

Case report

Erythema annulare centrifugum in association with hypothyroidism: a case report

Zahida Rani, *Tariq Rashid, Ijaz Hussain

Department of Dermatology, King Edward Medical College/Mayo Hospital, Lahore.

*Department of Dermatology, Allama Iqbal Medical College/Jinnah Hospital, Lahore.

Abstract Erythema annulare centrifugum is a type of annular erythema with a multifactorial etiology. We report a case of EAC in association with hypothyroidism, an association hitherto unreported.

Introduction

Erythema annulare centrifugum (EAC) is a type of annular erythema with multifactorial etiology including infections, drugs, pregnancy, autoimmune disorders and neoplasia.^{1,2} In a large number of cases no underlying cause is detectable.

Hypothyroidism is a common endocrinopathy endemic in certain regions of the world including Pakistan. It can be associated with many skin changes like rough and dry skin, carotenoderma, acquired ichthyosis, alopecia, madarosis, and slow nail growth.¹

Search on the MEDLINE (1966-2002) and local data (search words: erythema annulare centrifugum, hypothyroidism, thyroid disease) showed only three cases of EAC in association with thyroid disease, one each with hyperthyroidism,³ Hashimoto's thyroiditis⁴ (in French literature) and Graves' disease.⁵ To the best of our knowledge, hypothyroidism has never been reported in association

with EAC in the medical literature. Herein, we report a case of EAC in association with hypothyroidism.

Case report

A 60-year-old female presented at the department of dermatology, Mayo Hospital, Lahore, Pakistan with the complaint of multiple, pruritic, non-evanescent, erythematous, slightly indurated annular lesions over her trunk and limbs for the last 4 months. She also complained of diffuse hair loss. In addition, she had cold intolerance, weight gain and anorexia. There was no history of photosensitivity, joint pains, or any drug intake. Rest of the systemic inquiry was unremarkable.

General physical examination of the patient revealed she had a regular pulse with a rate of 70/min, a blood pressure of 110/70mmHg and a normal temperature and respiratory rate. Her skin was dry and diffusely yellow in colour. Examination of the lesions revealed multiple annular, polycyclic, erythematous, slightly scaly lesions with hypopigmented centre scattered over the trunk (**Figure 1**) and limbs. Diffuse palmoplantar keratoderma, non-scarring alopecia over scalp, madarosis and ichthyosis of the lower limbs were also present. Nails and

Address for Correspondence

Dr. Zahida Rani,
Dermatology Department,
Mayo Hospital, Lahore.
Email: zahidaraffad@yahoo.com



Figure 1 Erythematous annular plaque mild scaling

mucosal surfaces were normal. No sensory loss was seen. Motor system examination was also unremarkable. No abnormality was detected in any other system.

KOH examination of the scales for fungal hyphae was negative. Biopsy taken from the edge of the lesion revealed hyperkeratosis, mild acanthosis and spongiosis (**Figure 2**). A sparse perivascular mononuclear infiltrate was seen. Routine investigations including complete blood counts, peripheral blood film, ESR, blood urea, liver function tests, blood sugar, urine examination, and X-ray chest were normal. Abdominal ultrasonography didn't reveal any visceromegaly, mass or lymph node enlargement. Similarly, antinuclear antibodies, anti-Ro antibodies and VDRL test were negative. Thyroid function tests showed reduced T3 level, 9pmol/l (normal range 11-22 pmol/l), but normal T4 level, 2.25 pmol/l (normal range 1.1-3.00 pmol/l) while TSH, 7.36mu/l (0.30-4.0 mu/l) was raised. On these grounds she was labelled as a case of EAC secondary to hypothyroidism. She was prescribed topical steroids and emollients for symptomatic relief and referred to thyroid clinic for the management of hypothyroidism where she was put on thyroxin replacement 0.5 mg once daily. At three weeks follow-up, her cutaneous

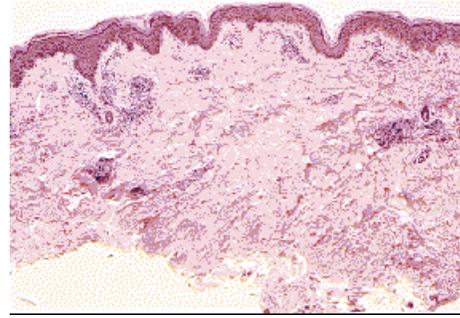


Figure 2 Hematoxylin-eosin stained section shows hyperkeratosis, mild acanthosis and spongiosis with sparse perivascular infiltrate, (original magnification x 40)

lesions had subsided with post inflammatory hypo-pigmentation.

