

TWENTY-NAIL DYSTROPHY: REPORT OF A PATIENT WHO WAS TREATED SUCCESSFULLY WITH ORAL STEROID THERAPY

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We report a 52-year-old female patient in whom all the finger- and toe-nails were rough and turbid. She had been treated as onychomycosis for a long time by the near-by dermatologists. Their treatments had not been effective at all. We diagnosed her nail lesions as twenty-nail dystrophy. She was treated by us with the oral steroid therapy. The nail lesions had been improved soon, and well controlled, by the steroid therapy.

Key words: twenty-nail dystrophy, onychomycosis, oral steroid therapy

INTRODUCTION

Because onychomycosis, which is frequently encountered in the dermatological clinics, manifests the deformity and turbidity of the nails, we often diagnose this condition of the nails as onychomycosis without any mycological examinations. However, the deformity and turbidity of the nails develop not only in the patients with onychomycosis, but also in those with nail diseases other than onychomycosis, such as twenty-nail dystrophy (TND), lichen planus and psoriasis vulgaris (1-4). In this paper, we report a Japanese female patient with TND who was successfully treated with oral steroid therapy. She had been misdiagnosed to be suffering from onychomycosis, and many antifungal therapies had been given inadequately by the near-by dermatologists for the previous several years.

PATIENT

A 52-year-old Japanese female patient visited our clinic on Oct. 29, 1998, due to the deformity and turbidity of all the twenty finger- and toe-nails. Approximately 14 years previously, she noticed the deformity and turbidity of all the toenails. Since, 4 years after that, all of her fingernails also became rough and deformed simultaneously, she visited some near-by dermatology clinics. All the dermatologists diagnosed her nail lesions as tinea unguium. The results of the mycological examinations they might have done were unclear at all. She was treated with the topical and oral anti-fungal drug therapies repeatedly. After about 10 years of the antifungal treatments without any improvement, she came to our clinic. All of her twenty nails manifested deformity, fragility, loss of luster and longitudinal ridgings (Figs. 1a and 1b). She was in good health except the nail disorders. Her nail folds, hairs, buccal mucous membranes and teeth were all normal. There were no family and personal history of the similar nail diseases. On the microscopic examinations of the nail specimens using the KOH-Parker ink method, we cannot detect any fungal element. We tried the examination repeatedly, but no fungal element was detected at any time. Next, the nail specimens from various sites were cultured on the Sabroud's glucose agar plates. No fungal colonies did grow on any agar plate. In addition, the HE and the PAS stainings of her nail specimen were performed, but no fungal element was seen (Figs. 2a and 2b). We thus concluded that her nail disorder had not been caused by fungal infection, but was TND.

We first tried topical steroid (Dermovate[®] scalp) and oral panthethine (Pantosin[®], 600 mg/day) therapies for a while. The nail lesions were improved, but only slightly. We then added oral steroid

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Fig. 1. All the finger- (a) and toe-nails (b) are rough and deformed.

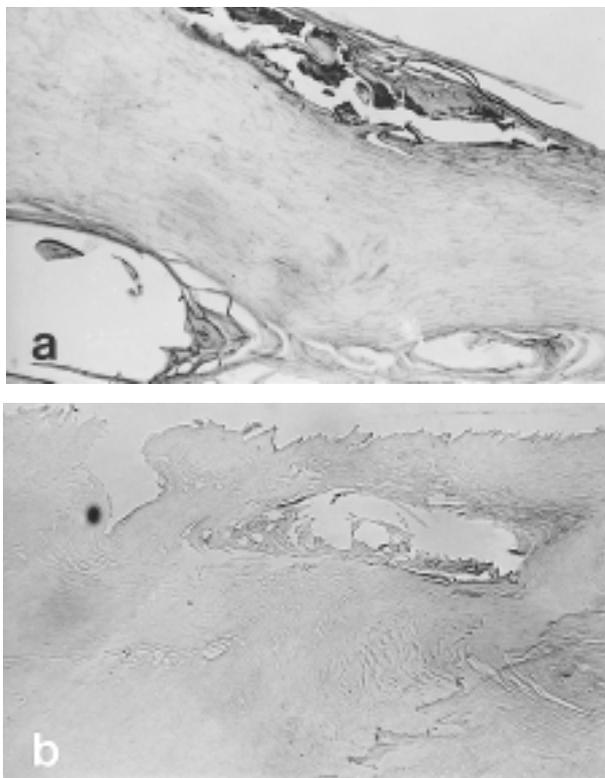


Fig. 2. HE (a) and PAS (b) stainings of the nail specimen. a; Only a low dense nail tissue was seen. No specific findings were seen in this specimen. b; Any fungal elements (PAS-positive) were not seen.

administration [1.5 mg/day of betamethasone (Rinderon®)]. After 2 months, the proximal portion of the nails became normal, and after the subsequent 2 months, the twenty nails all became normal. Her nails have been kept normal by the oral administration of 0.25 mg/day of betamethasone (Figs. 3a and 3b).

