by early diagnosis of testicular disease is evidence against "reseeding" of the marrow from the testis or merely evidence of failure to detect minimal disease in the testicle.

The reasons for the high incidence of testicular relapse in some centres are not clear; in general it appears that schedules associated with a high risk of testicular infiltration are also those associated with a high risk of relapse in boys after treatment is stopped and those in which boys tend overall to do worse than girls.[1 2] If, as seems probable, testicular relapse is a sign of widespread residual disease, more intensive systemic chemotherapy may eliminate this difference in outcome between the sexes and reduce the incidence of testicular leukaemia.

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References

[1] Medical Research Council Working Party on Leukaemia in Childhood. Effects of varying radiation schedule, cyclophosphamide treatment and duration of treatment in acute lymphoblastic leukaemia. *Br Med J* 1978;ii:787-91.
[2] Sather H, Miller D, Nesbit M, Heyn N, Hammond D. Differences in prognosis for boys and girls with acute lymphoblastic leukaemia. *Lancet* 1981;i:739-44.
[3] Medical Research Council Working Party on Leukaemia in Childhood. Testicular disease in acute lymphoblastic leukaemia in childhood. *Br Med J* 1978;i:334-8.
[4] Land VJ, Berry DH, Herson J, *et al.* Long-term survival in childhood acute leukaemia: "late" relapses. *Med Pediatr Oncol* 1979;**7**:19-24.
[5] Nesbit ME, Robison LL, Ortega JA, Sather HN, Donaldson M, Hammond D. Testicular relapse in childhood acute lymphoblastic leukaemia: association with pretreatment patient characteristics and treatment. *Cancer* 1980;**45**:2009-16.
[6] Medical Research Council Working Party on Leukaemia in Childhood. Analysis of treatment in childhood leukaemia. II. Timing and the toxicity of combined 6-mercaptopurine and methotrexate maintenance therapy. *Br J Haematol* 1976;**33**:179-88.
[7] Medical Research Council Working Party on Leukaemia in Childhood. Analysis of treatment in childhood leukaemia. V. Advantage of reducing chemotherapy during and immediately after cranial irradiation. *Br J Cancer* 1977;**36**:625-33.
[8] Rapson NT, Cornbleet MA, Chessells JM, Bennett T, Hardisty RM. Immunosuppression and serious infections in children with acute lymphoblastic leukaemia. A comparison of three chemotherapy regimes. *Br J Haematol* 1980;**45**:41-52.
[9] Chessells JM, Breatnach F. Late marrow recurrences in childhood acute lymphoblastic leukaemia. *Br Med J* 1981;**283**:749-51.
[10] Baum E, Nesbit M Jr, Tilford D, Heyn R, Krivit W. Extent of disease in pediatric patients with acute lymphocytic leukaemia experiencing an apparent isolated testicular relapse. [Abstract.] *Proceedings of the American Society of Clinical Oncology* 1979;**20**:435.
[11] Hustu HO, Aur RJA. Extramedullary leukaemia. *Clin Haematol* 1978;**7**:313-37.
[12] Oakhill H, Mainwaring D, Hill FGH, *et al.* Management of leukaemic infiltration of the testis. *Arch Dis Child* 1980;**55**:564-6.
[13] Wong KY, Ballard ET, Strayer FH, Kisker T, Lampkin B. Clinical and occult testicular leukemia in long-term survivors of acute lymphoblastic leukemia. *J Pediatr* 1980;**96**:567-74.
[14] Sullivan MP, Perez CA, Herson J, *et al.* Radiotherapy (2500 rad) for testicular leukaemia. Local control and subsequent clinical events: a south-west oncology group study. *Cancer* 1980;**46**:508-15.
[15] Atkinson K, Thomas PRM, Peckham MJ, McElwain TJ. Radiosensitivity of the acute leukaemic infiltrate. *Eur J Cancer* 1976;**12**:535-40.
[16] Ash P. The influence of radiation on fertility in man. *Br J Radiol* 1980;**53**:271.
[17] Thomas JA, Janossy G, Eden OB, Bollum FJ. Nuclear terminal deoxynucleotidyl transferase in leukaemic infiltrates of testicular tissue. *Br J Cancer* 1982;**45**:709-17.

