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Supporting Information

Additional Supporting Information may be found online in Supporting Information:

Table S1. Reported cases of lupus panniculitis showing a good response to Rituximab.

doi: 10.1111/ajd.12709

Case Letter

Dear Editor,

Fluconazole-induced Sweet's syndrome: A novel association

A 22-year-old woman presented with multiple, smooth, round, red-to-brown oedematous patches and plaques on palms and both wrists, as well as on her buttocks and vulval area (Fig. 1). She reported lesion tenderness, generalised malaise, arthralgia and intermittent fevers. There was no conjunctivitis or lymphadenopathy. All lesions appeared within hours of ingestion of a single fluconazole capsule which she was taking to treat her vulvovaginal candidiasis. She reported a strikingly similar eruption over the preceding 5 years in the same locations on three separate occasions associated with fevers, each time following the ingestion of an oral fluconazole capsule. The initial episode occurred 2 days after ingesting fluconazole; subsequent

episodes occurred within 4 h of ingesting fluconazole. Given the morphology and temporal association with a drug, the major differential diagnoses included fixed drug eruption, Sweet's syndrome and drug-induced urticaria.



Figure 1 Multiple, red-to-brown, round, oedematous dermal plaques on the palms bilaterally of a 22-year-old woman.

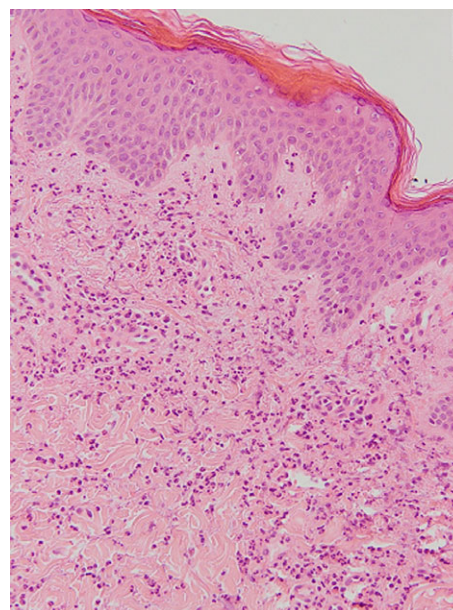


Figure 2 HE stain from a punch biopsy from the left buttock showing minimal spongiosis in the epidermis with no significant interface damage. Within the dermis there is a prominent interstitial infiltrate of neutrophils with associated oedema and rare eosinophils, without evidence of leukocytoclastic vasculitis, consistent with Sweet's syndrome (x200).

Conflict of interest: none.

Two punch biopsies from the left buttock revealed a dermal neutrophilic infiltrate with oedema and without evidence of leukocytoclastic vasculitis, consistent with Sweet's syndrome (Fig. 2). Fixed drug eruption was considered unlikely as histopathology did not show an interface dermatitis. The presence of fever, arthralgia and the characteristic histopathological findings favoured a diagnosis of Sweet's syndrome.

A full blood examination, blood film morphology, serum protein electrophoresis, renal function, liver function and C-reactive protein were within normal limits. Underlying malignancy was also considered unlikely as there were no B symptoms or paraneoplastic symptoms or signs. Her pap smears were up to date and normal. An infectious screen was negative (urine microscopy, culture and sensitivity, anti-streptolysin and Anti-DNase B titres, screen for viral hepatitis B and C and human immunodeficiency virus). The discontinuation of fluconazole, and the commencement of oral prednisolone (37.5 mg tapered over 1 month) and topical betamethasone dipropionate 0.05% resulted in the resolution of the cutaneous lesions.

Sweet's syndrome is characterised by an acute onset of tender erythematous plaques or nodules, with associated histopathological findings of a dense neutrophilic dermal infiltrate. Fever, malaise and leukocytosis often accompany the cutaneous features.¹ Sweet's syndrome may be classified as classical (idiopathic), malignancy-associated or drug-induced. The most commonly implicated drugs are granulocyte-colony stimulating factor and all-*trans* retinoic acid.^{1,2} Leukocytosis may be absent in the drug-induced form.³ Notably, neither oral fluconazole nor vulvovaginal candidiasis have been reported to be associated with Sweet's syndrome.

Walker and Cohen suggest five diagnostic criteria for drug-induced Sweet's syndrome: (i) abrupt onset of painful erythematous plaques or nodules, (ii) histopathological evidence of a dense neutrophilic infiltrate without evidence of leukocytoclastic vasculitis, (iii) fever >38°C, (iv) temporal relationship between drug exposure and clinical presentation or temporally related recurrence after oral rechallenge, and (v) temporally related resolution of lesions after drug withdrawal or treatment with systemic corticosteroids.³ These criteria were fulfilled in our patient.

The temporal relationship between commencing fluconazole and the abrupt onset of cutaneous symptoms, characteristic histopathological findings, rapid response to oral corticosteroids and a history of multiple recurrences associated with fluconazole exposure support a diagnosis of fluconazole-induced Sweet's syndrome. According to Naranjo and colleagues' validated adverse drug probability scale, fluconazole would be assigned to the probability category of 'definite'.⁴ There has been one previous report of Sweet's syndrome induced by ketoconazole.⁵

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doi: 10.1111/ajd.12689

Case Letter

Dear Editor,

A rare cause of alopecia: Lipedematous alopecia

A 31-year-old woman presented with a 3-month history of hair loss. This began with a decreased rate of hair growth, followed by the gradual swelling of the scalp. She was initially diagnosed with alopecia areata and treated with intralesional triamcinolone. A dermatological examination showed a 6 × 6 cm oval patch of alopecia with a spongy texture (Fig. 1a). A hair traction test was negative. She had a positive antinuclear antibody (granular pattern), but a rheumatological evaluation was normal. Ultrasonography showed that the scalp was 12.5-mm thick (Fig. 1b). Histopathology revealed a sparse, perivascular lymphocytic infiltrate and a dramatically expanded subcutaneous (s.c.) layer, prominently diminished follicles, lymphangiectasias and mild perifollicular infiltration (Fig. 2a,b). The subcutaneous layer measured 5 mm in thickness. Based on the clinical, ultrasonographic and histopathological findings, a diagnosis of lipedematous alopecia was made and the patient was commenced on hydroxychloroquine (200 mg/day). After 3 months of treatment there was minimal improvement.

Lipedematous alopecia is a rare disease characterised by an acquired, nonscarring alopecia with a thickened scalp due to the increased thickness of subcutaneous fatty tissue. It usually starts from the vertex and extends peripherally.¹

Conflict of interest: none.