blood biochemical values were: urea 21.2 mmol/l, creatinine 428 mmol/l, sodium 143 mmol/l, chloride 112 mmol/l, potassium 7.2 mmol/l. Muscle cramps due to dehydration and hyperkalaemia were diagnosed and sodium and potassium replacement was given. He was treated with an insulin-dextrose infusion and calcium requirement. Over the next two days his cramps improved considerably and potassium was kept at 3.1 mmol/l sodium 142 mmol/l, chloride 105 mmol/l, potassium 4.0 mmol/l.

This case highlights the potentially dangerous interaction between angiotensin converting enzyme inhibitors and potassium sparing diuretics and the resultant rise in serum potassium concentration. Administration of captopril to patients taking diuretics causes a rise in serum potassium concentration.1 This is associated with a fall in circulating aldosterone levels2 and reduced potassium loss from the distal tubule. Captopril and other angiotensin converting enzyme inhibitors block the production of angiotensin II, which is the main stimulant to aldosterone release. Concurrent administration of potassium sparing diuretics or potassium supplements with captopril has been associated with hyperkalaemia.3 This was probably the cause in our patient, with his mild chronic renal failure. The blood potassium level stopped. He was discharged on frusemide 40 mg/day and 10 days later was readmitted, this time with an associated plasma potassium level of 5.1 mmol/l sodium 142 mmol/l, chloride 105 mmol/l, potassium 4.0 mmol/l.

Psoriasis as a side effect of β blockers

Drs M Keefe, N W Hamlet, and Rebecca E I Kerr
(Department of Dermatology, Stobhill General Hospital, Glasgow G21 3UW) write: The many currently reported side effects of captopril have been rash.1 These are most often pruritic, maculopapular, and limited to the upper half of the body but may resemble herpetic lesions. In one case,1 a patient developed an urticarial erythema with eczematous features leading to erythroderma.2 We have recently seen a 40 year old woman with a 16 year history of psoriasis who had developed a drug eruption after she took captopril for hypertension. Her psoriasis had previously been a stable plaque type, affecting the scalp, knees, and elbows, with never any generalised, facial, or axillary involvement. In April 1987 she started taking captopril 12.5 mg twice daily for hypertension. She was taking no other drugs. After two weeks she developed a pruritic, scaly eruption around the mouth and face typical of guttate psoriasis, although unlike the usual plaque pattern of her disease. The distribution was mainly central, affecting the back, buttocks, and posterior thighs. There was no history of sore throat or other illness, and an antistreptolysin O titre was 160 Todd units/ml. Captopril was continued. Outpatient treatment was unsuccessful, but inpatient treatment with tar, dithranol, and ultraviolet light cleared the psoriasis in August, and she subsequently remained clear. We cannot be certain that captopril exacerbated her psoriasis, but it occurred at a time when it was an association plausible. The mode of presentation was consistent with a primary psoriatic reaction and we think it unlikely that psoriasis developed as an ischemic event after an ordinary drug rash. There was no need to withdraw the drug, but rashes during captopril therapy can be transient and do not necessarily require cessation of therapy. The mechanism of exacerbation is obscure, but rashes from captopril are thought to result from an allergic reaction to the sulphydryl component or from cutaneous kinin production due to angiotensin converting enzyme inhibition,3 although this has been disputed.4 We are not aware of published reports of this association, but patients on captopril should be warned of the possibility. The mechanism of exacerbation may be similar to that occurring in response to captopril and enalapril. Am J Med 1985;89:79-82.

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