

Clearly, multiple sclerosis plaques may mimic gliomas on computed tomography. Absence of a mass effect has been suggested as a diagnostic criterion for demyelination,⁵ but confusion may still occur. Doubts about the nature of such a lesion may be resolved by either biopsy or serial computed tomography.

- ¹ Currie S, Urlich H. Concurrence of multiple sclerosis and glioma. *J Neurol Neurosurg Psychiatry* 1974;**37**:598-605.
- ² Cala LA, Mastaglia FL, Black JL. Computerised tomography of brain and optic nerve in multiple sclerosis. Observations in 100 patients including serial studies in 16. *Journal of the Neurological Sciences* 1978;**36**:411-26.
- ³ Van der Velden M, Bots GTAM, Endtz LJ. Cranial CT in multiple sclerosis showing a mass effect. *Surg Neurol* 1979;**12**:307-10.
- ⁴ Nelson MJ, Miller SL, McLain LW, Gold LHA. Multiple sclerosis: large plaque causing mass effect and ring sign. *J Comput Assist Tomogr* 1981;**5**:892-4.
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Department of Neurology, St James's University Hospital, Leeds LS9 7TF

R J ABBOTT, MB, MRCP, senior registrar
J G HOWE, MB, MRCP, tutor
S CURRIE, MD, FRCP, consultant
I HOLLAND, MB, FRCP, consultant neuroradiologist

Renal artery thrombosis caused by antihypertensive treatment

Antihypertensive drugs must be used cautiously in older patients. We describe an elderly man with Leriche's syndrome who developed anuria due to renal artery thrombosis after his blood pressure was reduced by atenolol. We believe that this complication of antihypertensive treatment has not been previously reported.¹

Case report

A 70-year-old man was admitted to hospital on 21 November 1981 with a three-day history of anuria. An inferior myocardial infarction had occurred in 1968. He had had mild intermittent claudication since 1971. In 1972 translumbar aortography had shown blockage of the left external iliac and right femoropopliteal arteries with good collateral circulation. The state of the renal arteries had not been noted. Mild hypertension had been treated with cyclopentiazide 0.5 mg daily for two years.

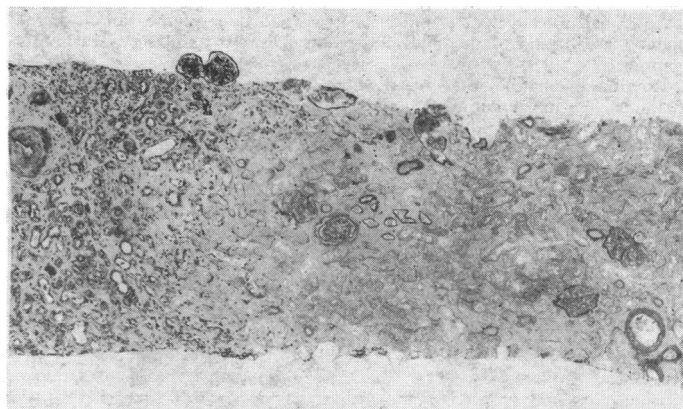
He had been given atenolol 100 mg on 18 November because his blood pressure was 240/115 mm Hg. Five hours after the first dose he had become faint and his blood pressure was 130/70 mm Hg. He was admitted to hospital three days later having been completely anuric since taking the single atenolol tablet; his general condition was good and there was no evidence of acute myocardial infarction. Blood pressure was 160/80 mm Hg. Femoral pulses were impalpable but blood supply to the legs was satisfactory. Blood urea concentration was 19 mmol/l (114 mg/100 ml). When urine was passed after seven days the sodium content was 6-12 mmol(mEq)/l.

Intravenous urography and sonography showed a normal-sized right and a small left kidney without obstruction. No renal uptake occurred on scanning with technetium diethylene triamine penta-acetic acid. Aortography was unsuccessful despite repeated puncture attempts at lower dorsal and upper lumbar levels. Biopsy of the right kidney showed extensive cellular necrosis (figure). Aortic thrombosis affecting the renal arteries was diagnosed. He was maintained on peritoneal dialysis for 42 days, but this was stopped when urine output remained below 250 ml/day.

Necropsy showed a small (67 g) fibrotic left kidney and a small (165 g) right kidney, not obviously infarcted. There was old thrombotic occlusion of the aorta extending above the renal arteries. The left renal artery was blocked by old thrombus and the right renal artery by more recent thrombus. There was severe coronary atheroma and considerable myocardial fibrosis.

Comment

The right renal artery thrombosis was almost certainly due to the fall in blood pressure caused by treatment. Because silent thrombosis of the abdominal aorta affecting the left renal artery had already occurred anuria resulted in this case. Antihypertensive treatment is not a previously reported cause of renal artery thrombosis.¹ As both



Renal biopsy specimen showing extensive cellular necrosis. Periodic-acid-Schiff X 20 (original magnification).

distal aortic thrombosis (Leriche's syndrome)² and renal artery thrombosis³ may be clinically silent, however, thrombosis of a stenosed renal artery after antihypertensive treatment might easily occur unrecognised.

Distal aortic thrombosis increases the likelihood of stenosis or thrombosis of a renal artery, and we suggest that the femoral arteries should be palpated to detect Leriche's syndrome before antihypertensive treatment is started in patients with peripheral vascular disease. Although the state of the renal arteries in 1972 was not recorded, renal artery stenosis of some degree may well have been present. Reconstructive surgery in patients with hypertension due to renal artery stenosis improves renal function in patients somewhat younger than ours.⁴ Our patient shows that the choice between reconstructive surgery, balloon dilatation, drug treatment, and no treatment may be difficult in hypertensive patients with arterial stenosis. Certainly drugs must be used carefully in older patients. In our patient the serum atenolol concentration would have been raised by impaired renal excretion.⁵

¹ Montoliu J. Renal artery thrombosis: systemic contraceptive-induced or spontaneous? *Ann Intern Med* 1979;**91**:657.

² Sabiston DC Jr. Disorders of the arterial system: In: Sabiston JC Jr, ed. *Textbook of surgery*. Philadelphia: W B Saunders, 1972:1939-42.

³ Lessman RK, Johnson SF, Coburn JW, Kaufman JJ. Renal artery embolism. Clinical features and long-term follow-up of 17 cases. *Ann Intern Med* 1978;**89**:477-82.

⁴ Mackay A, Brown JJ, Lever AF, Robertson JIS. Reconstructive surgery versus nephrectomy in renal artery stenosis: comparison of effects on total and divided renal functions and on blood pressure. *Br Med J* 1980;**281**:1313-5.

⁵ Sassard J, Pozet N, McAinsh J, Legheard J, Zeck P. Pharmacokinetics of atenolol in patients with renal impairment. *Eur J Clin Pharmacol* 1977;**12**:175-80.

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Royal Infirmary, Bradford BD9 6RJ

A B SHAW, MD, FRCP, consultant physician
S K GOPALKA, MB, BS, senior house officer in general medicine

Rastafarianism and the vegans syndrome

Low serum vitamin B₁₂ concentrations are common in vegans. We report the first account of the syndrome in members of the Rastafarian cult, which started in Jamaica more than 40 years ago.

Subjects, methods, and results

Ten Rastafarian men aged 18 to 40 who had been vegans for from two to 20 years presented with evidence of vitamin B₁₂ deficiency. Eight had neurological manifestations ranging from paresthesiae to subacute combined degeneration. Five patients had gastrointestinal symptoms: glossitis in three, anorexia in two (cases 6 and 7; case 6 also had epigastric discomfort), and