and CRP are common to occur. Moreover, on histology there were numerous neutrophils in the dermis, which is a histological feature of Sweet's syndrome. Also steroid is quite effective in both the diseases; hence one might get deceived by the improvement of patient's symptoms. Anesthesia over cutaneous lesions and enlarged and tender peripheral nerves point towards leprosy; and AFB positive bacilli on SSS and specific histopathologic findings aid in confirming the diagnosis. In our patient thickened and tender nerves and typical histology clinched the diagnosis of ENL. Interestingly, in our case, the reactional lesions were the presenting symptoms, as patient did not have a preceding diagnosis of leprosy, whereas most reported cases mimicking Sweet's syndrome have prior diagnosis of BL or LL.[1,3–5]

Corticosteroid is the drug of choice in T2LR, whereas thalidomide is the drug of choice for severe ENL. MDT should be started immediately or should be continued if a patient is already taking it. Corticosteroids suppress the inflammatory immune response to *M.leprae* antigens and also reduce intraneural and cutaneous edema.[1,3–5]

Awareness of Sweet's syndrome like T2LR and its prompt diagnosis and treatment is essential to prevent its dreaded sequelae in potentially treatable cases.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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Discoid Lupus Erythematosus in a Patient With Alopecia Totalis☆

Lupus eritematoso discoide en una paciente con alopecia totalis

Dear Editor:

We herein describe a rare case of discoid lupus erythematosus (DLE) in a patient with alopecia totalis.

A 42-year-old female developed alopecia on the scalp, which worsened and involved all areas of the scalp 10 years previously. She received various treatments, such as topical corticosteroids, topical carpronium chloride, oral prednisolone, intra-lesional triamcinolone acetonide, and cryotherapy, for five years without success, and thus discontinued therapy a few years previously. She visited the dermatology clinic at Hanawa Kousei Hospital, complaining of asymptomatic facial erythemas which appeared one year previously. Physical examination revealed infiltrative scaly erythemas on the cheek, nose, lips, and upper back (Fig. 1a,b). Total alopecia of the scalp was also observed (Fig. 1a). Her eyebrows fell out, while the eyelashes remained intact. Laboratory examination showed positive anti-nuclear antibody (ANA) (1:1280, speckled), whereas other data such as complete blood count, liver and renal function, serum complement levels, anti-double strand DNA antibody, anti-Sm antibody, anti-SS-A antibody, anti-SS-B antibody, and antiphospholipid antibody were all within normal range. A biopsy specimen taken from the cheek revealed individual cell keratinization, liquefaction of epidermal basal membranes, and focal mononuclear cell infiltration in the dermis (Fig. 1c). Examination by direct immunofluorescence showed linear deposition of IgG, IgM, and C3. A diagnosis of DLE was made. Facial and back erythemas much improved by oral hydroxychloroquine (Plaquenil®, Sanofi, Swiss) (200 mg and 400 mg per alternate day) six months later; however, her alopecia remained unchanged.

Our patient suffering alopecia totalis developed DLE with nearly nine years' interval, and alopecia was already stable when the DLE lesions appeared. Her scalp alopecia was non-scarring without erythema, and was therefore not identified as lupus alopecia, although biopsy was not carried out.

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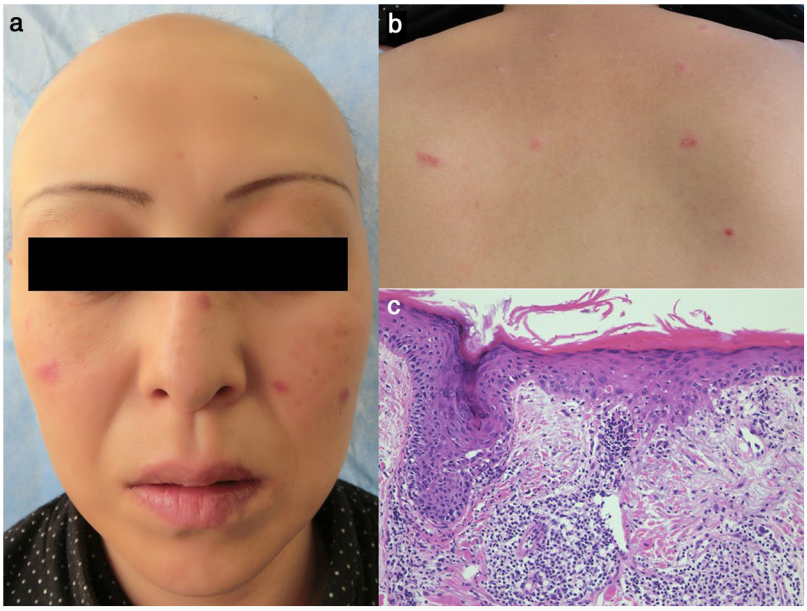

Figure 1 (a) Alopecia totalis on the scalp and erythema scattered on the cheek, nose, and lips. (b) Infiltrative erythemas on the upper cheek. (c) A biopsy specimen showing epidermal atrophy, liquefaction of the basement membrane, dyskeratotic epidermal cells, and focal mononuclear cell infiltration in the dermis.

