

CASE REPORT

Calcipotriol for erythema annulare centrifugum

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Summary

Erythema annulare centrifugum (EAC) is an uncommon inflammatory skin disease of unknown aetiology. No therapy is currently available. We describe a 73-year-old woman with a 3-year history of EAC that was resistant to topical and systemic glucocorticoids, antifungals, and psoralen plus ultraviolet A treatment. After 3 months of treatment with topical calcipotriol the lesions cleared completely and did not recur during a 6-month follow-up period. Vitamin D analogues may be of value in the therapy of EAC.

Key words: calcipotriol, erythema annulare centrifugum

Erythema annulare centrifugum (EAC) is an uncommon inflammatory skin disease consisting of polycyclic and annular lesions with tiny collarettes of scaling at their peripheries. Histologically it resembles pityriasis rosea, having a superficial perivascular lymphocytic infiltrate with moderate epidermal acanthosis, focal spongiosis and parakeratosis. Although the aetiology and pathogenesis are unknown, EAC may in some cases be triggered by drugs, bacterial infections (cystitis, appendicitis, tuberculosis), fungi (*Candida*, tinea), viruses (Epstein–Barr virus, molluscum contagiosum), parasites (*Ascaris*), autoimmune diseases (Sjögren's syndrome, hyperthyroidism), neoplasms (mainly Hodgkin's disease), liver diseases and dysproteinemias.¹ EAC must be differentiated from erythema figuratum (also called the deep form of EAC) and other morphologically similar but unrelated skin diseases (annular psoriasis, Lapière–Milian type annular centrifugal psoriasis, lupus erythematosus, bullous diseases, sarcoidosis, fungal infections).^{1,2}

Case report

A 73-year-old woman with a 12-year history of ischaemic heart disease, treated with diuretics, digoxin and nitrates, was referred because of a 3-year history of

pruritic annular lesions localized on the chest, arms and legs (Fig. 1a,b). Some lesions (e.g. the lesion on the chest shown in Fig. 1) persisted on the skin during the entire disease period whereas other elements, especially some lesions on the extremities, had a tendency for spontaneous exacerbations and resolutions over a period of several months. There was no previous personal or family history of skin disease. Before admission, her skin condition had been treated unsuccessfully with local and systemic glucocorticoids and fluconazole. Routine laboratory investigations including blood tests, serum electrophoresis, culture for fungi and bacteria from skin lesions and faeces, and antinuclear, SSA(Ro)/SSB(La) and *Borrelia* antibodies were negative. Chest X-ray showed mild cardiomegaly; abdominal ultrasound was unremarkable. Histological examination of skin lesions revealed a slightly acanthotic epidermis with discrete focal spongiosis and parakeratosis. There was a superficial lymphocytic infiltrate in the dermis with extravasation of erythrocytes. Lapière–Milian type annular centrifugal psoriasis was excluded on the basis of clinical features (lack of pustulation) and absence of intraepidermal neutrophils or subcorneal pustulosis on histology.³ The diagnosis of idiopathic EAC (superficial type) was made and our patient was treated with potent local glucocorticoid ointments for 6 months combined with a 6-week course of oral psoralen plus ultraviolet A phototherapy (total dose 23 J cm⁻²) with no improvement. Finally,