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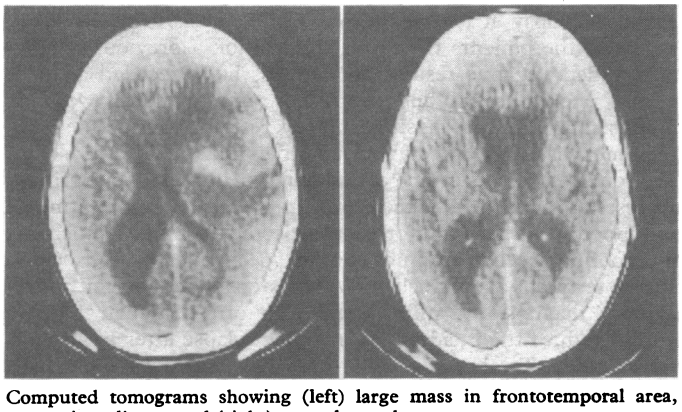
SHORT REPORTS

Multiple sclerosis plaque mimicking tumour on computed tomography

A cerebral lesion in multiple sclerosis may produce a mass effect visible on computed transmission tomography. We report a case in which cerebral tumour was wrongly diagnosed as a result of just such an appearance.

Case report

In 1976 a 24-year-old woman was referred to a general physician with spastic weakness of the right leg, which quickly resolved. Over the next three years she was seen intermittently with further symptoms affecting the right arm and leg, double vision, and slurred speech. Multiple sclerosis was diagnosed and several investigations performed. Visual evoked responses were normal, left temporal slow waves were present on electroencephalography, and computed tomography showed central atrophy (with large ventricles). She later developed complex partial seizures and was referred to a neurologist, who found non-fluent dysphasia and right hemiparesis but bilateral extensor plantar responses. The seizures were difficult to control but her overall condition improved slightly, only to deteriorate a year later.



Computed tomograms showing (left) large mass in frontotemporal area, suggesting glioma, and (right) central atrophy.

At that time slow waves were seen over the right frontotemporal area on electroencephalography, and a second computed tomogram, in October 1982, showed a large mass in the same area (figure (left)). A radiological diagnosis of glioma was accepted since malignant change may occur in multiple sclerosis plaques.[1] As it was thought that biopsy might worsen her condition without leading to any worthwhile treatment dexamethasone was given and the family told the prognosis.

One year later she suffered a severe relapse with spastic quadriplegia, internuclear ophthalmoplegia, pronounced cerebellar ataxia, dysarthria, and reduced visual acuity. Visual evoked responses were now abnormal, and a third computed tomogram (figure (right)) showed similar appearances to the first, obtained in 1979.

Comment

The resolution of the radiological lesion seen on the computed tomogram obtained in 1980 suggests that the lesion may have been a plaque of demyelination and not a malignant tumour. Published reviews of computed tomographic appearances in multiple sclerosis[2] have described the characteristic cerebral atrophy with ventricular enlargement seen in advanced cases. Acute plaques may appear as areas of low attenuation with or without enhancement after contrast injection. Later, well-defined areas of low attenuation may be seen in periventricular regions. Typically these lesions do not produce a mass effect. Two patients have been described, however,[3 4] in whom a multiple sclerosis plaque produced a shift of midline structures and was mistaken for a neoplasm. The nature of these lesions was confirmed histologically. The mass effect seen in our patient was more pronounced than in these two reports.

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, Urich 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.
[5] Sears ES, Tindall RSA, Zarnow H. Active multiple sclerosis. Enhanced computerised tomographic imaging of lesions and the effect of corticosteroids. *Arch Neurol* 1978;**35**:426-34.

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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 cyclopenthiazide 0·5 mg daily for two years.

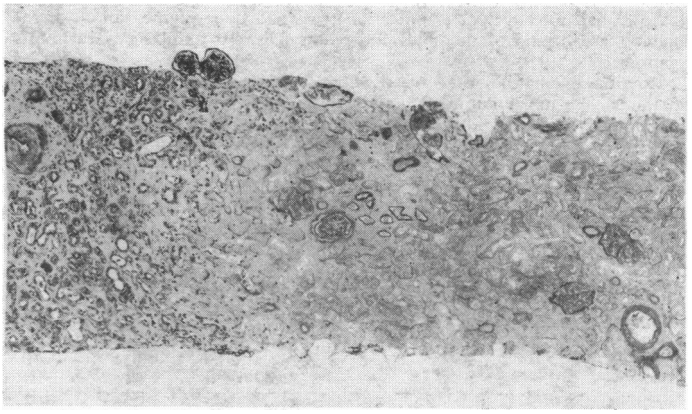
He had been given atenolol 100 mg on 18 November because his blood pressure was 240/115 mm Hg. Five hours after the first dose he had become faint and his blood pressure was 130/70 mm Hg. He was admitted to hospital three days later having been completely anuric since taking the single atenolol tablet; 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 × 20 (original magnification).

distal aortic thrombosis (Leriche's syndrome)[2] and renal artery thrombosis[3] 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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Rastafarianism and the vegans syndrome

Low serum vitamin B_{12} 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 B_{12} 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