BILATERAL FRACTURE OF THE FEMORAL NECK DURING A HYPOCALCAEMIC CONVULSION

A CASE REPORT

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Vitamin D deficiency occurs in up to 24% of the Asian immigrant population in the United Kingdom, but pathological fractures are relatively uncommon. We report a case of bilateral fracture of the femoral neck caused by a convulsion secondary to dietary-induced hypocalcaemia. To our knowledge such a sequence has not previously been reported.

Bilateral fractures of the humerus and femur, and fracture-dislocations of the shoulder and hip are recognised to occur in epileptic fits (Powell 1960; Jacobs, Patterson and Schultz 1970; Shaw 1971). Such injuries used to be common also among patients treated for psychiatric disorders by drug-induced convulsions (Meduna 1935; Hamsa and Bennett 1939; Meduna and Friedman 1939; Gissane, Blair and Rank 1940) or by electrically induced convulsions (Cerletti and Bini 1938; Hamsa and Bennett 1939; Gissane *et al.* 1940; Powell 1960), but the incidence has markedly decreased since the introduction in 1957 of muscle relaxants as an adjunct to therapy (Paterson and King 1957). Fractures and fracture-dislocations have also been reported following uraemic seizures and after water-soluble myelography (Morrey and O'Brien 1977). We could find in the literature only two cases of bilateral hip injury resulting from hypocalcaemic convulsions; both occurred after parathyroidectomy (Davies and Friedman 1966).

CASE REPORT

A 30-year-old Kenyan Asian woman was seen in the Accident Department of The Middlesex Hospital in January 1984. She had been breast-feeding her six-week-old baby and next remembers waking with her baby beside her on the bed. She was unable to move because of severe pain in both hips.

She had been resident in the United Kingdom for 12 years, and was a vegan who frequently ate chapatis. She had one other child aged two years who was healthy. There had been no complications during the pregnancy or delivery of either child. She gave no history of limb pain or of proximal muscle weakness. Three days before her admission she had noticed tingling in her hands and

feet and could recall two other lapses of consciousness; in neither had she caused herself injury.

On examination she was drowsy but orientated. There were no localising neurological signs. She had a graze on her forehead and a laceration on her tongue. Her reflexes were all increased and she had markedly positive Chvostek's and Trousseau's signs. Springing of the ribs caused pain anteriorly over the right fourth rib. Both legs were laterally rotated and any movement was extremely painful.

Radiographs showed subcapital fractures of both femora with Grade 4 displacement (Fig. 1) as well as a Looser zone in the right fourth rib. A skeletal survey revealed no other abnormality apart from an overall decrease in bone density. Her haemoglobin concentration was 11.8 g/dl with a mean corpuscular volume of 90 fl. Her serum biochemistry was abnormal with a serum calcium level of 1.53 mmol/l (normal 2.20 to 2.55 mmol/l), a serum phosphate level of 1.58 mmol/l (normal 0.6 to 1.3 mmol/l) and an alkaline phosphatase level of 976 iu/l (normal 20 to 70 iu/l). The levels of magnesium, albumin, total protein, urea and electrolytes were all normal. Her hypocalcaemia was treated with a combination of intravenous calcium gluconate (10%), calcitriol and Sandocal. Within 36 hours her calcium level was normal. She was given bromocriptine, 2.5 mg twice daily, to suppress lactation and thereby prevent further calcium loss.

Thirty-six hours after admission both fractures were reduced and internally fixed, using compression screws. A technetium-99m bone scan three days after operation showed a "cold" area in the right femoral head suggestive of an avascular segment. Her wounds healed uneventfully and after six weeks' bed rest she had hydrotherapy for two weeks and was then allowed up, taking no weight on the side showing an abnormal scan.

Three months later she had a complete range of painfree movement at both hips, radiographic union of both fractures (Fig. 2) and her bone scan now showed a normal vascular pattern in both femoral heads. Her cal-

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Fig. 1

Anteroposterior radiograph showing subcapital fractures of the femora, both with Grade 4 displacement.

Fig. 2

Three months after operation both fractures are united.

cium metabolism was normal and was maintained by dietary adjustment and calcium supplements.

DISCUSSION

Vitamin D deficiency occurs in up to 24% of the Asian immigrant population in the United Kingdom (Ford *et al.* 1972), but orthopaedic complications are uncommon. The causes of osteomalacia in this immigrant population include a high phytate diet (Brude and Callow 1934; Berlyne *et al.* 1973) and lack of exposure to sunlight (Rab and Baseer 1976). At the levels found in chapatis, phytate has been shown to be a potent inhibitor of calcification *in vitro* (Van den Berg, Hill and Stanbury 1972) and of calcium absorption from the gut (McCance and Widdowson 1942; Sognen 1964). Examples of phytate-associated osteomalacia and negative calcium balance have been reported (Wills *et al.* 1972; Berlyne *et al.* 1973; Reinhold *et al.* 1973).

Of particular relevance to our patient are the marked effects which pregnancy and lactation have on

calcium balance. The fetus requires 30 g of calcium during the last three months of pregnancy (World Health Organisation 1962) and breast milk contains 8 mmol/l of calcium (Department of Health and Social Security 1977); that is, three times the normal serum concentration. Unless the dietary intake is adequate, pregnancy and lactation severely upset calcium metabolism.

We suggest that in this patient a pre-existing negative calcium balance was exacerbated by the pregnancy and subsequent lactation, reducing the serum calcium to below a critical level and thereby causing convulsion.

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