the five haemorrhaging fingernails had reverted to normal. An adverse drug reaction (ADR) was rated as ‘probable’ (6 points) according to the Naranjo ADR Probability Scale [imputability decision scale ranging from ‘doubtful’ (0), to ‘possible’ (1–4), ‘probable’ (5–8) and ‘highly probable’ (9)].

Sports, housework, gardening, or playing percussion instruments are the usual traumatic causes of splinter haemorrhages. It may also be a manifestation of certain systemic diseases such as subacute bacterial endocarditis, antiphospholipid syndrome, cholesterol crystal embolization, haemodialysis and peritoneal dialysis. Several miscellaneous skin conditions causing splinter haemorrhages have been reported, including psoriasis, dermatitis, Sweet’s syndrome and onychomycosis. Although our patient had developed onychomycosis, the target nails had remained unchanged for almost 4 years, and the longitudinal subungual haemorrhages appeared 3 weeks after consecutive administration of terbinafine. In addition, three of the five fingernails reverted to normal within 3 months after changing the treatment. Thus, the imputability score was 6 and ADR probability classification was ‘probable’, and we believe this is a case of secondary splinter haemorrhage probably associated with terbinafine. Although cases have also been reported previously after administration of tetracycline hydrochloride or ganciclovir, our case differs from those in that haemorrhage only presented on the right five target fingernails during the treatment of onychomycosis with terbinafine, and the remaining uninfected fingernails and all toenails were entirely unchanged. The precise aetiology underlying this phenomenon warrants further investigation.

We believe that this man is the first case of splinter haemorrhages probably induced by terbinafine. Such a side-effect should be considered in the selection of antifungal drugs.

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Conflict of interest: none declared.
Accepted for publication 16 June 2005

References

Treatment of recalcitrant hidradenitis suppurativa with oral ciclosporin
doi: 10.1111/j.1365-2230.2005.01983.x

Hidradenitis suppurativa (HS) is a disorder of the terminal follicular epithelium within apocrine-bearing skin. The disease may run an unremitting course and result in significant physical and psychological morbidity. There are several medical and surgical approaches to managing HS, although evidence from randomised controlled trials is scarce. Ciclosporin has been reported anecdotally twice for the treatment of HS: two further cases are described here.

A 38-year-old woman was referred with a 20-year history of recurrent groin abscesses. Previous treatments included multiple surgical-drainage procedures and antibiotics. She was otherwise well. On examination she had large inflammatory lesions on the thighs and in the groin area, with scarring and sinus formation. Clinically the features were consistent with HS. Routine haematological and biochemical tests were normal. She was initially treated with controlled-release minocycline 100 mg twice daily together with a reducing course of oral steroids, starting at prednisolone 30 mg daily. The disease flared once the steroids were discontinued and the patient received a pulse of 500 mg of intravenous methylprednisolone. Despite this, she required surgery to excise areas of inflammation on the right thigh and labia majora, and subsequently received oral clindamycin and clarithromycin and intralesional steroids with no real improvement. Hence ciclosporin was introduced at 4 mg/kg daily with appropriate monitoring. This was well-tolerated, and after 3 months was reduced to 2 mg/kg daily. She has now been on this for 7 months and has had no severe episodes of inflammation during this period.

A 31-year-old man was referred with a 16-year history of severe nodulocystic acne that had failed to respond to 6 months of oral minocycline. He had also undergone previous surgery to excise groin abscesses, which resulted in a chronically discharging wound. On examination, he had florid acne vulgaris on his back (Leeds Acne Grading Scale score of 5) but also inflammatory lesions with scarring and sinus formation in the axillae and groin, which were typical of HS. Again, all baseline haematological and biochemical parameters were normal. He was commenced on 20 mg isotretinoin (0.25 mg/kg daily) and prednisolone 20 mg daily, the latter reducing by 5 mg each week. Over the subsequent 18 months he had four such reducing courses of prednisolone, and lymecycline
was introduced at 408 mg daily, without achieving good control of his HS. Hence ciclosporin was prescribed at 3 mg/kg daily, again with appropriate monitoring. Within 8 weeks he was weaned off prednisolone, as there was a marked clinical response in the HS and the residual acne had cleared completely. Ciclosporin was well-tolerated and was discontinued after 4 months in view of good disease control. The patient remained in remission for 4 months and the subsequent relapse was much milder than the pretreatment disease. The patient then elected to recommence ciclosporin, and had a further rapid response.

The use of ciclosporin in HS has only been reported on two previous occasions. Gupta et al.\textsuperscript{1} treated a 60-year-old man with 6 mg/kg daily for 6 weeks and described a moderate response. Buckley and Rogers\textsuperscript{2} reported the successful use of ciclosporin 4.5 mg/kg daily in the treatment of pyoderma gangrenosum associated with HS, both of which responded fully.

HS is a chronic suppurative disorder, the pathology of which is not fully understood and so it is difficult to know exactly how the calcineurin inhibitor ciclosporin is taking effect. However, ciclosporin is used in the treatment of a number of inflammatory dermatoses, and its ability to suppress cytokine production and T-cell activation is likely to explain the response reported here. It is possible that in these patients the condition remitted as a delayed effect of other treatments but this is unlikely. We believe that these cases illustrate an effective treatment option in this often intractable disease, though obviously randomised controlled trials to evaluate efficacy and safety would be ideal.

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Conflict of interest: none declared.  
Accepted for publication 17 August 2005

References


A sandfly in Surrey? A case of cutaneous leishmaniasis in the United Kingdom without history of recent travel to an endemic area

doi: 10.1111/j.1365-2230.2005.01984.x

A 51-year-old woman presented with a 6-month history of an asymptomatic but persistent 5-mm reddish-brown nodule on her left cheek. She distinctly remembered it starting after an insect bite while she was out walking on the heath at her daughter’s house in Surrey, UK. She had no history of foreign travel and the last time she had been abroad was 4 years previously, when she had visited Majorca, Spain.

Biopsy showed heavy dermal chronic inflammation, including a large number of epithelioid macrophages heavily parasitized with leishmanial organisms (haematoxylin and eosin, magnification × 400).

Two months later she was well and remained symptom-free for 9 months. Thereafter, she had a rapid relapse and was restarted on ciclosporin 1.5 mg/kg daily, which resulted in clear improvement within 6 weeks. She then elected to stop ciclosporin because of the side effects of renal impairment and hypertension.

The patient feels certain that the lesion started with an insect bite to her cheek but that implies a sandfly in Surrey. How could it get there and how could it survive?

The patient was staying exactly equidistant from Heathrow and Gatwick airports, at a distance of approximately 25 miles. Could this be so-called ‘airport leishmaniasis’ with the sandfly transmitted in the baggage? This unusual