Spasm Induced Spontaneous Bilateral Hip Fractures

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Citation

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Abstract

A previously unreported case of bilateral spontaneous fractures of the femoral necks is presented. Spasmodic muscular contractions due to tubercular spondyloarthropathy have not previously been instigated as a cause of bilateral hip fractures.

INTRODUCTION

We report on a case of a 75 year old man with paraparaesis (Grade IV of Tuli classification) [11], having suffered spontaneous bilateral fractures of the femoral neck due to flexor spasms. These spasms were due to longstanding tubercular spondyloarthropathy of the upper thoracic spine.

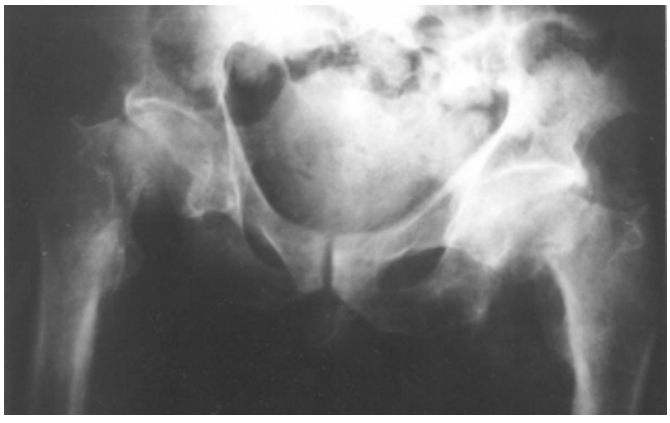
CASE REPORT

A seventy-five year old man with paraparaesis presented with history of abnormal crepitus and mild pain in both hip regions. The patient had longstanding tuberculosis of the upper thoracic (T5-T6) spine. Antero-lateral decompression (ALD) of spine had been performed 6 years ago for the treatment of grade IV spastic paraplegia, intermittent flexor (hip and knee) spasms of ilio-psoas and hamstring muscles and bowel-bladder involvement at that time. He had partial neurological recovery after the surgery. At the time of presentation at our institute, he had complete control of bowel and bladder functions, grade II power at hip, knee and ankle and partial sensory deficit. Intermittent flexor spasms with spasticity in both lower limbs had persisted after undergoing surgery. He was confined to a wheelchair. The patient complained of pain and crepitus arising from the left hip of one-month duration, similar complaints were noted on the right side two weeks later. The pain was exacerbated during flexion of hips. There had been no previous history of trauma, epilepsy, convulsions, renal disease, alcoholism or fluoride treatment. His general physical examination was unremarkable except for spine and hips. There was no inguinal lymphadenopathy.

Complete blood count, erythrocyte sedimentation rate, serum alkaline phosphatase and calcium and phosphate levels were all normal as were parathyroid hormone levels, T3, T4 and TSH (thyroxine and thyroid stimulating hormones). His values for 24-hour urinary excretion of calcium and phosphates, serum creatinine, creatinine clearance and serum testosterone were also normal. The radiographs showed bilateral displaced femoral neck fractures, osteopenia, and osteoarthritis of left hip without any evidence of tubercular pathology (Fig.1). The fractures were managed non-operatively because the patient had been confined to a wheelchair. Chemical or surgical neurectomy was not performed as lower limb sensation was partly spared. The patient was put on centrally acting muscle relaxants (Baclofen) and analgesics. Both fractures went into non-union. His pain resolved after 3 weeks.

Figure 1

Figure 1: X-ray of pelvis showing bilateral displaced femoral neck fractures



DISCUSSION

The common etiological factors identified for these very rare fractures have been reported to be trauma[5]; stress[2]; hypocalcaemic, epileptic, drug induced or electrically induced convulsions.[4,8,9,10] It has also been reported in cases

of ankylosing spondylitis[7], cirrhosis[6], renal disease[1] and osteomalacia[3]. Paraplegia with flexor spasms has not been reported as a cause of bilateral spontaneous displaced femoral neck fractures. It is well known that bone mineral density decreases after a spinal injury in 5 years time to fracture threshold levels. In the present case, the cause of fracture seems to be a combined effect of repeated stress from intermittent flexor spasms and osteoporosis secondary to immobilisation.

This patient demonstrates the fragility of hips in the elderly, after a period of immobilization. The stresses of frequent spastic movements may help to negate the effect of immobilization-induced osteoporosis, but when imposed on already weakened senile bone, can cause bilateral femoral neck fractures. This might be so because stress fractures in the intracapsular regions do not heal readily and osteopenia further impedes union and therefore predisposes to fracture displacement. Although relatively painless and inconsequential in paraplegic patients, these fractures maybe prevented by initiating prophylactic medical treatment for osteoporosis (SERMS and Alendronate) and surgical treatment (chemical or surgical rhizotomy, neurectomy, cordotomy, Baclofen infusion pumps etc). Patients with spasmodic muscular contractility in whom prolonged immobilization is anticipated are at risk.

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