they found no bone biopsy evidence of aluminium toxicity in a patient with a normal serum creatinine concentration who had received a total of 83 plasma exchanges with albumin solutions contaminated with aluminium.¹⁴ We do not agree, however, that the amount of aluminium delivered in infusions of albumin is too small to present a real risk.¹⁵ Detecting aluminium retention requires metabolic balance studies and bone biopsy and cannot be based on plasma aluminium concentrations alone.

As none of our patients was being treated by haemodialysis and only one (case 1) was receiving aluminium hydroxide as a phosphate binder (the two usual sources of aluminium in chronic renal failure), we conclude that the accumulation of aluminium in bone was a result of infusing albumin contaminated with aluminium.

Though aluminium induced osteomalacia is the best described effect of chronic aluminium toxicity, neurotoxicity is also well recognised⁷ and there is interest in the role of aluminium in Alzheimer's disease and other degenerative conditions related to aging.^{16 17}

Patients given infusions of contaminated albumin are receiving up to 100 times the amount of aluminium normally absorbed from the diet. Given the potential toxicity of aluminium, doctors should avoid the prolonged administration of large volumes of albumin (or other products contaminated with aluminium) to patients with renal impairment.

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References

- 1 Fell GS, Shenkin A, Halls DJ. Aluminium contamination of intravenous pharmaceuticals, nutrients, and blood products. *Lancet* 1986;ii:380.
- 2 Fell GS, Maharaj D. Trace metal contamination of albumin solutions used for plasma exchange. *Lancet* 1986;ii:467-8.
- 3 Maher ER, Brown EA, Curtis JR, Phillips ME, Sampson B. Accumulation of aluminium in chronic renal failure due to administration of albumin replacement solutions. *Br Med J* 1986;292:306.
- 4 Urbaniak SJ. Replacement fluids in plasma exchange. *Apheresis Bulletin* 1983;1(2):104-13.
- 5 McGinlay E, Mclay A, Boulton-Jones JM. Plasma exchange in the treatment of mesangiocapillary glomerulonephritis. *Nephron* 1985;40:385-90.
- 6 Boyce BF, Fell GS, Elder H, et al. Hypercalcaemic osteomalacia due to aluminium toxicity. *Lancet* 1982;ii:1009-12.
- 7 Willis MR, Savory JA. Aluminium poisoning: dialysis encephalopathy, osteomalacia, and anaemia. *Lancet* 1983;ii:29-33.
- 8 Malluche HH, Smith AJ, Abreo K, Faugere MC. The use of desferrioxamine in the management of aluminium accumulation in bone in patients with renal failure. *N Engl J Med* 1984;311:140-4.
- 9 Gardiner PE, Ottaway JM, Fell GS, Halls DJ. Determination of aluminium in blood plasma or serum by electrothermal atomic absorption spectrometry. *Analyst Chimica Acta* 1981;128:57-66.
- 10 Halls DJ, Fell GS. Determination of aluminium in dialysate fluids by atomic absorption spectrophotometry with electrothermal atomisation. *Analyst* 1985;110:243-6.
- 11 Kovalchik MT, Kaehny MT, Jackson T, Alfrey AC. Aluminium kinetics during hemodialysis. *J Lab Clin Med* 1978;92:712-6.
- 12 Alfrey AC. Aluminium. *Adv Clin Chem* 1983;23:69-91.
- 13 Charhon AS, Chavassieux PM, Meunier PJ. Serum aluminium concentration and aluminium deposits in bone in patients receiving haemodialysis. *Br Med J* 1985;290:1613-4.
- 14 Milliner DS, Shinabarger JH, Shuman P, Coburn JW. Inadvertent aluminium administration during plasma exchange due to aluminium contamination of albumin replacement solutions. *N Engl J Med* 1985;312:165-7.
- 15 Davenport A, Roberts NB. Serum aluminium levels in acute renal failure. *Lancet* 1986;ii:1397-8.
- 16 Candy JM, Oakley AE, Klinowski J, et al. Aluminosilicates and senile plaque formation in Alzheimer's disease. *Lancet* 1986;ii:354-6.
- 17 Ganrot PO. Metabolism and possible health effects of aluminium. *Environ Health Perspect* 1986;65:362-411.

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SHORT REPORTS

Anterior ischaemic optic neuropathy in the acquired immune deficiency syndrome

Anterior ischaemic optic neuropathy¹ is most common in elderly patients with temporal arteritis but may also occur in younger patients after acute hypotension or as a complication of hypertension, diabetes, or autoimmune disease. We report on a middle aged man in whom anterior ischaemic optic neuropathy was the presenting symptom of the acquired immune deficiency syndrome (AIDS).

