Hypoglycaemia induced by co-trimoxazole in AIDS

Dr A SCHWITTER, E SIMON, L GREEN, and R COLOSKY
Professor Z BINWEL (Department of Medicine C, Kaplan Hospital and Hadassah Medical School, Rehovot 76100, Israel) write: In recent years, trimethoprim-sulphamethoxazole has been widely used for the treatment of opportunistic infections in patients with AIDS.1 2

When adverse effects do occur they usually include minor gastrointestinal or cutaneous reactions. Severe toxic effects of sulphonamides and co-trimoxazole to cause hypoglycaemia, particularly in conjunction with other facilitating factors, was recognised long ago, but hypoglycaemia associated with trimethoprim-sulphamethoxazole has been encountered only rarely.1 2 We describe a patient with AIDS and Pneumocystis carinii pneumonia who had an episode of hypoglycaemia induced by co-trimoxazole. Neither our national drug monitoring centre nor the manufacturers of the drug have reported other cases of this side effect.

A 34 year old white homosexual man had been diagnosed as suffering from AIDS two years previously after developing weight loss, biopsy proved P carinii pneumonia, and Kaposi's sarcoma. He was also human immunodeficiency virus (HIV) positive and T4 cell depleted. He was taking co-trimoxazole twice daily, Faglipan, prochlorperazine and metoclopramide for progressive weakness necessitated his admission.

The main new findings included pancytopenia with a hypochromic microcytic anaemia, mild jaundice, hepatomegaly associated with the presence of hepatic transaminase and alkaline phosphatase activities. The drug was stopped and plasma glucose concentration measured 5:3 mmol/l. He was treated for the hypoglycaemia with glucose and discharged.

Acute painful proximal myopathy associated with naldixic acid

Dr s ANDREW J CAMMCHIEF and ANTHONY M MARTIN (Renal Unit, Royal Infirmary, Sunderland SR2 7JE) write: Several reports have implicated naldixic acid, including an acute painful proximal myopathy, but we are unaware of any reports implicating naldixic acid.

A 53 year old woman with chronic pyelonephritis, controlled hypertension, and impaired renal function (urea 29 mmol/l, creatinine 345 µmol/l) was prescribed full dose naldixic acid (Negrax) 1 g four times a day for a urinary tract infection. She had never received this antibiotic previously. Within 24 hours of starting treatment she noticed increased pain and numbness in the proximal muscles of the arms and legs. She stopped the treatment after taking four doses and her symptoms gradually resolved over the next three days. The pain and numbness were associated with skin excoriations of chlorhexidine and hydrochlorothiazide (Moduretic) two tablets a day, which she had taken regularly for two months before this episode. Serum potassium concentration was normal.

The Committee on Safety of Medicines has received five reports of myalgia associated with naldixic acid (personal communication) and the Australian Drug Evaluation Committee a further three.3 Given the strong temporal relation between the myopathy and naldixic acid treatment in this patient and the substantial number of suspected reports, we think it would be appropriate to include naldixic acid in the list of drugs associated with myopathy. We also advocate adherence to the manufacturer's recommendation to reduce the dose in renal impairment, which may have contributed to this impotence reaction.

1 Lane RJM, Macutaglia FL. Drug induced myopathies in man. 1979;35:360.

Severe salt and water deficiency associated with a combination of atenolol and chlorothalidone

Drs CHRISTINE A BOWMAN and WILLIAM J JEFFCOATE (City Hospital, Nottingham NGS 1PB) write: Professor W J MacLennan (4 June, p 151) discussed the electrolyte imbalances that may arise in elderly people on diuretics. Such adverse effects have been reported previously in patients on low dose combinations of a β blocker and a thiazide. We describe a case of salt and water deficiency associated with the combination of atenolol and chlorothalidone.

A 75 year old white woman presented with two days of vomiting, dizziness and a self diagnosis of dehydration. She was known to have end stage renal failure and hypothyroidism. She had been prescribed atenolol tablets once daily and chlorothalidone tablets once daily for hypertension.

She had been dehydrated, and her blood pressure was 109/60 mmHg and 80/40 mmHg sitting. Plasma electrolyte concentrations were: sodium 109 mmol/l, potassium 1·5 mmol/l, urea 6·8 mmol/l, and creatinine 5·3 mg/dl. After partial rehydration with 1·5 litres of isotonic saline and 121·5 mmol potassium/l intravenously her plasma sodium and potassium concentration were normal. Serum chloride concentration was 109 mmol/l. Several urinary values were 94 and 26 mmol/l and 532 mmol/l. Calcium and glucose concentrations were normal.

A bronchoscopic biopsy specimen of a hilar mass showed small cell anaplastic carcinoma. After full rehydration over 27 days ectopic adrenocorticotrophin was started and her diuretics were reduced. Her renal function improved and seven 5 mg tablets of prochlorperazine in the week before admission (for “dizziness”)

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