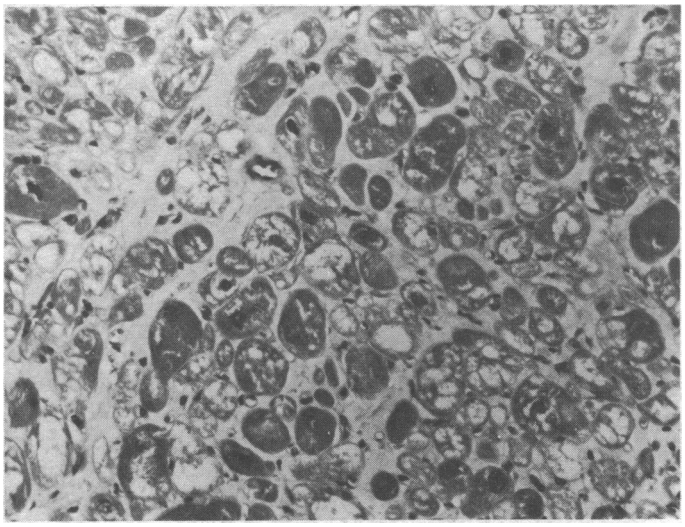
Cobalt cardiomyopathy in a patient on maintenance haemodialysis

Cobalt has been incriminated in cardiomyopathies in Quebec beer drinkers,[1] a person exposed to industrial cobalt,[2] and in patients on maintenance haemodialysis with[3] and without[4] cobalt therapy. We report a case of cardiomyopathy in a 17-year-old girl on maintenance haemodialysis who was given cobalt.

Case report

Membranoproliferative glomerulonephritis was diagnosed in this patient in 1969 when aged 9 and haemodialysis was started in 1974. She had intermittent hypertension from 1973 to 1976. Bilateral nephroureterectomy was performed in 1976 because of hypertension and ureteric reflux. From 1975 she had recurrent attacks of severe symptomatic anaemia which, despite iron and folic acid therapy, required frequent transfusions, causing fluid overload.



Photomicrograph showing an area of myocardial necrosis. (Haematoxylin and eosin. × 124.)

Cobaltous chloride 25 mg twice daily was given from August 1976 to April 1977 for the anaemia. In April 1977 she presented with dyspnoea and intermittent central chest pain. She was hypotensive and had raised venous pressure, gallop rhythm, and varying cardiac arrhythmias (supraventricular tachycardia and atrial fibrillation). A severe hyperkalaemia was probably due to the acidosis of poor tissue perfusion. Chest radiography showed globular cardiomegaly, and echocardiography indicated a moderate pericardial effusion and poor left ventricular contraction. Deterioration was rapid and intractable. In a situation of low cardiac output, raised venous pressure, hyperkalaemia, and haemodialysis a haemorrhagic pericardial effusion had to be excluded urgently. A pericardial window was fashioned at operation. Only 0·2 l of straw-coloured pericardial fluid was obtained. There was cardiomegaly and a pale myocardium with feeble contractions. Contractions did not improve after excision of part of the pericardium. The patient died a few days later from intractable biventricular failure.

Necropsy showed gross cardiac dilatation. The valves were normal and the coronary arteries had no atheroma. The myocardium looked strikingly unusual to the naked eye, with discrete pale patches due to subepicardial necrosis, quite unlike that seen in diffuse ischaemia. There was no histological evidence of pericarditis and muscle fibres were separated by fibrous tissue with relatively few inflammatory cells. The fibres were vacuolated with loss of striations and irregular, very large nuclei (figure). Some fibres contained PAS-positive granules. Stains for amylase and lipopolysaccharide deposition were negative. These changes were similar to those reported in men exposed to cobalt and also in experimental animals.[5] Assay of cobalt in necropsy specimens of myocardium by neutron activation analysis showed 8·9 parts per million (dried tissue). This is similar to concentrations found in other cases of cobalt exposure in maintenance dialysis. A "normal" value has been quoted as about 0·2 parts per million.[3]

Comment

The absence of gross pericardial effusion in this case, in contrast to the beer drinkers, may merely reflect the effect of haemodialysis on removal of fluid. The altered metabolism and nutrition of the dialysed subject may have rendered the myocardium more sensitive to cobalt toxicity. Experimental animals can be protected from cobalt toxicity by feeding high protein diets rich in SH-containing amino-acids. Nevertheless, the evidence in this case points to cobalt as the primary cause of the cardiomyopathy.

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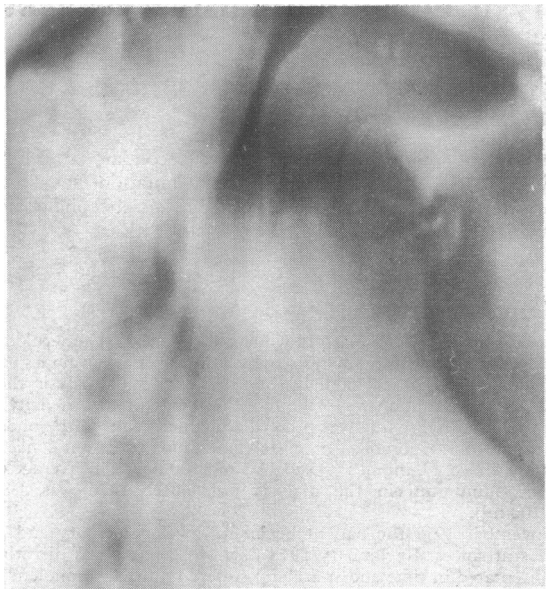
Value of oblique tomography in tracheal stenosis due to retrosternal goitre

Some time ago a patient with retrosternal goitre presented with stridor to our surgical department. Anteroposterior and lateral tomograms showed a normal calibre trachea, but severe stenosis of the trachea was found at operation. It was suggested that we should try oblique-view tomography in future problem cases. Since then we have collected three such cases. A computer search failed to show any reference to the use of oblique tomography in such cases.

Case 1

A 56-year-old woman presented with several years' history of swelling in the neck with a recent onset of difficulty in breathing. She was obese and had a goitre with retrosternal extension. Routine views showed no tracheal compression. She was advised to lose weight. Twelve months later she was again seen with noisy breathing. She had a definite stridor. Anteroposterior and lateral tomograms were again normal. Left oblique tomography showed pronounced narrowing of the trachea (see figure). This was confirmed at operation for a benign nodular goitre.



Oblique tomogram, 14-cm cut, showing pronounced narrowing of trachea.