Case report

A 45 year old white man presented in February 1986 with sudden onset of blurred vision in his right eye, deteriorating memory over the previous two years, and mild intermittent frontal headaches over the previous six months. He had worked in Zambia during 1970-3 and 1979-84 and had been sexually promiscuous, contracting gonorrhoea, non-specific urethritis, and genital herpes. He had not indulged in homosexual activity but had received a blood transfusion in 1980 while in Zambia.

Visual acuity was 6/36 in his right eye, which had a pale and swollen optic disc, especially over the upper half, with small haemorrhages on its surface. His right visual field showed an inferior altitudinal defect. He had several discrete lymph nodes in his groin, left axilla, and left posterior cervical triangle. His liver was slightly enlarged but not tender. Blood pressure and heart sounds were normal, and there were no carotid bruits. Formal psychometric testing confirmed mild impairment of verbal recall and immediate memory span with more severe impairment of his memory for words and faces.

Anterior ischaemic optic neuropathy was diagnosed clinically and confirmed by fluorescein angiography, which also showed delayed filling of the choroidal circulation in the upper nasal quadrant that was still evident in the early to middle retinal venous phase. When choroidal filling was complete the disc began to leak fluorescein, increasingly so in the late phase. The figure shows fluorescein staining of the wall of two short sections of the inferior temporal retinal vein, near the disc margin and just nasal and inferior to the fovea. His retinal circulation was otherwise normal.

Erythrocyte sedimentation rate was raised at 38 mm in the first hour, but routine haematological and biochemical variables (including blood glucose concentration) were otherwise normal. Fasting cholesterol and triglyceride concentrations were normal. Serological tests for syphilis, hepatitis B surface

antigen, and autoantibodies yielded negative results, but the serum titre of antibodies to human immunodeficiency virus was raised at 1/4000. Computed tomography of the brain showed nothing abnormal. Analysis of cerebrospinal fluid showed a normal cell count and total protein concentration and a high antibody titre to human immunodeficiency virus. Lymphocyte surface markers showed the characteristic features of infection with human immunodeficiency virus with selective reduction in numbers and percentage of T4 cells. Tests for toxoplasma and cytomegalovirus were negative.

Fluorescein angiography four months later showed complete resolution of all the changes. At his last ocular examination 10 months after the onset visual acuity was 6/18 and the upper half of the disc was atrophic. The inferior altitudinal visual field defect had not changed.

Fluorescein angiogram showing swollen disc with haemorrhages and staining of two sections of inferior temporal retinal vein.

Comment

This patient had subacute encephalitis, the commonest neurological manifestation of AIDS,^{2,3} which is characterised initially by mild cognitive changes and psychomotor retardation. Analysis of cerebrospinal fluid may show a mild pleocytosis, a rise in protein concentration, and a lowered glucose concentration.

The prevalence of ophthalmological signs in AIDS is at least 50% and is higher in those studied at necropsy.^{4,5} The commonest findings are microaneurysms, haemorrhages in the nerve fibre layers, and focal non-perfusion of small retinal vessels, producing cotton wool spots. These abnormalities are probably due to a microvascular retinopathy. Other intraocular complications include cytomegalovirus retinopathy, Roth's spots, and ocular candidiasis. Our patient's ophthalmological signs were caused by ischaemia of the optic nerve head from affected posterior ciliary arteries. The localised areas of fluorescein staining of the retinal veins suggest that the retinal circulation was also affected.

1 Miller NR, ed. *Walsh and Hoyt's clinical neuro-ophthalmology*. 4th ed. Baltimore: Williams and Wilkins, 1982:212-26.

2 Snider WD, Simpson DM, Nielsen S, et al. Neurological complications of acquired immune deficiency syndrome: analysis of 50 patients. *Ann Neurol* 1984;14:403-18.

3 Carne CA, Adler MW. Neurological manifestation of human immunodeficiency virus infection. *Br Med J* 1986;293:462-3.

4 Newsome DA, Green WR, Miller ED, et al. Microvascular aspects of acquired immune deficiency syndrome retinopathy. *Am J Ophthalmol* 1984;98:590-601.

5 Palestine AG, Rodrigues MM, Macher AM, et al. Ophthalmic involvement in acquired immunodeficiency syndrome. *Ophthalmology* 1984;91:1092-99.

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Disseminated cholesterol embolism presenting as neuromyelitis optica

Embolisation of cholesterol crystals from eroded aortic atherosclerotic plaques may result in various clinical manifestations affecting the kidney, the skin, the gastrointestinal tract, the heart, the retina, and the brain.¹

We report a case that suggests that neuromyelitis optica should be included in the clinical range of cholesterol embolism.

