CASE REPORT

Pathologic femoral neck fracture due to renal osteodystrophy is rare. We report the case of a young adult patient with chronic renal failure who presented with bilateral spontaneous femoral neck fractures due to renal osteodystrophy. The pathophysiology of renal osteodystrophy and the treatment of hip fractures in patients with renal failure is discussed.

CASE REPORT

A 23-year-old male adult was referred to us because of pain in both hips, that had lead to immobilisation for three months. He had been operated for myelomeningocele at the lumbosacral level just after birth, but as a sequel he had a paralytic bladder and obstructive uropathy resulting in renal problems. He had been diagnosed as having chronic renal failure seven months previously and had received dialysis three times a week for 3 months.

Pelvic X-ray examination showed bilateral femoral neck fractures with surrounding sclerosis (fig 1). There was no history of trauma, seizure, steroid medication, fluoride treatment, alcohol abuse or smoking. On physical examination there was no active bilateral motion of the hips. At that time he had moderate anemia and serum creatinine was 5.65 mg/dl (N: 0.5-1.6 mg/dl), urea was 107 mg/dl (N: 7-21 mg/dl), calcium was 7.9 mg/dl (N: 8.1-12.2 mg/dl), phosphorus was 6.7 mg/dl (N: 2.6-5.9 mg/dl), vitamin D was 8 ng/ml (N: 9.2-34 ng/ml) and parathormone level was 147 pg/dl (N: 9-60 pg/dl).

Pelvic CT was performed and showed bilateral reactive bony sclerosis and nonunion (fig 2). Dual-energy X-ray absorptiometry of the lumbar spine and hips revealed values well below normal, which were accepted as osteopenia. Bilateral hydronephrosis and thickening of the bladder wall were found on abdominal ultrasonography.

After normalising the complete blood count and electrolyte values by giving erythrocyte suspensions and applying dialysis, a cemented total hip arthroplasty was performed bilaterally in two separate operations (fig 3). He received appropriate postoperative physical therapy and rehabilitation.

Fig. 1. — Pre-operative radiograph showing bilateral femoral neck fractures.

SPONTANEOUS BILATERAL FEMORAL NECK FRACTURES IN A YOUNG ADULT WITH CHRONIC RENAL FAILURE

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At six months both hips had recovered a normal range of passive motion. He had no pain and could walk easily without support.

**DISCUSSION**

Bilateral fractures of the proximal femur are uncommon. Possible causes have been identified as trauma (8), abnormal anatomy (2), seizures (1, 13, 17) and osteoporosis (6, 12). Bilateral fractures of the proximal femur have also been reported in patients with chronic renal failure, especially when they were receiving fluoride therapy (6).

Renal osteodystrophy encompasses a number of skeletal abnormalities, including osteitis fibrosa, osteomalacia, hyperosteoïdosis, osteosclerosis and several types of developmental problems in children (4). Although the occurrence of fractures in patients with these skeletal abnormalities has been established (4), pathologic fracture of the femoral neck (9) is rare and bilateral occurrence is all the more uncommon.

The mechanism by which renal osteodystrophy occurs in patients with glomerular failure begins with failure of filtration, which results in phosphate retention (15). Hyperphosphatemia (5) plays a key role in renal osteodystrophy similar to ectopic calcification. The combination of diminished tubular filtration and increased serum phosphate concentration causes a decrease in 1,25 dihydroxyvitamin D synthesis. These changes and the increase in the amount of phosphate in gastro-intestinal cell cytoplasmic matrix cause a significant decrease in calcium absorption from the gastro-intestinal tract (15). Despite the adequate oral intake of calcium and vitamin D, calcium is only minimally absorbed. The profound reduction in serum calcium promotes a marked secondary hyperparathyroidism, usually a clear-cell hyperplasia affecting all four glands (14, 15). As a result, the serum calcium rises partially to near normal values, at the expense of the bones. This increase in ionic calcium places the patient at risk for ectopic calcification and, less often, for ossification, by exceeding the critical solubility product for CaHPO₄ (14, 15). This type of calcification occurs in the conjunctivae, the blood vessels, the skin and the peri-articular tissues (15, 18).

Besides this, three different syndromes may affect patients with chronic renal failure. Severe nutritional osteoporosis may develop in some of the patients who are very ill and undernourished (18). In some others, aluminum toxicity may occur. These patients take aluminum hydroxide gels orally to decrease the blood phosphate concentration. Normally aluminum is excreted through the kidneys but, in patients with renal failure, it is retained and deposited in the brain and the bones (15). Aluminum alone can cause a neurodepressant or an osteomalacic-like syndrome, or both,
and each is refractory to treatment and may require the use of chelating agents such as desferrioxamine to diminish the concentration of the metal \(7\). The third possible complicating metabolic bone disorder may occur due to excessive parathyroid hormone production and secretion beyond the level needed to maintain a normal serum calcium concentration by the hyperplastic parathyroid glands (tertiary hyperparathyroidism). Such individuals, in whom the serum calcium concentration exceeds 12 mg/dl, are at risk for the development of progressive extraskeletal calcification or even calciphylaxis (ischemic lesions of the soft tissues due to small-vessel calcification) and may require partial parathyroidectomy for control \(10, 15\).

The combination of a hip fracture and renal failure is associated with high mortality \(11\). The one-year mortality rate from the hip fracture event was found to be nearly two and a half times higher in the dialysis patients compared with the general population \(3\). Related medical problems, including diabetes and cardiovascular disorders as a cause of renal disease beside the endocrine, cardiovascular, gastro-intestinal and infectious conditions concomitant with renal insufficiency and its therapy, contribute to increased mortality \(11\).

In the treatment of hip fractures in patients with renal failure, recent reports favored operative treatment. Schaab \(16\) reported 11 cases of femoral neck fracture in 11 patients with chronic renal failure, seven of whom underwent operations. Morbidity and mortality were analysed to compare operative and conservative management of femoral neck fractures in dialysis patients. All fractures occurred in older individuals, who had been dependent on dialysis for an average of 9.2 years. Operative management was found to be superior to conservative treatment as far as complications and mortality rates were concerned.

Klein \(11\) reported the first-year mortality after surgical treatment of hip fractures in patients with renal failure to be 38\%. That study included nine fractures in eight patients with an average age of 63 years; six patients were dependent on dialysis and two patients had functioning renal transplants. Three patients died within one year of the operation for reasons not associated with surgery.

The authors stated that operative treatment and aggressive mobilisation allow patients with renal failure to regain ambulation and return to the level of independence they had before their hip fracture and to avoid skin breakdown. In that study, delaying the surgery until the patients were considered to be in a medically stable condition, as judged by the nephrologists on the basis of haematocrit (greater than 25\%), serum potassium levels (generally less than 5.5 mEq/L) and fluid loading status, has been reported as being useful to decrease postoperative complications.

In our patient, who was a younger adult than the patients described by Klein \(11\), chronic renal failure was the only cause of osteopenia owing to renal osteodystrophy, resulting into spontaneous bilateral femoral neck fractures. It is therefore, important to evaluate the severity of renal osteodystrophy and to take therapeutic measures such as the control of phosphorus by phosphate binders and the use of vitamin D analogs to suppress hyperparathyroidism. In conclusion, we emphasise the high complication and mortality rate of the dialysis-dependent patients \(3, 11, 16\). It is suggested that providing stable medical conditions before surgery, with a careful team approach of nephrologist and surgeon, and a close medical follow-up, can limit short term complications and mortality.

REFERENCES