Alopecia areata is sometimes associated with other autoimmune or allergic diseases, and there has been increased incidence of alopecia areata and lupus erythematosus.[1,2] To date, several cases of DLE in association with alopecia have been reported.[3,4] Among patients with severe types of alopecia such as alopecia totalis and universalis, thyroid disease was most prevalent, followed by vitiligo, diabetes, atopic dermatitis, dyslipidemia, hypertension, psoriasis, and internal malignancy.[5] However, to our knowledge, co-existence of DLE and alopecia totalis has not yet been reported. The present case showed a high titer of ANA (1:1280), suggesting that immunological abnormalities may have developed the DLE. She did not have any other specific autoantibodies, or other symptoms suggestive of systemic autoimmune diseases. Regarding the pathogenesis of alopecia areata, Th2 type cytokine predominance is seen in localized type while Th1 predominance in generalized type.[2] Furthermore, recent findings suggest an important role of IL-17 in alopecia;[6] however, the role of Th17 cells in DLE remains unclear. The cutaneous inflammatory infiltrates are dominated by Th1, but not Th17 cells, in the DLE lesion.[7] A genome-wide study has suggested that several genomic regions are significantly associated with alopecia areata, lupus erythematosus, and other autoimmune diseases. Such immune-mediated imbalance may have contributed to the development of alopecia totalis and DLE in our case. The facial lesions of our patient were eventually improved with hydroxychloroquine, which did not show any effects on the alopecia lesions.

Conflict of interests

The authors declare that they have no conflict of interest.

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Cryptococcoid Sweet Syndrome: A Clinical and Histologic Imitator of Cryptococcosis☆

Síndrome de Sweet criptococoide: simulador de criptococosis tanto clínica como histológicamente

To the Editor:

Cryptococcoid Sweet syndrome is a term coined recently by Wilson J et al[1] to describe a rare variety of Sweet syndrome, which has a histologic profile that is indistinguishable under hematoxylin and eosin staining from gelatinous cryptococcosis. Diagnosis is established by positivity for staining with myeloperoxidase in the presence of negative stains for fungi. To date, 8 cases of neutrophilic dermatosis have been reported with these histopathology characteristics, all of them in dermatopathology journals.[1–4] We describe a new case of cryptococcoid Sweet syndrome, where infection by *Cryptococcus* spp was considered not only because of the histopathology profile but also because of the clinical characteristics of the skin lesions.

Case Description

An 18-year-old woman presented a history of episodes of different neutrophilic dermatoses (pyoderma gangrenosum and Sweet syndrome) between the ages of 6 and 12 years. She subsequently developed antineutrophil cytoplasmic antibody (ANCA)-positive vasculitis, with terminal kidney failure and malignant hypertension, causing her to be put on hemodialysis. The patient was undergoing treatment with azathioprine, prednisone, losartan, valaciclovir, and trimethoprim–sulfamethoxazole. Forty-eight hours after undergoing magnetic resonance angiography, she presented molluscum-like lesions on the face (Fig. 1) and blisters and erythematous lesions on the backs of the hands, accompanied by fever and neutrophilia of 9910 neutrophils/mm^3 (82.5% leukocytes). An adverse reaction to the contrast medium was suspected and boluses of methylprednisone were administered; the patient was assessed by the dermatology department. An infectious process (cryptococcosis, tuberculosis, histoplasmosis, etc.) was suspected and biopsies were performed and microbiology samples were taken. The lesions resolved after a few days and the patient remained lesion-free and in excellent general health.

Histology revealed a dermal inflammatory infiltrate with neutrophils, vacuolated spaces, and yeast-like structures

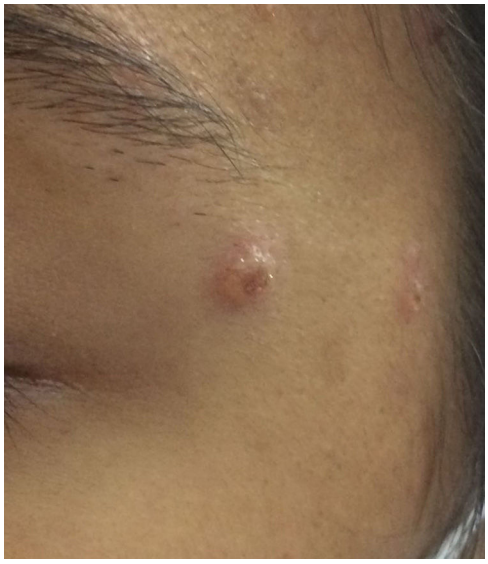

Figure 1 Molluscum-like lesion on the face.

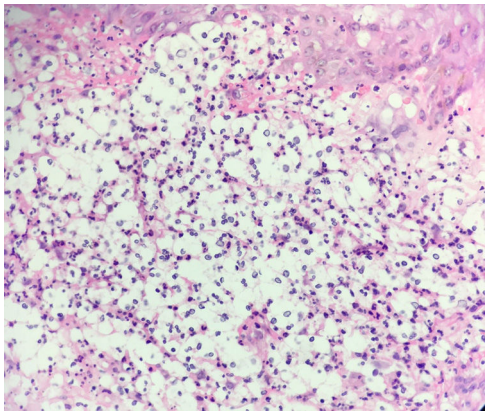

Figure 2 Biopsy of molluscum-like lesion on the face showing vacuolated spaces, neutrophils, and yeast-like structures that appear to have a capsule, as is seen in gelatinous cryptococcosis (hematoxylin–eosin, ×400).

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