atropine, and hyoscine and have been ingested, especially in the USA, for their hallucinogenic properties. Deaths have resulted.⁵

Our patients ate Surama cigarettes, which contain 92.5% stramonium leaf (equivalent to 1.5-2.5 mg alkaloids per cigarette). They displayed many features typical of anticholinergic poisoning but recovered without specific treatment. In severe poisoning physostigmine salicylate may be of value.

The occurrence of two or possibly four cases in so short a time suggests that the potentially lethal practice of ingesting such cigarettes for kicks may be enjoying a vogue.

We are grateful to Dr A T Proudfoot and Dr L F Prescott for permission to report details of patients under their care and for invaluable help in preparing the manuscript.

¹ Dean, E S, *Journal of the American Medical Association*, 1963, **185**, 882.

² Goldsmith, S R, Frank, I, and Underleider, J T, *Journal of the American Medical Association*, 1968, **204**, 169.

³ Gowdy, J M, *Journal of the American Medical Association*, 1972, **221**, 585.

⁴ Harrison, E A, and Morgan, D H, *British Medical Journal*, 1976, **2**, 1195.

⁵ Mikolich, J R, Paulson, G W, and Cross, C J, *Annals of Internal Medicine*, 1975, **83**, 321.

Regional Poisoning Treatment Centre, Royal Infirmary of Edinburgh, Edinburgh EH3 9YW

A BALLANTYNE, BSC, MB, senior house officer

P LIPPIETT, MB, BS, registrar in psychiatry

J PARK, MRCP, registrar in medicine

Postural proteinuria and lipid nephrosis of 58 years' duration

Lipid nephrosis of childhood usually undergoes remission at least during adult life and is not associated with accentuation of proteinuria with the upright position.¹ Postural proteinuria, on the other hand, is defined with normal quantitative protein on recumbency. The following case is therefore interesting.

Case report

In August 1968 a 60-year-old woman was admitted to the Thomas Jefferson University Hospital. Her history had begun in 1919 when, aged 11, she presented with intermittent swelling of the legs and face that was diagnosed as "nephritis." She was next seen in 1941, again because of swollen legs, and was told she had severe proteinuria. Oedema was nearly always present. For as long as she could remember she had had nocturia; she had to rise two to three times nightly for copious voiding. In 1965 she began taking diuretics.

Examination in 1968 showed pedaloedema, blood pressure 146/90 mm Hg, urine analysis 3+ - 4+ protein with daily excretion varying from 1.5 to 3 g depending on her position, and no urinary sediment. Serum urea was 1.6 mmol/l (10 mg/100 ml), serum creatinine 44.2 μ mol/l (0.5 mg/100 ml) (repeated) with a creatinine clearance rate of 110 ml/min, and two-hour postprandial blood sugar 74 mg/100 ml. Plasma albumin:globulin ratio was 21:27 g/l, cholesterol 4.7 mmol/l (183 mg/100 ml). Chest radiograph and ECG showed normal findings. (On admission at other hospitals serum albumin concentrations below 21 g/l had sometimes been recorded.)

She was readmitted in August 1969. She had mild oedema of the legs and there was no venous insufficiency. Blood chemistry showed serum urea 2.5 mmol/l (15 mg/100 ml); creatinine 53 μ mol/l (0.6 mg/100 ml); phosphate 1.42 mmol/l (4.4 mg/100 ml); Ca 4.4 mmol (mEq)/l, CO₂ 28, K 4.5, Na 138, and Cl 100 mmol (mEq)/l. Protein electrophoresis showed albumin 3.0, globulins: α_1 0.3, α_2 0.9, β 0.6, and γ 0.4. Twenty-four hour urinary protein was about 2 g. Plasma immunoglobulins were: IgG 6.9 g/l (690 mg/100 ml),

IgA 1.8 g/l (180 mg/100 ml), IgM 1.92 g/l (192 mg/100 ml). Blood volume was 2960 ml (normal for patient's height and weight). X-ray films of stomach and small bowel and jejunal biopsy were all normal. Labelled chromium study did not show any protein wasting in the bowel. An excretory urogram did not show ptosis of the kidney in an upright position.

Special studies on the effect of posture on protein and NaCl excretion showed reversal of normal diurnal cycle of NaCl excretion, which was restored to normal by 24-hour recumbency. Protein excretion averaged 1.5 g (7 am-7 pm) while she was up and about and 0.5 g (7 pm-7 am) while she was recumbent. The same pattern (but not degree) occurred with 24-hour recumbency. The renal biopsy result is shown in the figure. Light microscopy showed nearly normal glomeruli with occasional minimal focal changes. Electron microscopy (only one compressed glomerulus could be found in the sample) showed some fusion of foot processes but no thickening of basement membrane nor deposits.

Follow-up to 1976 showed that she was in excellent health with oedema well controlled on diuretics, a high-protein diet, and thermelastic stockings.

High-power view of a representative glomerulus. (PAS stain. $\times$ 310.)

Comment

This case of proteinuria is interesting because of the condition's unusually long course (58 years) with normal renal function and histology. The long duration and severe postural accentuation of proteinuria is unusual for lipid nephrosis,¹ yet it is equally unusual for postural proteinuria because of considerable losses during recumbency.

This reopens the controversy as to the lesion in lipid nephrosis, and whether "pure" lipid nephrosis is not a group of disorders. Rich² suggested many years ago that the lesion of focal glomerular sclerosis may first become manifest in the juxtamedullary glomeruli and thus could easily be missed with a conventional renal biopsy. Furthermore, some recorded cases of lipid nephrosis with progressive renal insufficiency are really examples of focal glomerular sclerosis² or very early stages of membranous³ or proliferative glomerular nephritis. The precise diagnosis in this case is still not clear.

This study was supported by General Clinical Research Centre Grant FR-72 from the National Institutes of Health.

¹ Saito, H, *Tohoku Journal of Experimental Medicine*, 1968, **95**, 207.

² Rich, A R, *Johns Hopkins Hospital Bulletin*, 1957, **100**, 173.

³ White, R H R, Glasgow, E F, and Mills, R J, *Lancet*, 1970, **1**, 1353.

⁴ Churg, J, et al, *New England Journal of Medicine*, 1965, **272**, 165.

Jefferson Medical College, Philadelphia, Pennsylvania, USA

M L SIMENHOFF, MB, MRCP(SA), associate professor of medicine