

Candida Folliculitis Mimicking Tinea Barbae

Sir,

Candidiasis is caused by the *Candida* species, which is a dimorphous fungus growing in both yeast and filamentous forms on the skin. Based on the period of onset, recurrence, immunity and distribution, the spectrum of the infection may be divided into 3 groups: acute mucocutaneous candidiasis; chronic mucocutaneous candidiasis; and disseminated candidiasis (1). Acute mucocutaneous candidiasis, the common, benign, self-limited form of candidiasis, mainly occurs in the interfollicular epidermis and mucosa, but rarely in the hair follicles. When it does occur in the hair follicles, it is named candida folliculitis. Candida folliculitis is divided clinically into 3 types: impetigo contagiosa; folliculitis simplex; and tinea barbae-like (2). We present here a case of Candida folliculitis mimicking tinea barbae.

CASE REPORT

An 87-year-old Japanese man presented with a 3-month-history of indurated erythemas and papules on the bearded areas of his upper and lower lips. His medication consisted of a topical ointment (which consisted of betamethasone valerate and gentamicine) and the oral administration of a broad-spectrum antibiotic (cefcape pivoxil hydrochloride) for 2 months, as prescribed by his doctor. During treatment, however, the patient's eruption deteriorated and become painful and tender, and pustules arose in the lesion. The patient's past history included a 15-year course of oral administration for diabetes mellitus and a wide resection for squamous cell carcinoma on his face 11 years before. A physical examination showed diffuse erythematous swelling with crusted papules and papulopustules on the bearded areas of his upper and lower lips. The bearded hairs became sparse and tender, and there were purulent discharges from the hair orifice under pressure. Smears from the crust revealed no fungal elements. A biopsy specimen from the lesion of the upper lip showed a prominent subcorneal abscess and an intense neutrophilic infiltrate through the dermis. The epidermis showed hyperkeratosis and acanthosis. The dermal infiltrates were composed of many neutrophils, macrophages with faintly vacuolated cytoplasm, lymphocytes and eosinophils. The deep dermis showed the granulomatous reaction including foreign-body giant cells mixed with other inflammatory cells, such as neutrophils and lymphocytes. Proliferated and elongated capillary vessels were also seen in the vicinity of the granulomatous reaction. The infiltrate of neutrophils was seen in the epidermis, follicular epidermis and eccrine dermal ducts and glands. Prominent lymphatic ectasia were also seen in the upper dermis. Fungal elements were also demonstrated in the stratum corneum and the crusts, but not in the dermis and follicular epidermis, by periodic acid-Schiff staining of the sections. The fungal elements consisted of pseudohyphae and ovoid spores.

Candida albicans was isolated from both the biopsy specimen and from the purulent discharges from the follicles. No other fungus or bacterium were cultured. The results of routine laboratory studies showed diabetes mellitus; hyperglycaemia (glucose in hunger: 200 mg/dl) and elevation of HbA1c (7.5%). Candida antigen could not be detected in the serum. Delayed-type skin reaction to *Candida albicans* antigen and tuberculin showed normal responses. The test for normal lymphocyte transformation after stimulation with *Candida albicans* antigen was normal. The serum level of IgE for *Candida albicans* was normal. Antibodies against the human immunodeficiency virus could not be detected. We diagnosed candida folliculitis. Subsequently, oral therapy with itraconazole (50 mg/day) and topical therapy with sulconazole were initiated. Four weeks later, the symptoms mentioned above began to gradually fade. This therapy was effective without

scarring or aberrant effects, and it was discontinued 12 weeks after initiation. There was no recurrence.

DISCUSSION

The present case showed both clinical and histological characteristics similar to tinea barbae. There are only 3 cases of Candida folliculitis similar to tinea barbae reported in the English literature (3, 4), and all were in healthy patients. A few cases of other types of candida folliculitis, such as folliculitis simplex, acne or perioral dermatitis-like, have also been reported, some associated with other lesions or conditions (5–10). The association has been reported following situations of prolonged intravenous hyperalimentation (5) and hypothyroidism (8). Candida folliculitis has also occurred in systemic candidiasis. Predisposing factors may also be present, such as diabetes mellitus and the general or local impairment of immune defences. Oral thrush and enteral candidosis may also be the source of infection (2). Anticandidal resistance is associated with the *in vivo* occurrence of strong delayed-type hypersensitivity (DTH) to Candida, the accumulation of neutrophils, the detection of highly fungicidal effector macrophages, and the presence in the serum of Candida-reactive antibodies (11, 12). It is also clear that although phagocytosis occurs at similar levels in diabetics and non-diabetics, killing of Candida by the diabetic neutrophil is impaired under conditions of hyperglycaemia and ketosis (13). Our patient had diabetes mellitus, which was poorly controlled under medication, and had undergone long-term treatment of a perioral skin lesion with topical glucocorticoids and oral antibiotics. These factors may have played significant roles in forming the Candida folliculitis mimicking tinea barbae. In fact, an experimental study has shown that topical application of corticosteroids dramatically shifts the host-parasite relationship in favour of Candida with the deletion of intraepithelial CD4+T cells (14). In collected cases of Candida folliculitis, only 3 cases, including ours, underwent a Candida skin test, and all were positive (3, 10). In addition to this, 2 cases, including ours, underwent a lymphocyte transformation after stimulation with *Candida albicans* antigen, and both were normal (3). Although the histology of the acanthotic epidermis, a dense accumulation of neutrophils and the granulomatous response in the dermis are compatible with Candida granuloma in chronic mucocutaneous candidiasis (15), the results of the DTH and the lymphocyte function confirmed that Candida folliculitis is different from chronic mucocutaneous candidiasis and is categorized as acute mucocutaneous candidiasis.

In the present case, bulky accumulations of neutrophils were detected in the subcorneal region and throughout the dermis. Three possible mechanisms for this phenomenon exist: (i) a direct activation of the alternative complement pathway by the organisms in the lesions; (ii) direct chemotactic activity in components of *Candida albicans* (11); (iii) a secretion of cytokines with chemotactic activity. The last mechanism is explained by the following events: (i) protective antifungal immunity is associated with a predominant Th1 response (16); (ii) corticosteroids enhance the capacity of macrophages to induce Th2 cytokines in CD4+

lymphocytes (18); and (iii) these types secrete a granulocyte-macrophage-colony stimulating factor which is chemotactic for neutrophils (17). Both Th1 and Th2 responses have been observed in an experimental *Candida albicans* infection in mice (12). It seems that in our patient with *Candida* folliculitis a similar phenomenon occurred in the lesion.

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