NOTE Internal Medicine

Arthroderma benhamiae Infection in a Rabbit

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ABSTRACT. The isolate from the rabbit with dermatophytosis which was transmitted to the owners was proved to be Arthroderma benhamiae (-) by mating experiments as well as by chitin synthase 1 (CHS1) gene analysis.

KEY WORDS: Arthroderma benhamiae, chitin synthase 1 gene, rabbit.

Rabbit dermatophytoses have been reported mainly in laboratory rabbit colonies and rabbits for commercial breeding [1, 3, 8] and were caused mostly by Trichophyton mentagrophytes. Although dermatophytosis is one of important zoonoses, there are few reports describing human cases transmitted from rabbits. Today many rabbits share the environment with the owners as companion animals. Therefore, it is important to diagnose a rabbit dermatophytosis correctly in small animal clinics as well as on the viewpoint of public health.

T. mentagrophytes was confirmed to be a complex including at least 3 species; Arthroderma benhamiae, A. simii and A. vanbreuseghemii [1, 7, 8, 9]. However, it is difficult to identify T. mentagrophytes isolates at the teleomorphic state by routine mycological examination. Recently molecular analysis was shown to be useful to differentiate these 3 species [5], indicating that some clinical isolates of T. mentagrophytes from human and animal dermatophytoses could be determined by molecular analysis [5]. In 1998, the first isolate of Arthroderma benhamiae in Japan from a 2-month-old rabbit was reported [6]. A. benhamiae was reported at first in U.S.A. and then isolated in Europe and Africa [1]. We report a rabbit case infected with A. benhamiae, which was investigated by molecular analysis as well as mycological examination.

January the 15th, 2000, a 4-month-old female dwarf rabbit was referred to the animal hospital in Urawa, Saitama, Japan. The chief complaint was hair loss, which had started several weeks before and had been gradually expanding. The rabbit had been kept for 3 months by the married couple. Physical examination revealed no abnormality except skin lesions, such as alopecia with scaly erythema on the face, head, auricles, dorsal neck and hind legs (Fig. 1) and reddish swelling at the external genital area. The rabbit owners also had skin lesions on their hands, legs, face or axilla.

Based on the clinical signs, skin lesions of the rabbit were suspected to be caused by a dermatophyte and the symptoms around the mouth and the external genital area suggested Treponema infection. The specimens were collected from the lesions for the microscopic examination. The hyphae and arthroconidia of a dermatophyte were detected (Fig. 2), but spirocheta could not. Oral administration of griseofulvin (25 mg/kg, sid) and topical administration of ketoconazole cream (1%) were carried out for dermatophytosis [2, 3]. Chloramphenicol palmitate (50 mg/kg bid p.o.) was also used.

After 4-week treatments, lesions on the face and genital area were cured and lesions on the other areas were also partially improved. Administration of chloramphenicol palmitate was continued for 6 weeks. Ten weeks after starting medication, the rabbit showed recovery almost all over the body except for auricles.

From skin scrapings, colonies developed on DTM (dermatophyte test medium) and 1/10 diluted Sabouraud’s dextrose agar. The colony of this clinical isolate grew a white and powdery surface after 2 week-incubation on sunflower seed agar at 24°C. Many tear-shaped microconidia and well-developed spirals were observed (Fig. 3). Therefore, the isolate was identified to be T. mentagrophytes. The same dermatophyte was isolated from skin lesions of the owners.

Isolates from the patient rabbit and the owners were examined by mating experiments as well as chitin synthase analysis.

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Fig. 1. The skin lesions showed alopecia and redness, with scales and scurf on the rabbit face.
gene 1 analysis [5].

DNA from the clinical isolates from the rabbit and the owner were prepared by the method of Kano et al. [5, 6]. Briefly, the sequences of the degenerate primers for CHS1 gene were based on the previous study reported [5]: primer 1, 5'-CTG AAG CTT ACT(ACG) ATG TAT(C) AAT(C) GAG(A) GAT(C)-3' and primer 2, 5'-GTT CTC GAG (C)TTT (A)GTA (C)TTC (A)GAA (A)GTT (T)CTG-3'. The PCR amplification of the clinical isolate DNA with degenerate CHS1 primers yielded fragments of about 620 bp and this fragments were cloned into plasmid vector and sequenced using an ABI PRISM 310 Genetic Analyzer (Perkin-Elmer, Foster City, U. S. A.).

Amplification of the clinical isolate DNA with degenerate CHS1 primers yielded fragments of about 620 bp, consistent with the sizes of CHS1 gene fragments from fungal species previously reported [5]. Nucleotide sequence analysis of the CHS1 gene fragments from the clinical isolates as well as tester strains of A. benhamiae (DDBJ data base AB003558), indicated that the sequence similarities were 99% among them. Based on these results, it was concluded that the clinical isolate was identical to A. benhamiae.
The mating experiments [4, 6] also confirmed those rabbit and human isolates to be \textit{A. benhamiae} (-) mating type (Table 2).

Dermatophytoses due to \textit{T. mentagrophytes} are sometimes transmitted to pet owners from their animals. Each animal species appears to have different susceptibility to dermatophyte species. In human cases, \textit{T. mentagrophytes} should be identified at a teleomorphic state, since it is important on the epidemiological points of view.

In this case, \textit{A. benhamiae} was isolated as an etiologic dermatophyte from a patient rabbit and owner. \textit{A. benhamiae} has been isolated from dermatophytosis of dog, horse, guinea pig and rabbit [1, 8]. These animals exist close to human environment. Since this infection could be transmitted to a human from affected animals, a prudent care should be taken whenever handling infected animals.

### REFERENCES