

CASE REPORTS

CAPTOPRIL INDUCED PEMPHIGUS VULGARIS

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A 51-year-old man was taking captopril 200 mg daily for the management of essential hypertension. Twenty two days after starting captopril, he developed flaccid vesicles and bullae over his scalp, face, trunk and mucous membranes of the mouth. Clinical and histopathological features were consistent with the diagnosis of pemphigus vulgaris. Complete disappearance of the skin lesions within 15 days of withdrawing the drug and absence of any recurrences in one year follow-up suggest that captopril was the probable offending drug.

Key words : Captopril, Pemphigus vulgaris.

Captopril, an oral inhibitor of angiotensin-converting enzyme, is a new approach for treating hypertension and cardiac failure.¹ The cutaneous side effects reported so far includes : maculo-papular rashes, urticaria, pityriasis rosea like² and lichenoid eruptions,³ and pemphigus.⁴ These side effects are remarkably similar to those of d-penicillamine. Both compounds possess a sulfhydryl group which has been considered responsible for these reactions.^{4,5}

Case Report

A 51-year-old man was being treated with 50 mg hydrochlorothiazide daily, since June 1984, and captopril 200 mg daily since December, 1985 for the management of essential hypertension. Twenty two days after starting captopril, he developed flaccid vesicles and bullae over his scalp, face and trunk. Nikolsky's sign was positive. Oral mucosa showed superficial erosions, but conjunctiva, palms and soles were normal.

Haemoglobin, TLC, DLC, ESR, blood sugar, urea, serum proteins and bilirubin were

within normal range. KOH preparation from buccal mucosa for candida was negative. Multiple acantholytic cells were seen in the Tzanck smear taken from the base of a vesicle. The diagnosis of pemphigus vulgaris was confirmed by histopathological study which showed suprabasal, intra-epidermal bullae containing numerous, large, acantholytic cells. Captopril was stopped and he was given 1% aluminium acetate solution and topical corticosteroids following which the lesions resolved within 15 days. One year follow up showed no recurrence.

Comments

Occurrence of the vesiculo-bullous eruption following approximately 3 weeks of the drug, complete disappearance of the eruption on stoppage of captopril and no recurrence in one year follow up suggested that captopril was the offending drug. Similar type of eruptions have been reported with prolonged d-penicillamine treatment. It has been estimated that 7% of patients taking d-penicillamine for at least six months develop pemphigus.⁶

The chemical structure of penicillamine and captopril are strikingly similar. Both have a highly reactive, negatively charged sulfhydryl

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group in a stable stereochemical relation to a similarly negatively polarised axo group. The stereochemical similarities between penicillamine and captopril, combined with the clinical evidence of their ability to produce similar disorders, suggest a similar mechanism of toxicity.⁴

References

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