal cultures from domestic animals performed to estimate the route of infection resulted in the growth of colonies similar to patient isolates from three cats. The molecular biology of the strains was consistent with that isolated from the patient (data not shown). *M. canis* is classified into 20 genotypes by a combination of microsatellite markers. We attempted to classify this isolate using three primer sets, MS2, MS4, and MS7.[3] Capillary electrophoresis did not result in a classification consistent with a previous report[3]; however, it showed that the peak positions of all isolates were consistent (Figure 1G), suggesting that the *M. canis* was transmitted from the domestic cats.

We did not have a direct evidence that the case was kerion celsi caused by *M. canis*. However, *M. canis* grew on the tape stripping culture before treatment but not after treatment, suggesting that the *M. canis* on the scalp surface was probably pathogenic. Tinea capitis with inflammation mimicking scarring alopecia was reported and its clinical manifestation was similar to that of our case.[4] We hypothesize that our patient had strong immunity to *M. canis* after the topical inoculation of the dermatophytes.

There appear to be two routes of *M. canis* among cats via a pet shop for domesticated animals and via the outdoors in strays.[5] Our patient's Persian cats were purchased from a pet shop. However, the family had many cats and dogs, and they might have had contact with stray animals. A microsatellite marker analysis can be a useful method for tracing the route of transmission.

FIGURE 1 (A) Clinical lesion on the occipital area of the scalp. Erythema and hair loss were observed. (B) Dense inflammatory cell infiltration was demonstrated through the dermis (hematoxylin–eosin, original magnification ×40). (C) Granulomatous inflammation with inflammatory cells, histiocytes, and a few multinucleated giant cells (hematoxylin–eosin, original manifestation ×400). (D) Culture findings, the colony grown on Sabouraud dextrose agar at 14 days at 25°C had a cream color with a dense, cottony surface. (E) Lactophenol cotton blue staining showed hyphae and spindle-shaped conidia (×400). (F) The erythema disappeared, and new hair grew back three months after the treatment. (G) Representative image of peaks by capillary electrophoresis of microsatellite markers. Peaks of the same sizes as formed by the microsatellite marker MS7. MS7 peaks of the strains isolated from patient, cat1, cat2, and cat3 were located at 123.1 bp, 123.1 bp, 123.0 bp, and 122.9 bp, respectively


(A)
(B)
(C)
(D)
(E)
(F)
(G)
Strain from patient
4000
3000
2000
1000
0
110
115
120
125
130
135
Strain from cat1
6000
4000
2000
0
Strain from cat2
Strain from cat3
10000
8000

CONFLICT OF INTEREST

None declared.

DECLARATIONS SECTION

Approval of the research protocol: No human participant was involved in this study.

Informed Consent: The patient has provided informed consent for the publication of the images submitted with this article.

Registry and the Registration No.: N/A.

Animal Studies: N/A.

Akira Shimizu MD, PhD[1,2]
Yuko Kuriyama MD, PhD[2]
Taketoshi Futatsuya MD, PhD[1]
Kazushi Anzawa[1]
Mayu Nishio MD[2]
Saki Kanai [2]
Osamu Ishikawa MD, PhD[2]
Sei-ichiro Motegi MD, PhD[2]

[1]*Department of Dermatology, Kanazawa Medical University, Uchinada, Japan*
[2]*Department of Dermatology, Gunma University Graduate School of Medicine, Maebashi, Japan*

Correspondence
Akira Shimizu, M.D., Ph.D., Department of Dermatology, Kanazawa Medical University, 1-1 Daigaku, Uchinada-machi, Kahoku-gun, Ishikawa 920-0293, Japan.
Email: ashimizu@kanazawa-med.ac.jp

ORCID

Akira Shimizu https://orcid.org/0000-0001-7742-079X
Sei-ichiro Motegi https://orcid.org/0000-0001-8286-0669

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