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Case Report

Volume 2 Issue 1 *Journal of Orthopaedics and Skeletal Medicine*

Unusual Etiology for Bilateral Insufficiency Fracture of the Femoral Neck

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Citation: Zitouna K, Ben Lassoued N, Barsaoui M, Drissi G and Kanoun ML (2017) Unusual Etiology for Bilateral Insufficiency Fracture of the Femoral Neck. J Orthop Skeletal Med 2(1): 101

Introduction

The simultaneous and bilateral femoral neck fracture is rare. An underlying cause must be considered and actively sought if a low-energy trauma is the cause.

Case report

We report the case of a 45 year old male who consulted our emergency for bilateral femoral neck fracture following a traumatic fall from the bed occurring the same day. The anamnesis found a cerebral meningioma four years ago with bilateral blindness as sequelae. Due to the seizure risk, the patient was put under phenobarbital.

The patient was neither smoking nor alcohol drinker; he was autonomous in home, but helped by a third party for daily outings.

Clinical examination was poor and not contributing to found a metabolic, endocrine or neoplastic etiology.

The Xray showed a bilateral femoral neck fracture Garden IV without obvious decrease in bone mineralization or other abnormalities of bone matrix.

The standard laboratory tests associated with calcémia and thyroid function tests were correct apart from the elevation of alkaline phosphatase to 251 U / L (nl: 40-150). The bone resorption markers such as N-telopeptide of type I collagen (NTX) and carboxy-terminal telopeptide of type I collagen (ICTP) have not been made. The tumor markers were negative. A CT scan confirmed the presence of both fractures but showed no pathological process (Figure 1). A magnetic resonance imaging pulled same conclusions.

Due to the high risk of osteonecrosis of the femoral head, bilateral metal-on-Polyethylene cemented arthroplasty was decided.

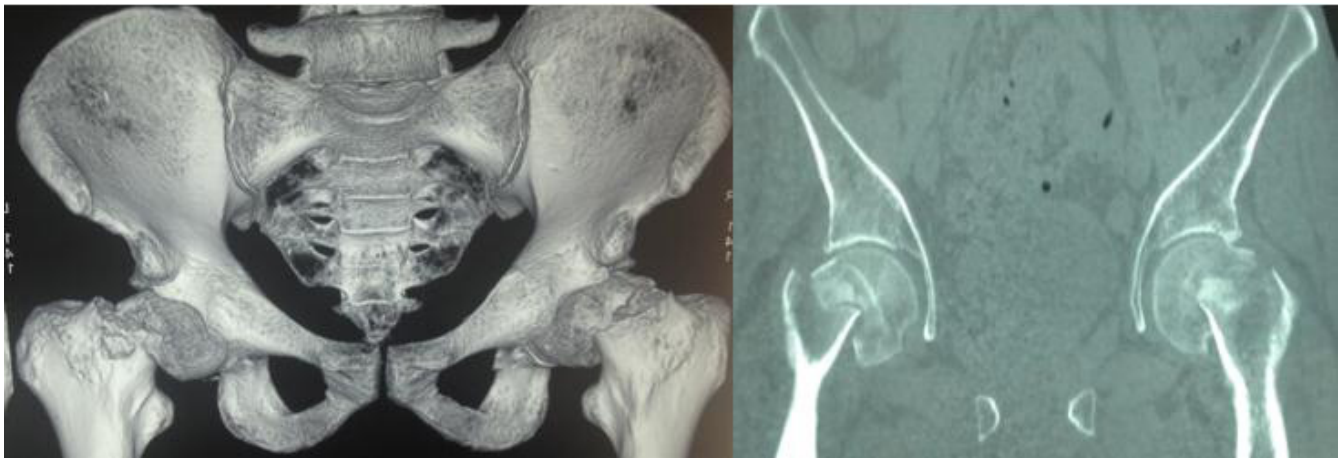

Figure 1: Pelvic scan demonstrates bilateral Garden IV fracture of femoral neck without signs of malignancy

Two interventions were scheduled, two weeks apart, beginning with the more painful side.

