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Case Report

# Erythematic Annulare Centrifugum: A Rare Case Report in Association with Coronary Artery Disease

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### ABSTRACT

Erythema annulare centrifugum is one of the figurate erythemas, where majority of cases are idiopathic. Some cases are associated with internal diseases and infections, though the causal association has not been definitely proved. This clinical reaction pattern commonly occurring on the trunk and proximal extremities is characterized histopathologically by dermal perivascular lymphohistiocytic infiltrate. We hereby report, a rare clinical presentation of erythema annulare centrifugum, sparing the trunk, along with its rare association with coronary artery disease.

*Keywords:* Erythema annulare centrifugum, coronary artery disease, parakeratosis, etanercept.

## **INTRODUCTION**

Ervthema annulare centrifugum (EAC) is one of the chronic annular and figurate erythemas. In a large majority of cases the aetiology remains obscure even after prolonged observation and investigation. A broad range of associations has been described as causes of EAC. Though some associations that have been described may be coincidental, and a causal association could not always be proven, search for an underlying cause should be done to exclude any foci of infection, internal diseases and neoplasms. <sup>[1,2]</sup> We report a rare association of EAC with coronary artery disease.

## **CASE REPORT**

A 54-year-old male presented with 3

years history of recurrent eruptions of multiple, annular, non pruritic, erythematous scaly lesions; starting on lower legs and evolving to involve bilateral thighs, buttocks, left upper arm and pubic region; sparing the trunk, mucosa, scalp, face, palms and soles. The crops of lesions began as small red firm papules which slowly enlarged over 3-4 weeks to large (10-15cms) rings showing central flattening and fading with peripheral scaling and erythema, which then resolved slowly over 4-5 weeks. leaving behind post inflammatory pigmentary changes and loss of hair. The lesions recurred after a period of 1-2 months. No family history of similar lesions. He gave history of treatment for coronary artery disease since 3 years. On further history taking, it was found that, first appearance of the lesion dated back to 1 month after the detection of coronary artery disease by angiogram and he underwent angioplasty 1 year later.

Dermatological examination showed multiple (10-15), annular, erythematous plaques, on his extremities, size ranging from 5 cm x 5cm to 15 cm x 8 cm, with central fading and a peripheral trailing scale at the inner border of the indurated edge [Figure 1 and 2].



Figure 1: Erythematous annular plaque with trailing scale.



Figure 2: Large erythematous annular plaques on both thighs with indurated borders and loss of hair.

Erythematous papules and purpura were noted in some of the plaques. The various stages of evolution noted during the follow up period were pink papules, red patches with fine scaling, fissuring of margins, oozing of blood tinged serous discharge, resolving patches with pigmentary changes and loss of hair [Figure 3, 4 and 5].



Figure 3: Erythematous plaque with fine scaling on the arm.



Figure 4: Thighs showing evolving erythematous plaques with trailing scale on the edges.



Figure 5: Resolving stage of annular erythema with trailing scale.

Α detailed history, examination, investigations and oncology screening failed to detect any foci of infection or neoplasms or any other internal diseases other than coronary disease. His artery electrocardiogram, angiogram and echocardiogram findings showed evidence of coronary artery disease.

A differential diagnosis of erythema annulare centrifugum, generalized granuloma annulare, atypical mvcosis fungoides, secondary syphilis, lymphoma cutis, porokeratosis and tinea corporis were considered. Direct microscopic examination of scales with potassium hydroxide (KOH) failed to reveal any fungal hyphae. Blood investigations were done to rule out syphilis, blood dyscrasias and immunological abnormalities.

Histopathological examination of punch biopsy taken from the edge of the lesion on the showed focal arm parakeratosis, spongiosis, perivascular infiltration of lymphohistiocytes in the upper dermis with focal areas showing extravasation of red blood cells in papillary dermis [Figure 6 and 7]. This was consistent with the diagnosis of erythema annulare centrifugum.

