day. Since then his blood pressure remained steady at 140/90 mm Hg on oxprenolol 80 mg three times a day alone.

Comment

There seems little doubt that this hypertensive episode was caused by phenylpropanolamine. Although the combination of methyl dopa and intravenous propranolol has occasionally produced severe hypertension² there was no evidence of any major fluctuation in the patient's blood pressure until phenylpropanolamine was introduced. Methyl dopa is known to increase the pressor effects of sympathomimetic amines³ and it has been postulated that beta-blockade may have a similar action by allowing an unopposed alpha-constrictor response to adrenergic stimulation.⁴ The combination of methyl dopa and oxprenolol may therefore have caused a particular severe hypertensive reaction. The patient's renal failure might have exacerbated the effect since metabolites of methyl dopa with possible pressor effects are increased in uraemia.⁵

In view of the widespread use of methyl dopa and beta-blockers in combination it is surprising that an interaction with this amine has not been reported before. On this occasion the hypertension did not result in any permanent damage, but the consequences might be serious in patients with ischaemic heart disease.

The manufacturers of Triogesic do give hypertension as a contraindication to its use but no mention is made of beta-blockers that might be given for other reasons. In addition phenylpropanolamine is a constituent of many cold cures obtainable without prescription, and although warnings are printed on the packet these may well be disregarded.

It seems desirable that wider publicity should be given to the potentially serious effects of interaction of phenylpropanolamine with beta-blockers, methyl dopa, and other drugs, which might well outweigh its very limited role as a nasal decongestant.

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Detection of urinary amyloid in familial Mediterranean fever

Derosena *et al*¹ and Neale² reported finding amyloid fibrils by electron microscopy in the urinary sediment of two patients with renal amyloidosis. It was suggested that this technique could be used to diagnose renal amyloidosis when renal biopsy cannot be performed. We have used this technique to diagnose renal amyloidosis in a patient with familial Mediterranean fever, who subsequently showed a good response to colchicine.

Case report

A 72-year-old man was admitted to this hospital with a history of recurrent fever of undetermined origin for 35 years. Urine had a specific gravity of 1.011, contained protein 4+, and was negative for glucose. Microscopical examination of the urinary sediment showed hyaline, granular, waxy, and fatty casts. Doubly refractive fat bodies were visible with polarised light. The urinary protein was 4.2 g/24 hours, and the total serum protein 61 g/l (albumin 25 g/l). Pedal oedema and Muehrcke's lines were present. Packed

cell volume (PCV) was 0.26 (26%), blood urea nitrogen 23.2 mmol/l (65 mg/100 ml), and serum creatinine 398 μ mol/l (4.5 mg/100 ml). An intravenous pyelogram showed poor excretion and decreased size of both kidneys. Rectal biopsy was positive for amyloid, but renal biopsy was contraindicated because of the possibility of bleeding and the decreased renal function and small kidneys.

Urine was processed according to the method of Derosena *et al*,¹ and sections of the sediment were examined by electron microscopy. This showed fibrillar material with the characteristics of amyloid (see fig). The patient's mother was of Sephardic Jewish descent, and the working diagnosis of familial Mediterranean fever was made. The patient was maintained on colchicine 0.6 mg by mouth twice daily and remained asymptomatic and free of oedema for four months. At the time of writing, the urinary protein was 2 g/24 hours, serum albumin 37 g/l, serum creatinine 327 μ mol/l (3.7 mg/100 ml), and PCV 0.31 (31%).

Electronmicrograph of urinary sediment showing fibrillar material having size and periodicity of amyloid fibrils. (Uranyl acetate and lead citrate stain. $\times 32\,500$.)

Comment

Familial Mediterranean fever occurs most commonly in Sephardic Jews, Armenians, and Arabs, but a few cases have been reported in Ashkenazic Jews.³ It is characterised by intermittent acute episodes of fever and polyserositis, usually peritonitis, and its most serious sequela is renal amyloidosis.³ Some patients may have fever as the only manifestation of the disorder³ and are therefore subjected to repeated investigations for fever of unknown origin. This was true in the present case, and it was only after the appearance of the nephrotic syndrome and amyloidosis that the diagnosis was established.

Colchicine prevents acute exacerbations of familial Mediterranean fever, and response to the drug has been recommended as the most reliable diagnostic test for the disorder.⁴ Since renal amyloidosis is notoriously refractory to treatment,⁵ the apparent response in this patient is noteworthy. Renal biopsy may be hazardous in patients with renal amyloidosis because of an increased bleeding tendency. This is particularly true in the presence of renal insufficiency. The detection of urinary amyloid fibrils by electron microscopy is a practical and non-invasive alternative diagnostic method for patients suspected of having renal amyloidosis. Evaluation of its reliability and sensitivity will require further study.

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