Aceclofenac-Induced Erythema Annulare Centrifugum

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Abstract
Erythema annulare centrifugum (EAC) is characterised by slowly enlarging annular erythematous lesions and is thought to represent a clinical reaction pattern to infections, medications, and rarely, underlying malignancy. Causative drugs include chloroquine, cimetidine, gold sodium thiomalate, amitriptyline, finasteride, etizolam etc. We present a case of 40-year-old woman who presented to us with a 10 days history of nonpruritic, peripherally growing annular erythematous eruption. She had a history of recent onset of joint pain, for which she was taking aceclofenac 90 mg once a day for 5 days prior to the onset of the rash. This was confirmed on biopsy as EAC. The rash promptly subsided after stopping the drug. We report this case as there was no previous report of aceclofenac induced EAC.

Key Words: Aceclofenac, drug induced, erythema annulare centrifugum

Introduction
Erythema annulare centrifugum (EAC) is characterised by slowly enlarging annular erythematous lesion and is thought to represent a clinical reaction pattern to infections, medications, and rarely, underlying malignancy. Causative drugs include chloroquine, cimetidine, gold sodium thiomalate, amitriptyline, finasteride, etizolam etc.

Case Report
A 40-year-old woman presented to us with a 10 days history of nonpruritic, peripherally spreading annular erythematous eruption. She had a history of recent onset joint pain, for which she was taking aceclofenac 90 mg once a day for 5 days before the onset of the rash. On cutaneous examination, there were multiple well-defined annular plaques on the trunk, upper arms and thighs.

The lesions had slightly raised bright red borders with trailing scales behind the advancing edges. No other significant findings were present during the physical examination, nor signs of Sjogren’s syndrome or malignancy.

Results of the laboratory examinations, including complete blood count and biochemistry profile, were normal or negative. Tests for antibodies against syphilis, anti-nuclear antibody, anti-dsDNA antibody, and anti-SS-A and SS-B antibodies were negative. Thyroid profile, Mantoux test, chest x-ray, throat swab culture as well as serology for HIV, hepatitis B, and hepatitis C were found to be normal.

A skin biopsy was taken from the erythematous border of the lesion from the back. In the biopsy, there was mild spongiosis and focal parakeratosis. Features were compatible with the superficial variant of EAC.

As we suspected a drug eruption, we discontinued administration of aceclofenac, and the lesions gradually subsided and disappeared after 2 weeks.

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Pityriasis rosea (PR) and pityriasis rosea like drug eruption were considered as differential diagnosis. Lesions not arranged along the line of cleavage, relatively larger size of the lesions and the presence of trailing scale unlike PR where the scales are attached peripherally and free at the inner margin, helped in ruling out pityriasis rosea clinically. In addition, drug-induced PR is usually known to cause intense itching whereas the patient in this case was asymptomatic and no bright red erythema.
was appreciated which is described in drug-induced pityriasis rosea. In histopathological examination, although parakeratosis and spongiosis are seen in both drug-induced EAC and pityriasis rosea, the absence of apoptotic keratinocytes and eosinophils in this case favours EAC above pityriasis rosea. Furthermore, no blood eosinophilia was present in our patient which is usually seen in pityriasis rosea like drug rash.

**Discussion**

EAC has been categorised into deep and superficial variants.[6] The deep form of EAC, originally described by Darier in 1916, nonscaly annular lesion with indurated edges.[7] Histopathologically, a dense perivascular infiltrate is present in the mid and lower dermis.

On the other hand, the superficial form has less induration but shows scaling along the ring-shaped or gyrate border. In addition to a superficial perivascular infiltrates, epidermal changes like parakeratosis and spongiosis are also found in the superficial form. The eruptions seen in our patient are typical of the superficial variant of EAC, both clinically and histopathologically.

EAC is thought to represent a reaction pattern to a variety of underlying infectious, tumoral or immunological diseases, or to certain drugs and foods. The drugs which have been linked as a trigger are diuretics, nonsteroidal anti-inflammatory drugs, antimalarial, gold, finasteride, amitriptyline, etizolam, etc. The lesions in our patient were due to aceclofenac administration. This conclusion was based on the temporal relationship with drug administration, resolution of the lesions after discontinuation of the drug, and after ruling out other common causes.

In summary, aceclofenac, as a widely prescribed drug, should be considered as a possible causative agent for EAC.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient had given her consent for her images and other clinical information to be reported in the journal. The patient understood that her name and initial would not be published and due efforts would be made to conceal her identity, but anonymity cannot be guaranteed.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

**What is new?**

- Aceclofenac induced EAC has not been reported previously.
- Strong suspicion can help identify and withdraw the culprit drug.
- Withdrawal of the drug aids in resolution of lesions.

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