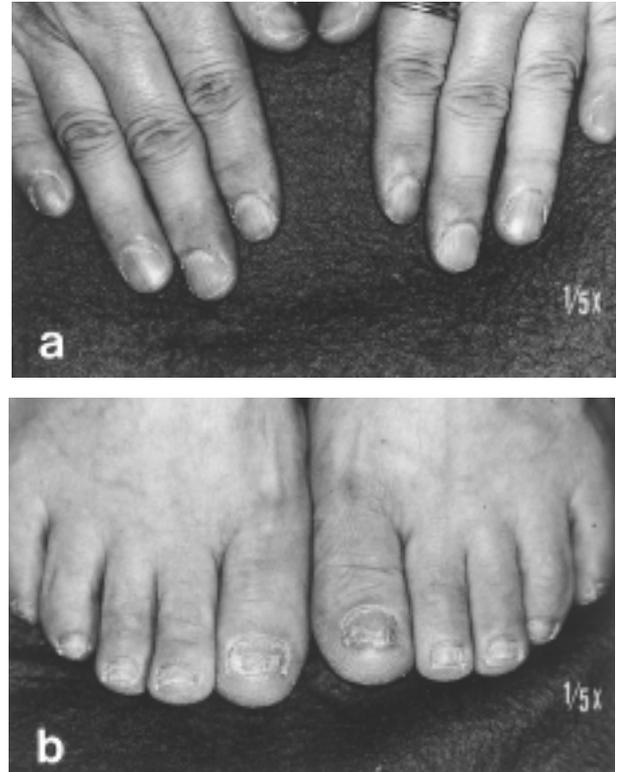


Fig. 3. The fingernails (a) and toenails (b) of the patient 5 months after the oral steroid therapy. The nails became normal.

DISCUSSION

TND, the term of which has been coined by Hazelrigg et al. in 1977 (1), is a new entity of the rare nail disorder with simultaneous and characteristic changes of all the twenty nails, lacking the lesions of the skin and buccal mucous membrane which appear with nail disorders such as psoriasis and lichen planus. The characteristics of the TND nails consist of longitudinal ridging, loss of luster and fragility. TND may appear in both the children and adults (1,2,5). The similar nail disorders to TND are frequently and sometimes exhibited in onychomycosis, psoriasis and lichen planus (1-4). The results of microscopical and cultural examinations for fungi can distinguish the onychomycosis from

TND. In usual cases of psoriasis and lichen planus, the nail disorders are observed only in a few nails and the nail changes are not uniform. Moreover, the psoriatic nails are characterized by pitting, hypertrophy, irregularity and onycholysis (1). The nails of lichen planus have the characteristic clinical feature called dorsal "pterygium" (a gradual shortening of the proximal nail groove) (1,6). There should be some evidence of psoriasis or lichen planus on the skin and buccal mucosa somewhere if the nail lesions are a part of psoriasis or lichen planus.

In our patient, all the twenty nails had been affected. Onychomycosis was denied because of the results of mycological examinations. There had not been any skin and buccal membrane lesions compatible with psoriasis and lichen planus. We thus diagnosed her to be suffering from TND (1,7).

The pathogenesis of TND has been still unknown (8). Other nail disorders such as nail lichen planus and nail psoriasis often come up as differential diagnoses. Thus, some insist that lichen planus or psoriasis may be a cause of TND, and try to explain the pathogenesis of TND. However, it has been controversial that lichen planus or psoriasis may be a cause of TND. Takeuchi et al. (9) have reported a TND case which might be caused by the allergic reaction for metal. In our patient, there was no episode of metal allergy. Assuming the involvement of autoimmune process, Ohta and Katsuoka (8) have implied that the histopathological findings from the nail biopsies in their TND case could become a clue to clarifying the pathomechanism of TND, because the histopathology showed the cell infiltration into the nail matrix with spongiotic foci. In any case, the pathogenesis of TND has remained unclear.

Topical PUVA (the combination of psoralen application and UVA radiation) (10), and systemic steroid therapy (9) have been proposed as the effective therapies for TND. In our patient, the systemic steroid therapy was very effective, although the PUVA

was not tried.

Our patient showed that the nail diseases clinically simulating onychomycosis may include the other nail diseases such as TND.

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