Histological examination of the femoral head has not showed neoplastic cells but found in osteoporosis.

Bone mineral density measured by dual energy absorptiometry confirmed the decrease bone mass with a T-score -2.3 SD at the lumbar spine and -2.1 at the femoral neck. The patient was put under bisphosphonates, vitamin and calcium supplementation associated rules lifestyle modifications.

Actually, 2 years post-operatively, the patient regained his initial level of activity without bone pain (Figure 2). The diagnosis of bilateral hip fracture of osteoporotic bone due to chronic use barbiturates was made.

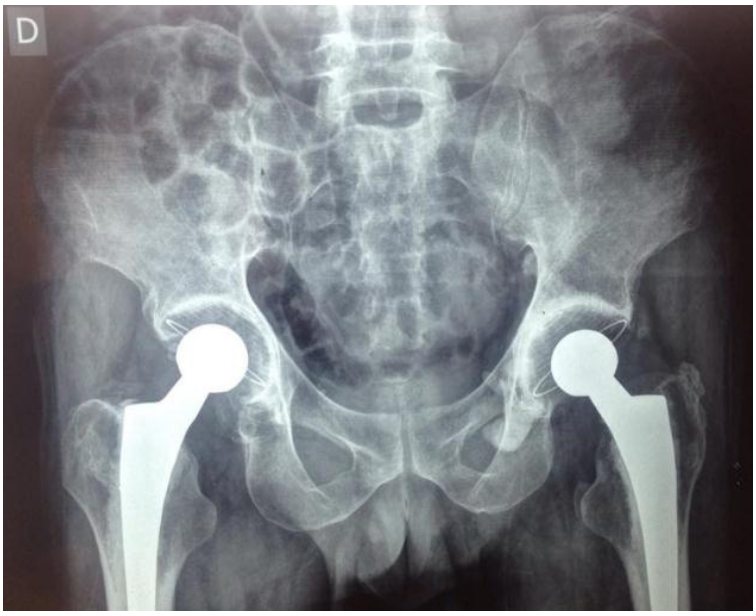

Figure 2: Postoperative radiographs after bilateral femoral head arthroplasty

Discussion

Traumatic hip fracture is a common injury that is seen most often in the Old person. His two-sidedness is uncommon and occurred after a weak energy trauma, should prompt the therapist to seek an underlying cause.

Some publications about bilateral traumatic fractures by cervical bone failure have been reported and implicated several etiologies: osteomalacia (by nutritional deficiency in the elderly, following an anorexia nervosa gestational, metabolic), idiopathic osteoporosis, hyperparathyroidism, hypocalcemia induced by renal failure or abuse of narcotics [1-8].

Bone fragility can also be pharmacologically induced [9,10]. Among the drugs like barbiturates and corticosteroids, chronic use of enzyme inducing antiepileptic drugs (AEDs) exposes of multiple side effects including osteoporosis and osteomalacia [9,11]. The mechanism of action of these molecules on bone metabolism remains unknown and widely debated. For Pack., *et al*. osteopenia would be the result of the enzyme-inducing effect of these molecules [12]. Indeed, it suggests that the induction of cytochrome P450 enzyme system is causing an increase in the catabolism of vitamin D. Furthermore, Pregnane X Receptor (PXR) can be activated by a variety of pharmaceutical agents including phenytoin, phenobarbital, carbamazepine and rifampicin. Emerging evidence shows that these PXR activators can increase the expression of the 24-hydroxylase (CYP 24), a vitamin D receptor target gene in cultured cells and in vivo in mice. CYP 24 is an enzyme that directs the side chain oxidation and cleavage of 25 (OH)2 D3 and 1β, 25 (OH)2 D3 to carboxylic acid end products (calcitroic acid), resulting in lower cellular concentration of active vitamin D. This induces a state of vitamin D deficiency and results in hypocalcemia, secondary hyperparathyroidism and increased bone turnover predisposing to low bone density and bone loss.