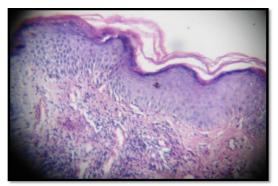


Figure 6: Skin biopsy showing focal parakeratosis, spongiosis and perivascular infiltration of lymphohistiocytes in upper dermis (H and E, 10x).

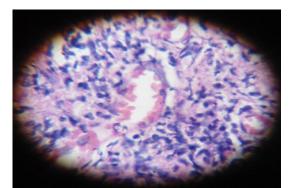


Figure 7: Perivascular lymphohistiocytic infiltrate in the upper dermis (H and E, 40x).

The patient was treated with potent topical corticosteroids, emollients and antihistamines with which he showed symptomatic response during the follow up period.

### **DISCUSSION**

The term erythema annulare centrifugum was originally used by Darier in 1916. <sup>[3]</sup> It is considered to be a reactive condition with a mean duration of eruption of 11 months. <sup>[4]</sup>

#### Aetiopathogenesis

Almost certainly EAC is not a single disease but a clinical finding with many causes. The inciting factors leading to hypersensitivity reaction may include viral (Epstein - Barr virus), fungal (dermatophyte, candida), bacterial (Streptococcal) infections. infestations (Ascaris, Borrelia. Trypanosomiasis), malignancies (lymphoproliferativeparaneoplastic erythema annulare centrifugum eruption-PEACE), blood dyscrasias, dysproteinemias, immunological disturbances, drug hypersensitivity (non steroidal antiinflammatories. gold, aldactone, thiacetazone, amitryptilline, hydrochlorthiazide, contraceptives), internal diseases (liver- auto immune hepatitis), or endocrine abnormalities (hyperthyroidism, hashimoto's thyroiditis). <sup>[2,5]</sup> An idiopathic annually recurrent form and familial type with dominant inheritance has been described.<sup>[6]</sup> But the cause of most cases of EAC is still unknown. In one series, 24 patients were closely evaluated, and in none of the cases was any definite cause found.<sup>[7]</sup>

The exact pathophysiology of EAC is not known. One report suggested that it is a tumor necrosis factor (TNF)- $\alpha$  dependent process because the patient responded to treatment with etanercept with complete clearance. <sup>[8]</sup>

# **Clinical Features**

EAC, characterized by erythematous, annular plaque, is subtyped clinically and histologically into superficial and deep forms. The superficial variant has an indistinct border with a characteristic peripheral trailing scale, whereas the deep type is non-scaly and has a firm indurated border. <sup>[1]</sup>

# Histopathology

A clinicopathologic study of 82 biopsies suggested that EAC with a superficially situated infiltrate is different to that with a deeper dermal infiltrate. In the superficial variant, there is a superficial, perivascular, tightly cuffed lymphohistiocytic infiltrate with endothelial cell swelling and focal extravasation of erythrocytes in papillary dermis with epidermal spongiosis and focal parakeratosis. In deep indurated form is mid and deep dermis is characterized by dense perivascular lymphohistiocytic infiltrate in coat sleeve like pattern.<sup>[9]</sup>

## Management

EAC is a reactive process with self limited course and good prognosis. If EAC is due to an underlying disorder, the lesions will usually resolve once the latter has been successfully treated. But the discovery and elimination of the cause is seldom possible. Lesions tend to be recurrent over months to years, waxing and waning in severity, with most cases eventually subsiding spontaneously. Topical corticosteroids. calcipotriol, tacrolimus. macrolides. phototherapy, oral antimicrobials like sulphonamides, metronidazole, corticosteroids, immunosuppressives. chloroquine, hydroxychloroquine (eosinophilic variant) and etanercept have been tried with variable responses. <sup>[1,2,8]</sup>

# CONCLUSION

The recognition and exact diagnosis of EAC is important because it may be a quite stressful condition and lead to unnecessary over-treatments. It is now widely accepted that EAC is a clinical reaction pattern rather than a separate disease entity. In this report, we seek to highlight the rare presentation of EAC characterized by truncal sparing with loss of hair in the annular erythema and its rare association with coronary artery disease.

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