Simultaneous bilateral hip fractures secondary to hypocalcemic convulsion—Case report and literature review

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1. Introduction

Simultaneous bilateral hip fractures are uncommon, but have been identified in association with epileptic fits when excessive muscle contractions place unusual stress on the bone. Certain medical conditions may predispose a patient to this rare mechanism by diminishing bone strength. One such condition is renal osteodystrophy (ROD) whilst the combination of uremia and hypocalcemia can cause bone demineralisation and severe osteomalacia. Pathological fractures may occur when hypocalcemic convulsions exposed bone to the above-mentioned unusual stresses. To the best of our knowledge, the literature has reported few similar cases. This study reports a rare case of bilateral hip fractures followed by hypocalcemic convulsion and reviews the literature to examine the pathophysiology and treatment algorithm of this rare injury.

2. Case report

A 57-year-old man was witnessed to develop tonic and clonic seizures during a regular hemodialysis session. His seizures were stopped with using intravenous diazepam and calcium gluconate infusion. After the convulsions ceased, the patient complained of bilateral hip pain. Plain radiographs showed right femoral neck transcervical fracture and left femoral neck basicervical fracture (Fig. 1).

The patient had chronic renal failure secondary to chronic glomerulonephritis and had received regular hemodialysis as an out-patient for 2 years. Moreover, the patient was diagnosed with secondary hyperparathyroidism 3 months previously. The parathyroid hormone (PTH) concentration was significantly elevated (1300 pg/ml; normal range, 11–62 pg/ml). Additionally, the dual-energy X-ray absorptiometry (DEXA) showed significantly reduced bone density (the patient had average bone mineral density of 269 mg/cm², which is 53.48% bone mineral density of young reference group with T-score about −2.84). Laboratory findings revealed patient hemoglobin concentration of 7.9 g/dl (normal range, 14–18 g/dl), blood urea nitrogen (BUN) level 49 mg/dl (normal range, 7–20 mg/dl), creatinine level 9.8 mg/dl (normal range, 0.6–1.3 mg/dl), serum albumin level 4.3 g/dl (normal range, 3.4–5.0 g/dl), serum calcium 6.5 mg/dl (normal range, 8.4–10.2 mg/dl). Serum levels of sodium and potassium were normal. Computed tomography (CT) scanning of the brain revealed no focal brain lesion.

The patient’s seizures were controlled with anti-epileptic drugs and his hypocalcemia was treated with a combination of intravenous calcium gluconate (10%), oral calcium and vitamin-D supplements. Five days following admission, the left hip fracture was internally fixed with a 135° dynamic hip screw. Cemented bipolar hip hemiarthroplasty was performed on the right side (Fig. 2). The patient’s wounds healed uneventfully and he was allowed to discharge after 1 week.

The patient was told to ambulate with a wheelchair for 6 weeks after the operation and then ambulate with walker support for another 8 weeks. Plain radiographs performed at 3 months showed union of left hip fracture and no significant change in right hip endoprosthesis. The patient could walk with waddling gait at his last visit, approximately 1 year and 2 months post-operatively.

3. Discussion

The hip is a biomechanically vulnerable area and thus a common site of fractures. Fractures may occur when the bone is subjected to unusual loads or when a pre-existing medical condition reduces bone strength. Chronic renal failure is characterised by progressive bone disease, including osteitis fibrosa, osteomalacia, hyperosteo-dosis and osteosclerosis in association with marked alterations in calcium and phosphorus metabolism, resulting in secondary hyperparathyroidism, also known as renal osteodystrophy (ROD). In this situation, bone quality is thus reduced and lacks mineral content, which is reflected by the low bone mineral density. The reduced bone strength in patients with ROD increases their susceptibility to fractures in the event of powerful muscular contractions occurring as a result of generalised convulsions.

A literature review identified only three reported cases of bilateral hip fractures caused by renal-failure induced hypocalcemic convulsion.
The pathophysiology makes this area most susceptible to seizure-related fractures because it serves as the lever arm of the hip joint which carries all the acting muscle and weight bearing forces, which is a danger point of torque concentration.8 The patient was diagnosed with secondary hyperparathyroidism, which is characterised by excessive secretion of PTH by the parathyroid glands in response to hypocalcemia. Despite administration of regular calcium supplements, the patient did not comply well with the therapeutic regimens. The serum calcium levels during the previous 3 months before the injury were 5.5, 5.4, and 5.7 mg/dl. The patient experienced several episodes of short duration tetany during the previous 2 months, each subsiding after administration of calcium supplements.

Dialysis patients were observed to have higher 1-year mortality rate following hip fractures than the general population.2 Surgery to help patients rapidly regain mobilisation can help them resume previous levels of independence. Surgical options for treating displaced femoral neck fracture are prosthetic replacement surgery and screws fixation. In uremic patients with secondary renal osteodystrophy, the bone is highly osteoporotic making it more difficult to achieve a stable implant-bone construct for fracture fixation. This is especially true for femoral neck fracture whilst higher bone density results in more stable intrinsic fixation.8 Furthermore, avascular necrosis of the femoral head occurs in 11–18% of patients with chronic renal failure making prosthetic replacement arthroplasty a better option.7 On the left side, the femoral neck fracture type was displaced and bascervical. The standard treatment is internal fixation with dynamic hip screw plus an additional anterotator screw.

In conclusion, it should be emphasised that primary care physicians should be aware and alert to the possibility of fractures in patients with hypocalcemic convulsions. Orthopaedic surgeons and nephrologists should work together as a team to reduce short-term complications and mortality in uremic patients with hypocalcemic hip fractures.

### References