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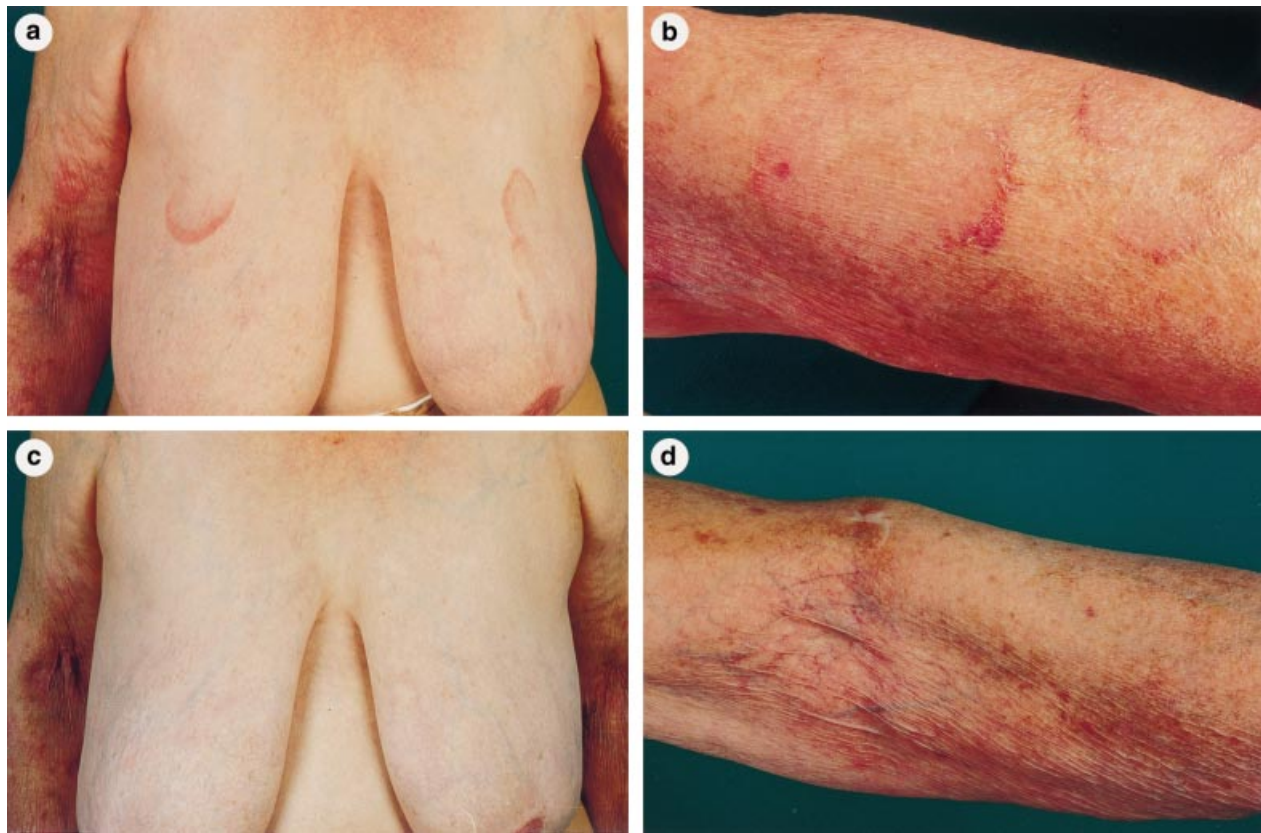


Figure 1. A 73-year-old woman with erythema annulare centrifugum on the chest (a) and under surface of the arms (b) before (a,b) and after (c,d) topical therapy with calcipotriol.

calcipotriol ointment $50 \mu\text{g g}^{-1}$ (Daivonex[®]) once daily was instituted as a monotherapy for the lesions on the chest and arms but not on the lower extremities. After 3 months of treatment, the treated lesions cleared completely (Fig. 1c,d) but the untreated lesions on the legs persisted. Our patient reported a burning sensation and the augmentation of pruritus at the beginning of therapy, which gradually subsided over 3 weeks. The lesions in the treated areas did not recur during a 6-month follow-up period after the discontinuation of calcipotriol therapy, whereas the untreated lesions on the lower extremities persisted. However, the latter lesions did not cause any discomfort and our patient did not desire calcipotriol treatment for these.

Discussion

EAC is an often self-limiting disorder that resolves spontaneously over several weeks. However, in some individuals, such as the patient described here, the disease may persist for many years.¹ No routinely effective therapy is currently available unless a

triggering factor can be identified and eliminated. This report indicates that vitamin D analogues may be of value in the therapy of EAC. The rationale for treating our patient with calcipotriol was that vitamin D analogues inhibit keratinocyte hyperproliferation, regulate epidermal differentiation, and in some situations inhibit cutaneous inflammation,^{4,5} the processes that are likely to contribute to the development of skin lesions in EAC.

The results of therapeutic trials on single patients are often difficult to interpret because of the possibility that the observed response reflects the natural evolution of the disease rather than the effect of the medication. However, in the present case it is likely that a real therapeutic effect took place because the untreated lesions persisted during the treatment and follow-up periods. Moreover, the disease had a chronic course and was unresponsive to aggressive treatment with glucocorticoids and phototherapy, which also makes unlikely (but does not rule out) a possible placebo effect of calcipotriol. Clinical trials are needed to confirm the efficacy of vitamin D analogues for EAC.

References

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