The direct inhibitory effect on the proliferation of osteoblasts, the decrease in calcium absorption and endocrinopathy are other causes suggested in the pathophysiology of this fragility bone induced [13]. An increased risk of fracture and / or bone loss is also observed with antiepileptic not enzyme inducers such as sodium valproate suggesting the existence of other mechanisms [14].

The fracture risk is increased by chronic use of IAIA. Also this risk is increased in epileptics treated with enzyme inducers since the bone insufficiency induced are added the central pharmacological side effects (drowsiness, dizziness) on the one hand and the trauma throughout seizure at the other hand [15]. Twenty observations of bilateral and concomitant fractures of the femoral neck at the waning of seizures were also reported [16].

Although these fractures occur on major seizures, true fractures trauma unconstrained were reported in epilepsy suggesting bone fragility induced by treatment [17,18]. Sariyilmaz., *et al*. reported the original observation of bilateral cervical stress fracture of the femur lasting for 3 months in a 26 years under epileptic carbamazepine [19]. The etiological evaluation concluded that treatment induced fracture in this case.

To prevent a possible fracture in patients taking antiepileptic drugs in the long term, it is logical to propose an evaluation of bone risk. Currently, there is no consensus on the management. Recommendations published in Britain recommended evaluating the one risk every two to five years, in long-term treated epileptics, with bone densitometry by dual-energy X-ray absorptiometry (DXA) and biology (calcium, phosphorus, alkaline phosphatase and vitamin D) [20].

The research group and information about osteoporosis (GRIO) recommends performing a dosage of 25 (OH) vitamin D before initiating supplementation to adjust the loading dose [21].

Pack recommends supplementation with vitamin D and calcium as well as control of the bone mineral density, particularly if there are other bone pathologies [22].

Good bone health practices include regular weight-bearing exercise, adequate sunlight exposure, adequate intake of calcium and avoidance of risk factors for osteoporosis such as smoking and alcohol use. High risk patients should be identified before start of AED treatment and evaluated.

High risk patients include institutionalized and non-ambulatory subjects, those with poor dietary habits and limited sun exposure such as at higher latitudes, those on multiple AEDs and with increased duration of AED use.

Those with multiple traditional osteoporosis risk factors as well as low calcium and vitamin D levels should also be considered at high risk and treated aggressively.

Prophylaxis with vitamin D has been recommended for all subjects using AEDs. Due to increased catabolism of vitamin D, higher than normally recommended doses (up to 4000 IU per day) of vitamin D may be required for optimal effect, particularly for those with low vitamin D levels, high risk of bone disease and/or with documented low BMD [11,21,22]. Since the current RDI of 400 IU of vitamin D is not considered sufficient even in healthy adults, a dose of 800–1000 IU/day of vitamin D is reasonable as a preventive therapy in subjects using AEDs [11].

For those with documented vitamin D deficiency, treatment with 50,000 IU/week for 8 weeks has been recommended and can be repeated if vitamin D levels remain low after initial treatment. This may be followed by supplementation with vitamin D 50,000 IU once a month to maintain the levels above the threshold of insufficiency [11].

The history of meningioma surgery, the fact that this rare fracture occurs in a man and a result a low-energy trauma explain because a tumor was initially suspected.

However, bone fragility associated with any underlying osteopenia (metabolic, endocrine, and hematologic) has not been ruled out.

Faced with the increased risk of osteonecrosis, bilateral arthroplasty rather than conservative treatment was decided.

The originality of our work is that it is the first observation that involves directly phenobarbital in the installation of osteoporosis; since our patient is epileptic but he is under barbiturates.

Conclusion

The long-term use of anti-epileptic sets out an increased risk of bone insufficiency.

Regular monitoring is essential and bone densitometry at the first sign must be practiced. The treatment of the osteopenia is so started as soon as possible to warn possible fracture.

Preventive measures to be taken are easily accessible; it is enough that the attending physician keeps this risk in mind.

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