Case report

A 28-year-old European woman presented with nausea and mild diarrhoea. She was taking pyrimethamine 25 mg twice weekly for malaria prophylaxis. A blood film showed malaria parasites. Unfortunately facilities were not available for species identification, but the malaria in the district was generally assumed to be due to *Plasmodium falciparum*. She was given a course of chloroquine by mouth to a total of 1800 mg. For a body weight of 45 kg that amply met the WHO recommended standards. She had no diarrhoea or vomiting while taking the drug. She had never had malaria before nor had she previously taken chloroquine. The treatment did not relieve her symptoms—in fact, they were added to by the drug's side effects. Five days after her initial presentation a blood film, examined at a different laboratory, showed abundant parasites. Her haemoglobin concentration was 12 g/dl. She was therefore given Fansidar (pyrimethamine 25 mg, sulphadoxine 500 mg) three tablets immediately.

Four days later she again attended, feeling weak and dizzy, with a temperature of 38°C. There were still a few plasmodia in the blood film. She was given chloroquine 800 mg immediately, to be repeated once weekly for three weeks—although only one of these doses was taken. She slowly improved, and two weeks after her first attendance her blood film was clear. No parasites were found in a repeat film after a further two weeks.

Comment

While we have no absolute proof of the existence of chloroquine-resistant malaria in our part of Africa we strongly suspect it because of several cases like this one, mostly in Africans. I have chosen to report this case because it is well documented although it is atypical in that the patient was a European taking regular prophylaxis. I am sure I am by no means alone in believing that resistance of *P falciparum* to chloroquine exists in Africa and that our regimens of treatment and chemoprophylaxis therefore need to be reconsidered.

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(*Accepted 25 March 1980*)

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Captopril-induced pemphigus

Captopril (SQ 14225), an oral inhibitor of angiotensin-converting enzyme, offers a new approach to treating several forms of hypertension. The commonest unwanted effects are morbilliform or maculopapular rashes and fever, which usually resolve on stopping or reducing the dose.[1] Other unwanted effects include aphthous ulcers of the mouth, temporary ageusia, the nephrotic syndrome, and reversible renal failure.[1] We report a case of pemphigus induced by captopril.

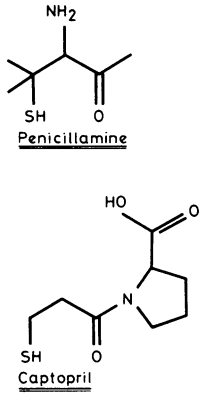
Case report

A 45-year-old previously fit English Jew began captopril 25 mg thrice daily in July 1979 for essential hypertension. The dose was increased weekly for four weeks to 150 mg thrice daily. Propranolol 10 mg thrice daily was added, being increased at weekly intervals until control was achieved with 80 mg thrice daily. He remained on the same dose of both drugs until January, when he presented with a two-week history of lesions on the trunk, scalp, and face and painful tongue erosions. The skin lesions were sore, red patches which had rapidly lost their surface, oozed, and crusted. Pemphigus erythematosus was diagnosed. Captopril was stopped, and after five weeks the skin and mouth ulcers had healed, though one new cutaneous lesion appeared. Blood cell count, erythrocyte sedimentation rate, blood glucose concentrations, liver and renal function values, protein electrophoresis, and immunoglobulin and serum complement concentrations were normal; antinuclear factor was speckled positive at 1/10.

Autoantibody to epidermal intercellular cement substance was positive at 1/40. Skin biopsy of the trunk showed an intraepithelial bulla high in the epidermis, containing a few acantholytic cells. On direct immunofluorescence upper epidermal intercellular IgG and lower epidermal intercellular C3 were detected in a biopsy specimen of a lesion. There was basement-membrane deposition of C3 in perilesional skin. These appearances were consistent with pemphigus erythematosus. The HLA genotype was HLA-A1, 3/8, 18/BL, -/-/6/DRW3, DRW5.

Comment

Though several hundred patients have been treated with captopril for at least six months, this is the first report of captopril-induced pemphigus. In Jewish patients with pemphigus vulgaris there is a particularly high prevalence of HLA-A10 and DRW4.[2] Our patient had neither of these.



Chemical structures of captopril and penicillamine.

Prolonged penicillamine treatment may also induce pemphigus. It has been estimated that 7% of patients taking penicillamine for at least six months develop pemphigus.[3] Most patients have had pemphigus foliaceus, a few pemphigus erythematosus (as in our patient), and one pemphigus vulgaris. The side effects of captopril,[1] such as maculopapular and urticarial eruptions, which occur in the first few weeks of treatment, mouth ulcers, temporary ageusia, the nephrotic syndrome, and now pemphigus, are the same as those of penicillamine.

The chemical structures of penicillamine and captopril are strikingly similar (figure). Each compound has a highly reactive, negatively charged sulphydryl group in a stable stereochemical relation to a similarly negatively polarised oxo group. The stereochemical similarities between penicillamine and captopril, combined with the clinical evidence of their ability to produce similar disorders, suggest that the toxicity of both drugs may prove disconcertingly similar.

We thank Dr F Marsh, Dr H Baker, and Dr J Pegum for advice during investigation of this patient and preparation of the manuscript.

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(*Accepted 15 April 1980*)

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Systemic lupus erythematosus and nephritis: severe relapse with disappearance of antinuclear antibodies

Exacerbations of systemic lupus erythematosus with active renal disease are almost always associated with high plasma titres of antinuclear factor (ANF) and DNA-binding antibody (anti-ds DNA).[1] We describe a patient with classical systemic lupus erythematosus and initial high titres of antinuclear antibodies in whom severe relapse resulting in renal failure coincided with a prolonged reversion to normal of these serological indices.

Case report

A 20-year-old woman developed the nephrotic syndrome and a butterfly rash. Results of investigations were: haemoglobin concentration 9·6 g/dl; platelet count 87×10^9/l (87 000/mm^3); serum electrolyte, urea, and creatinine