Discussion

EAC is a reactive inflammatory vascular dermatosis belonging to the poorly defined category of figurate erythemas. It is characterized by slowly migrating annular or configurate erythematous lesions.^{1,2} Extension occurs slowly, sometimes in weeks or months. The lesions may be present on any part of the body although buttocks, thighs and upper arms are more commonly involved. The lesions may resolve spontaneously or wax and wane over many years.^{1,2}

Histopathologically, EAC can be divided into two types; superficial and deep.⁶ In the superficial type, there is spongiosis, acanthosis, and parakeratosis with a lymphohistiocytic perivascular infiltrate and sometimes upper dermal edema. Clinically, this is the type which presents as flat to slightly indurated erythema, usually with scaling. No epidermal changes are seen in the deep type. It shows lymphohistiocytic perivascular infiltrate of the superficial and deep dermis and sometimes papillary dermal edema. This

type shows indurated erythema without scaling or vesiculation.

The differential diagnosis of EAC include other figurate erythemas^{1,2,6} e.g. erythema gyratum repens, erythema chronicum migrans, annular urticaria, erythema multiforme, tinea corporis, annular psoriasis, annular subacute lupus erythematosus, autoimmune blistering disease, sarcoidosis and secondary syphilis. A detailed history, thorough clinical examination and certain simple investigations will help to distinguish these diseases from EAC.

The exact etiology and pathogenesis of EAC is unknown. It has been hypothesized that the lesions are a result of interaction between inflammatory cells, their mediators and ground substance when foreign antigens penetrate the skin.⁶ Different infections e.g. dermatophytosis,⁶ candida,⁶ molluscum contagiosum,⁷ Epstein-Barr virus,⁶ ascariasis,⁶ inhalants, autoimmune disorders, pregnancy,⁸ sarcoidosis,⁹ liver disease,¹⁰ internal malignancies e.g. Hodgkin's disease, dysproteinemia, blood dyscrasia⁶ including acute myeloblastic leukemia and medications⁶ like ampicillin, cimetidine, amitriptyline, spironolactone, piroxicam, hydroxychloroquine and thiacetazone have been implicated as the causative factor of EAC. Although, thyroid disease has been reported as an association, hypothyroidism has not been reported along with EAC. Whether this is a chance occurrence or shares some common pathogenesis remains to be determined.

References

1. Dowd PM, Champion RH. Disorders of blood vessels. In: Champion RH, Burton JL, Burns DA, Breathnach SM, eds. *Textbook of dermatology*. 6th edn. Oxford: Blackwell Science; 1998. p. 2073-97.
2. Burgdorf WHC. Erythema annulare centrifugum and other figurate erythemas. In: Freedberg IM, Eisen AZ, Wolff K *et al.*, eds. *Fitzpatrick's Dermatology in general medicine*. 5th edn. New York: McGraw Hill; 1999. p. 1149-52.
3. Launay D, Blanc D, Paris B *et al.* Erythema annulare centrifugum disclosing hyperthyroidism (French). *Ann Dermatol Venereol* 1988; **115**: 721-3.
4. Thess F, Rigon JL, Cuny JF *et al.* Erythema annulare centrifugum and Hashimoto's thyroiditis (French). *Ann Dermatol Venereol* 1986; **113**: 1087-8.
5. Braunstein BL. Erythema annulare centrifugum and Grave's disease. *Arch Dermatol* 1982; **118**: 623.
6. Kim KJ, Chang SE, Choi JH *et al.* Clinicopathologic analysis of erythema annulare centrifugum. *J Dermatol* 2002; **29**: 61-7.
7. Furue M, Akasu R, Ohtake N, Tamaki K. Erythema annulare centrifugum induced by molluscum contagiosum. *Br J Dermatol* 1993; **129**: 646-7.
8. Rosina P, D'Onghia FS, Barba A. Erythema annulare centrifugum and pregnancy. *Int J Dermatol* 2002; **41**: 516-7.
9. Altomare GF, Capella GL, Frigerio E. Sarcoidosis presenting as erythema annulare centrifugum. *Clin Exp Dermatol* 1995; **20**: 502-3.
10. Tsuji T, Kadoya A. Erythema annulare centrifugum associated with liver disease. *Arch Dermatol* 1986; **122**: 1239-40.