results for LE cells. These abnormalities resolved on stopping the drug, and the authors suggested that sulphonamides might unmask a latent "lupus diathesis" in certain patients with ulcerative colitis. The HLA-B8 antigen is associated with various immunologically mediated diseases,¹³ and it may have predisposed our patient to develop a drug-induced lupus syndrome.

This case highlights the possibility of a sulphonamide-induced SLE syndrome occurring in any patient with colitis who develops polyarthropathy or vasculitis while receiving sulphasalazine. The patient's slow-acetylator phenotype permitted the development of high serum concentrations of sulphapyridine, even with modest doses of sulphasalazine, and this may have facilitated the appearance of the lupus-like syndrome.

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Stevens-Johnson syndrome due to rifampicin

The recorded side effects of rifampicin include nausea, skin rashes, liver dysfunction, hypersensitivity reactions, leucopenia, thrombocytopenia, and ocular abnormalities.^{1,2} We report a case of overt Stevens-Johnson syndrome apparently caused by rifampicin.

Case report

An African man of about 40 years of age was diagnosed as having pulmonary tuberculosis, and started on a regimen of rifampicin 600 mg, streptomycin 1 g intramuscularly, and isoniazid 300 mg daily. He made little clinical improvement, however, and after four weeks ethambutol 800 mg daily was added. One week later he developed severe exfoliative dermatitis, buccal ulceration, blepharoconjunctivitis, and anterior uveitis. He was feverish and extremely ill. All antituberculous drugs were stopped, and he was treated with high doses of oral prednisolone with local steroid applications to the eyes and skin. He improved slowly, and three weeks later was considered well enough to have the antituberculous drugs reintroduced one at a time. Rifampicin alone was started, but four days later he once again began to develop dermatitis, buccal ulceration, and conjunctivitis similar to his previous condition. The rifampicin was promptly stopped and steroids begun again. He improved quickly, and streptomycin, isoniazid, and ethambutol were added stepwise with no further complications.

Comment

The recurrence of Stevens-Johnson syndrome in our patient after the reintroduction of rifampicin strongly suggests a causal relation. Interestingly, the patient tolerated the first four weeks of rifampicin treatment, but became ill one week after the addition of ethambutol. Lees *et al*³ have recorded a case of ocular side effects in a patient on

rifampicin and ethambutol. Thus ethambutol may act as a trigger-factor to the toxic effects of rifampicin.

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Severe anaphylactoid reactions to dextran 70

We report two cases of anaphylactoid reactions to dextran 70 in asthmatic patients. After vigorous resuscitation one patient recovered completely but the other died.

Case reports

Case 1—This patient, a 24-year-old primigravida, had suffered from asthma since the age of 3, for which she used a salbutamol sulphate inhaler (Ventolin), and took Franol tablets (ephedrine hydrochloride 11 mg, theophylline 120 mg, phenobarbitone 8 mg). She was delivered 19 days preterm by elective lower-segment caesarean section, the indications being fetal tachycardia of 180 beats/min and moderately severe pre-eclampsia. Her blood pressure had risen from 110/70 mm Hg at admission to 140/100 mm Hg four days before operation. After the caesarean section her systolic blood pressure fell to a level of 60-70 mm Hg. Dextran 70 (Lomodex 70, Fisons) was infused intravenously, and she developed severe bronchospasm and a florid urticarial rash on her limbs. Dextran 70 infusion was replaced with compound sodium lactate injection immediately. The bronchospasm failed to respond to intravenous aminophylline (150 mg), but responded to intravenous salbutamol sulphate (0.5 mg). Her subsequent recovery and her puerperium were uneventful.

Case 2—A 25-year-old woman (gravida 2, para 1) with bronchial asthma and on treatment with prednisolone, beclomethasone dipropionate, salbutamol sulphate, and sodium cromoglycate (Intal) was admitted to the labour ward from the antenatal ward, where she had been for two weeks because of mild pre-eclampsia, in early labour. She was given 100 mg hydrocortisone intramuscularly as the first dose of cover in labour. Labour was accelerated by amniotomy, an infusion of 5% dextrose was started, and epidural analgesia instituted using bupivacaine hydrochloride (Marcain) because the patient's blood pressure was varying between 140/90 mm Hg and 150/100 mm Hg. Ten minutes later her blood pressure dropped to 90/69 mm Hg, so an intravenous dextran 70 (Lomodex 70, Fisons) infusion was started. After 200 ml had been infused for 10 minutes her blood pressure became unrecordable, she complained of tightness in the chest, and within one minute she became cyanosed and pulseless. Vigorous resuscitation was started with cardiac massage; oxygen 100% was given through an endotracheal tube, using intermittent positive-pressure ventilation; intravenous adrenaline 0.1 mg, hydrocortisone 400 mg, sodium bicarbonate infusion, and aminophylline 250 mg were given. During resuscitation the heart went into asystole and ventricular fibrillation several times and was defibrillated about a dozen times. Resuscitation was stopped after 80 minutes. The main problem during attempted resuscitation was severe bronchospasm, making it impossible to ventilate the lungs properly. Necropsy examination (performed by Professor H L Sheehan) showed subdiaphragmatic oedema, congestion of gastric mucosa and of kidneys, and incoagulability of the blood. Amniotic fluid embolism was excluded by gross and microscopical examination of the lungs and other organs. There were no pathological changes related to the history of asthma.