Case report

An 80 year old woman was admitted because of paraplegia and urinary incontinence of recent onset. Six months earlier she had experienced a transient loss of vision in the left eye, which had been attributed to optic neuritis. Physical examination showed dehydration, petechiae on the toes, flaccid paraplegia with bilateral Babinski's sign, and a well defined loss of sensation in response to heat at the level of T 9. Funduscopic examination yielded normal results. The results of biochemical tests showed an increased erythrocyte sedimentation rate (117 mm in the first hour) and renal insufficiency (blood urea nitrogen concentration 36.7 mmol/l, serum creatinine concentration 295 µmol/l). Antinuclear antibody was present at a titre of 1/320; antibodies to double stranded deoxyribonucleic acid and smooth muscle were absent. The test for lupus erythematosus yielded negative results, and there were no clinical or serological signs of systemic lupus erythematosus. No proteinuria was found, urine sediment was normal, and renal function improved after rehydration. Analysis of cerebrospinal fluid yielded unremarkable results, and myelography excluded medullary compression. Visual evoked potentials showed increased latency on the left side.

Neuromyelitis optica was diagnosed, and the patient received prednisolone 1 mg/kg/day without improvement of her neurological state. She died in a marasmic state four weeks later. At necropsy the thoracic and abdominal aorta showed ulcerated atheromatous plaques covered by friable material. The spinal cord showed extensive necrotic lesions at the thoracic level. Several distal branches of the anterior and posterior spinal arteries had been obliterated by needle shaped, crystalline clefts characteristic of cholesterol emboli (figure). In addition, hyaline thickening of small arterioles was noted. No specific changes were found in the brain except an area of demyelination in the optic chiasma. The eyes were not available for pathological studies. Examination of other tissues

Spinal cord at thoracic level (Luxol fast blue), showing extensive necrotic lesions of white and grey matter. Arrow indicates a cholesterol embolus in a branch of posterior spinal artery, and inset shows higher power view of cholesterol cleft in a spinal vessel.

showed disseminated cholesterol embolism with arterial reactions at different stages. Moreover, characteristic crystalline clefts were found in small and medium sized arteries of the kidneys, the liver, the stomach, the pancreas, the spleen, the adrenal glands, and the skin and in a mediastinal lymph node.

Comment

Neuromyelitis optica, or Devic's disease, is characterised by the association of optic neuritis with spinal cord lesions. This clinical entity has been described in association with viral infections, tuberculosis, and systemic lupus erythematosus but is most often idiopathic.²

Pathological and clinical data indicate that neuromyelitis optica is usually a demyelinating disease related to multiple sclerosis. Recently, attention has been drawn to peculiar vascular changes observed in some patients with the disease. These changes, reminiscent of those reported in patients with subacute necrotic myelopathy, are characterised by the proliferation of thickened, hyalinised small vessels in areas of severe demyelination.²

In our patient similar changes were found associated with cholesterol embolisation. Atheromatous emboli have previously been implicated in spinal cord infarcts,^{3,4} but, to our knowledge, this is the first description of neuromyelitis optica associated with cholesterol embolism.

Cholesterol embolism usually develops after surgery on a severely atherosclerotic aorta or after major vessel angiography, but, as in our patient, it may also occur spontaneously. The protean clinical manifestations may mimic systemic vasculitis, and laboratory findings may add to confusion as erythrocyte sedimentation rate is often increased, transient eosinophilia is common, antinuclear antibody may be present, and abnormal results of urine analysis are common.⁵

Neuromyelitis optica is viewed as a clinical syndrome with several possible aetiologies.² Our findings indicate that cholesterol embolisation should be considered to be a possible cause of the syndrome, especially in elderly patients with severe aortic atheromatosis.

1 Perdue GD, Smith RB. Atheromatous microemboli. *Ann Surg* 1969;169:954-9.

2 Lefkowitz D, Angelo JN. Neuromyelitis optica with unusual vascular changes. *Arch Neurol* 1984;49:1103-5.

3 Perier O, Demanet JC, Henneaux J, Nunes Vicente A. Existe-t-il un syndrome des artères spinales postérieures? A propos de deux observations anatomocliniques. *Rev Neurol (Paris)* 1960;103:396-409.

4 Yutani C, Imakita M, Ishibashi-Ueda H, et al. Cerebrospinal infarction caused by atheromatous emboli. *Acta Pathol Jpn* 1985;35:789-801.

5 Smith MC, Chose MK, Henry AR. The clinical spectrum of renal cholesterol embolisation. *Am J Med* 1981;71:174-80.

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