Erythema annulare centrifugum in a patient with chronic myeloid leukaemia on ponatinib

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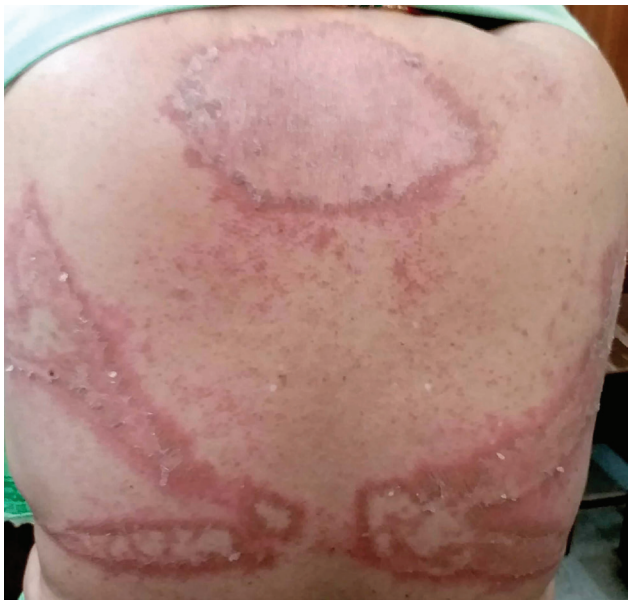
Skin has often been a window to systemic diseases, with skin manifestations ranging from nonspecific rashes to specific lesions. A 55-year-old female from central Nepal with chronic myeloid leukaemia (CML) who was being treated with ponatinib, presented to the Department of Dermatology with an 8-week history of pruritic erythematous lesions over her chest, back, thigh and calves, initially starting at the upper back. She was diagnosed to have CML 14 years ago, and received treatment with imatinib initially. Two years ago, she was switched to ponatinib due to imatinib resistance.

Examinations revealed wide annular polycyclic plaques in the trunk, thigh and calves (Figure 1). Screenings for fungal infections, syphilis, hypothyroidism and connective tissue disorder were all negative. The skin biopsy showed mild spongiosis with focal keratosis in epidermis and perivascular infiltration by lymphocytes and histiocytes in superficial dermis (Figure 2), suggestive of erythema annulare centrifugum (EAC). EAC is a rare chronic cutaneous disease characterised by erythematous and violaceous, arcuate and annular plaques usually involving the trunk or lower extremities. EAC has been associated with different conditions, including infections, food allergy, drug reactions and malignancies.[1]

Treatment and eradication of the underlying disease often resolves EAC but this was not possible in our patient. She was initially prescribed with cream clobetasol propionate 0.05% for local application and cetirizine for her pruritus, but her lesions progressed. She was then managed with low-dose prednisolone (0.5 mg/kg) for 1 week, gradually tapered over

Figure 1 Wide annular polycyclic erythematous plaques on the back



1 month, which resulted in marked improvement. Ponatinib was continued for CML.

The diagnosis of EAC should be followed by a full physical examination and diagnostic workup to exclude an underlying disorder. However, sometimes no causative agent can be identified.[2] In our patient, the EAC appeared years after the diagnosis of CML but it was present after switching to ponatinib from imatinib. Hence, it seems logical to

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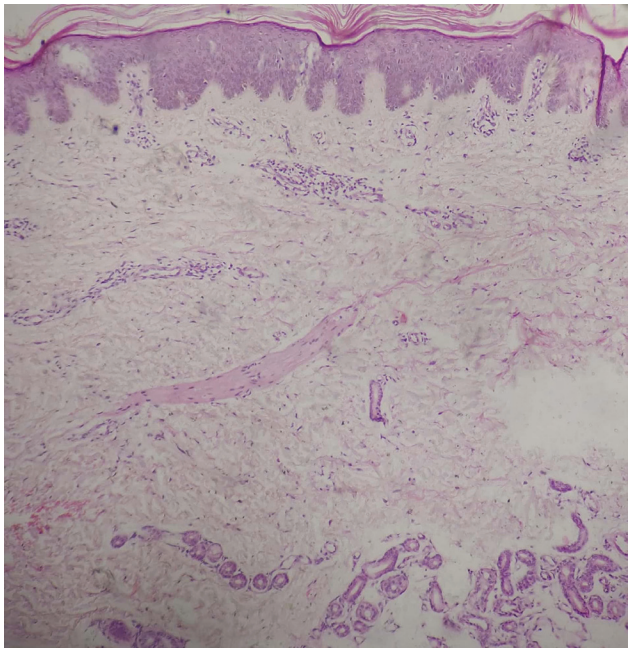
Figure 2 Histopathological examination of skin lesion biopsy showing mild spongiosis with focal keratosis in epidermis, and perivascular infiltration by lymphocytes and histiocytes in superficial dermis



deduce that the use of ponatinib might have triggered the development of these lesions. However, in the present case, whether it was due to the CML or the use of ponatinib remains speculative.

References

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