Comment

The number of hypersensitivity reactions¹⁻⁵ (ranging from mild urticaria to anaphylactoid reactions) reported to Fisons Ltd, manufacturers of dextran 70 (Lomodex 70), which we used in the above two cases, was one per 22 852 bottles sold during 1972-6. Thirty-five per cent of the patients who developed hypersensitivity to dextran 70 had a history of bronchial asthma and 17% had a history of allergy (Fisons Ltd, personal communication).

Dextran 70 is widely used as a plasma expander for patients in circulatory collapse, a condition which could mask a superimposed anaphylactic shock or make physical signs difficult to interpret. If the administration of intravenous dextran leads to a further drop in blood pressure in a patient in collapse, increasing the rate of infusion may have disastrous effects.

It therefore seems advisable to avoid using dextran 70 in patients with a history of allergy or bronchial asthma. A doctor should be present during the first 200 ml of a dextran 70 infusion, and if an untoward reaction appears dextran should be discontinued. A separate infusion of saline should be running simultaneously at a speed determined by the degree of shock. For the first 15 minutes the dextran infusion should be at the rate of 10 drops/min, and cardio-respiratory resuscitation drugs, equipment, and personnel should be immediately available.

We are greatly indebted to Mr S Bender for his guidance in reporting the above cases.

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Heart block in mianserin hydrochloride overdose

So far there have been no published reports on the effects of mianserin hydrochloride on the conducting tissue of the heart in man, although animal experiments have indicated that it has few if any cardiotoxic effects.¹ This is believed to be the first documented case of heart block occurring after mianserin hydrochloride overdosage.

Case report

A 39-year-old woman was admitted to hospital on 6 January 1977, three hours after taking 58 10-mg tablets of mianserin hydrochloride (Bolvidon, Organon), seven 5-mg diazepam tablets, and six 5-mg nitrazepam tablets.

She induced vomiting soon after taking the tablets, and gastric lavage was carried out on admission.

She was drowsy and complained of a dry mouth. Blood pressure was 115/95 mm Hg and pulse rate 90 beats/min. The electrocardiogram (ECG) on admission showed first-degree heart block (P-R interval 0.28 seconds). Serial ECG tracings showed that she remained in first-degree block until 14 hours after ingesting the tablets. For the next two hours the block was variable, and by 16 hours after ingestion she had spontaneously reverted to normal rhythm (P-R interval=0.18 seconds). An ECG recorded a year previously at a routine outpatient visit was normal.

Blood was removed at intervals for drug estimation by Organon Laboratories Ltd, and the results are shown in the table. The peak serum concentration was four times the therapeutically desirable level.

Serum concentrations of mianserin hydrochloride

Hours after ingesting tablets:	3½	9	21	27
Serum mianserin hydrochloride (µg/l) ..	439	157	86	70

Peak level during normal treatment with 20 mg thrice daily is 100-120 µg/l.

Comment

The cardiotoxic effects of tricyclic antidepressants in cases of self-poisoning have been well documented,² and even therapeutic doses of these drugs affect intracardiac conduction.³ Recently new antidepressants with a tetracyclic structure have been marketed, which are claimed to have no cardiotoxic effects at normal doses. Animal studies have shown that effects are dose related and that a proportionately higher dose of mianserin hydrochloride is required to cause arrhythmias than with tricyclic antidepressants.

In our patient the effect on the conducting tissue of the heart seemed to be dose related, in that conduction returned to normal when the blood concentrations descended to the therapeutic range. Only the P-R interval was abnormal in this patient. The QRS and S-T complexes and Q-T interval were normal.

This drug is claimed to have no adverse cardiac effects, but this case suggests that, in common with tricyclic antidepressants, mianserin hydrochloride in very high doses can interfere with intracardiac conduction.

We thank Organon Laboratories Ltd for kindly undertaking the serum determinations.

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SHORT REPORTS

The Sparks mandril in femoropopliteal bypass

Autogenous long saphenous vein is undoubtedly the most suitable material for femoropopliteal bypass in patients with limb-threatening ischaemia.¹ Unfortunately, in an appreciable number of patients the vein is absent (as a result of previous surgery) or unsuitable,¹ and cloth prostheses below the inguinal ligament, which give generally poorer results, have to be used.² The Sparks mandril graft aims to obviate these problems by producing an autogenous, Dacron-supported tube, of suitable dimensions, which can be grown in situ. Initial clinical results were published in 1972³ and were sufficiently

encouraging for the technique to be cautiously adopted in Nottingham. We describe here our experience with 10 patients.

Patients, methods, and results

The series consisted of seven men and three women aged 44 to 70 years (mean 60 years). Two patients were diabetic, five were hypertensive, and all were heavy smokers. Ischaemic ulceration had occurred in seven patients, and the remaining three were severely incapacitated by claudication. Seven failed arterial reconstructions and three failed sympathectomies had previously been performed, and as no patients had a suitable ipsilateral saphenous vein (five had been used for bypass, four were too small, and one had been removed because of varicosity) femoropopliteal bypass was achieved using the Sparks mandril graft.³