Cells were considered abnormal only if they were clearly deformed as classified by Bessis⁴ as either echinocytes II or echinocytes III.

The proportion of echinocytes was not significantly affected in the controls, affected boys, or carriers by the method of blood sampling or number and duration of saline washes. In addition the purity of glutaraldehyde, osmolality, and duration of fixation had no effect. There was no relationship between age or sex and the proportion of echinocytes.

There were significant differences in the proportions of echinocytes between the controls and the affected boys ($P < 0.01$), and between the controls and the definite carriers ($P < 0.001$) (table). Nevertheless, there was considerable overlap between the controls and carriers and even between the controls and affected boys. There was no apparent correlation between the proportions of echinocytes and serum levels of creatine kinase in either the affected boys or the carriers.

Proportion of Echinocytes in Controls, Boys with Duchenne Muscular Dystrophy, and Carriers

	No.	Age Range (Years)	Percentage of Echinocytes (Mean $\pm$ S.D.)
Controls	17	10-50	3.86 $\pm$ 1.93
Boys with Duchenne muscular dystrophy	9	8-18	8.64 $\pm$ 6.12
Carriers { Definite	5	40-60	11.23 $\pm$ 8.08
{ Definite*	1	37	9.75
{ Possible	9	18-60	6.45 $\pm$ 3.66

*Manifesting carrier with severe weakness.

Comment

Though these findings to some extent confirm those of Matheson and Howland,¹ it seems unlikely that the test will prove more reliable than the estimation of serum levels of creatine kinase in detecting carriers. It might, however, prove helpful in the antenatal diagnosis of Duchenne muscular dystrophy should it prove possible to obtain samples of fetal blood in early pregnancy.

¹ Matheson, D. W., and Howland, J. L., *Science*, 1974, 184, 165.

² Miale, T. D., Frias, J. L., and Lawson, D. L., *Science*, 1975, 187, 453.

³ Matheson, D. W., and Howland, J. L., *Science*, 1975, 187, 454.

⁴ Bessis, M., *Nouvelle Revue Française d'Hématologie*, 1972, 12, 721.

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Immobilization Hypercalcaemia in Patients on Regular Haemodialysis

Immobilization of normal people causes reabsorption of calcium from bone,¹ a small rise in serum ionized calcium,² and, rarely, frank hypercalcaemia.³ The hazard is increased when patients with renal osteodystrophy are immobilized because of pathological fractures.

Case Reports

Case 1.—A man aged 27 with obstructive uropathy from childhood who had been on regular haemodialysis for six years developed renal osteodystrophy despite prolonged treatment with vitamin D, changed in October 1972 to dihydrotachysterol (DHT) with aluminium hydroxide 30 ml/d for indigestion. In May 1973 he sustained a fracture of the femoral shaft after minor trauma and was immobilized in a Thomas splint. Two weeks later conjunctivitis with corneal calcification developed and the serum calcium reached 3.9 mmol/l (15.8 mg/100 ml) (fig.). DHT was stopped and the dialysate calcium reduced to 1.0 mmol/l (4 mg/100 ml). Despite these measures the serum calcium fell slowly and was still 3.6 mmol/l (14.6 mg/100 ml) after 30 days. The conjunctivitis persisted for two months.

Case 2.—A 46-year-old woman with chronic pyelonephritis started regular haemodialysis in January 1973 after a uraemic fit in which she fractured her right femur. This was treated by pin and plate, and bone biopsy confirmed

Case 1 Serum Alkaline, phosphate, and Alkaline phosphatase levels and treatment used.

renal osteodystrophy. She was given daily doses of DHT 0.25 mg and aluminium hydroxide gel 30 ml until February 1975, when DHT was changed to 1- α -hydroxycholecalciferol (OHCC) 2 μ g/d because of the persistence of histological osteitis fibrosa and osteomalacia. In March the pin and plate had worked loose, and since the fracture was still ununited she was admitted for bone grafting. OHCC was stopped on admission but after 16 days of immobilization her serum calcium had risen to 3.3 mmol/l (13.4 mg/100 ml). Ten days after she was mobilized the serum calcium was 2.9 mmol/l (11.8 mg/100 ml).

Case 3.—A 19-year-old man with medullary cystic disease presented five years previously with growth retardation, myopathy, and severe osteitis fibrosa. Bone disease persisted despite treatment with vitamin D and subsequently daily doses of DHT 0.5 mg and aluminium hydroxide gel 60 ml. In September 1974 he started regular haemodialysis, and in the November, DHT was changed to 1,25-dihydroxycholecalciferol ((OH)₂CC) 1 μ g/d. On 3 February 1975 he was admitted for pinning of a slipped femoral epiphysis and (OH)₂CC was stopped. By 18 February the serum calcium had risen from 2.6 to 3.7 mmol/l (10.5 to 14.9 mg/100 ml). He was mobilized on 25 February, and by 5 March his serum calcium had fallen to 3.0 mmol/l (12.2 mg/100 ml). This fall towards normal continued.

Case 4.—A man aged 31 with obstructive uropathy since birth and old renal rickets started regular haemodialysis 15 months previously. He had histological evidence of osteitis fibrosa and osteomalacia for which he received calcium carbonate 6 g/d for seven months, aluminium hydroxide gel 30 ml/d for five months, and DHT 0.25 mg on alternate days for two months. All drugs were stopped on admission but reinstituted 18 days later, DHT in a dose of 0.75 mg/d. Three days later his serum calcium had risen from 2.5 mmol/l (10.2 mg/100 ml) on admission to 2.9 mmol/l (11.6 mg/100 ml) and it continued to rise to 4.5 mmol/l (18.2 mg/100 ml), when he developed confusion, nausea, and vomiting despite stopping the DHT. Serum calcium fell slowly to 2.6 mmol/l (10.6 mg/100 ml) after two months.

Discussion

Immobilization was apparently the main cause of the hypercalcaemia in cases 1 to 3 and a contributory cause in case 4, in which the patient also received DHT and calcium carbonate⁴ at the crucial time. Fluctuations in plasma phosphatase due to variable adherence to the prescribed dose of aluminium hydroxide⁵ may also have contributed in two cases. A critical review of all treatment is necessary when patients with renal bone disease are immobilized, and careful monitoring of serum calcium and phosphate is mandatory.

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