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Osteoporosis Related Simultaneous Four Joints Fractures Associated with Seizure

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A case of steroid induced osteoporosis associated with multiple fractures and dislocations after a seizure is reported. The patient had two years history of steroid, given for aplastic anemia, no supplement or anti-resorptive therapy was given. The patient suffered simultaneous bilateral femoral neck fractures, bilateral shoulder surgical neck fracture dislocations and a Smith's fracture after one episode of seizure. Bone mineral density showed severe osteoporosis with T score of -2.9. There was a delay in the diagnosis which affected an otherwise good outcome in such situation. It is recommended that patients on steroid should be given calcium, vitamin D, and an anti-resorptive. Furthermore, a meticulous clinical examination is required in patients who are on steroids and suffer from epileptic seizures to rule out skeletal injury.

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Prolonged use of corticosteroids causes osteoporosis by both decreasing osteoblastic and increasing osteoclastic activity[1-3]. It is well established that long term use of glucocorticoid use increases risk of fractures[2-3.] Skeletal trauma after seizure is well-known.

We are reporting a case of simultaneous bilateral femoral neck fractures and bilateral humeral neck fractures after one episode of seizure. The patient had two years use of high dose steroid, for aplastic anemia, with no anti-resorptive therapy. Post injury investigation revealed osteoporosis. This is the first reported simultaneous four joints fractures, in a patient after a seizure attack.

The Case

A thirty-six year old Saudi male was brought to the emergency room following a generalized convulsions and loss of consciousness. He was driving a car and felt an acute sharp pain at right shoulder, few seconds later, he developed blurring of vision and he was able to stop the car and lost consciousness for 15-20 minutes. He was observed to have rolling up of the eyes with frothy salivations and trauma to the tongue. The patient complained of severe arthralgia and pain on minimal movement of the joints.

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On examination, he was noted to have petechiae over his body, no signs of meningeal irritation and there was a lesion over the scapula which was diagnosed as a snake bite and treated initially as such. Blood Investigations were normal and computerized tomography of the brain was normal. The patient was loaded with phenytoin 25 mg/Kg body weight.

He developed ecchymotic lesions on the skin and upper extremities, with recurrent vomiting, distended abdomen, paralytic ileus and deteriorating renal and liver function tests. In the intensive care unit, the patient was treated by antivenom and supportive therapy. At the end of the7^{th} day, the general condition improved and the patient started to complain of painful hips and shoulders. On further inquiry, the patient gave a history of using 15 mg prednisolone daily for aplastic anemia for two year. No supplement or anti-resorptive therapy was given to him.

Clinical examination showed both shoulder joints were dislocated and any attempt to move the hips and right wrist caused severe pain. Radiographs showed that bilateral anterior dislocation of the shoulders and bilateral humoral surgical neck fracture, Smith's fracture of the right radius and bilateral fracture of the neck of femurs (Garden IV, see Figures 1, 2).

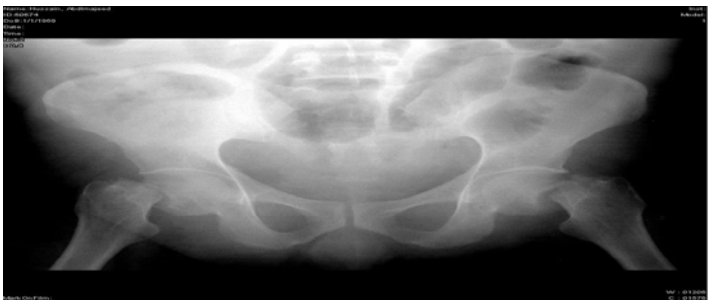

Figure 1: Radiograph of the Pelvis Showing Bilateral Fracture of Neck of Femurs

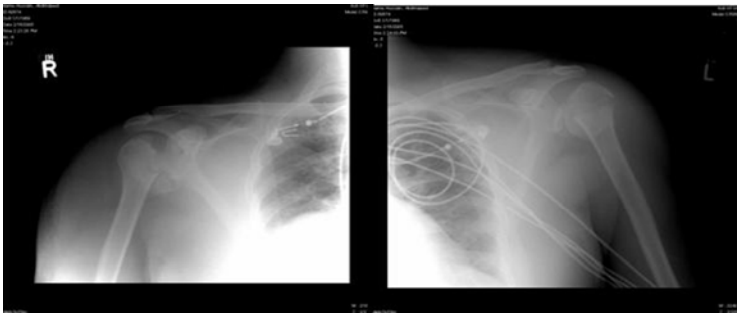

Figure 2: X-ray of Both Shoulders Revealing Dislocations

Under general anesthesia both shoulder joints were reduced, fracture necks of femur were fixed with two cannulated screws (Figure 3), Smith's fracture was reduced and plaster of paris was applied. Two weeks later, Bone Mineral Density of the Spine (Dual Energy X-ray Absorptiometry, DEXA scan) showed severe osteoporosis with T score of -2.9.

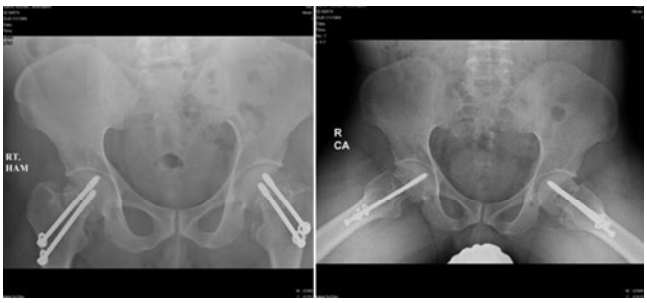

Figure 3: Post Fixation of Fracture Neck of Femurs

The last follow up appointment was four years after the incidence and it revealed no pain but rather painless limping. Examination showed right hip and left hip had limited range of movements and pain at extreme degree. The ranges of movements of the shoulder joints were normal.

Because of delayed diagnosis, the patient had bilateral avascular necrosis of head of humerus, see Figure 4.

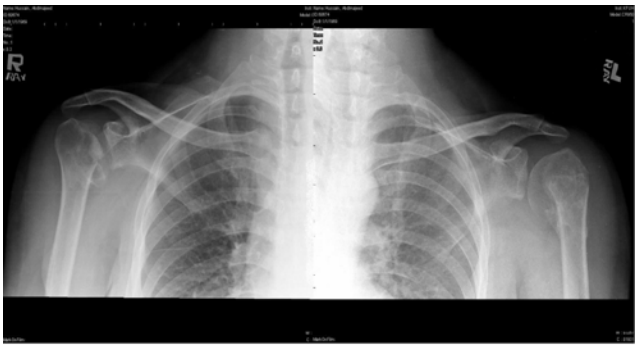

Figure 4: Radiograph Showing Bilateral Avascular Necrosis of Head of Humerus

DISCUSSION

Corticosteroids cause low bone mass by cellular apoptosis causing both decreased osteoblastic and increased osteoclastic activity[1]. It is well established that long term use of glucocorticoid use increases the risk of fractures[2-3]. De Vries reported that a daily dose of higher or equal to 15 milligrams have a substantial higher risk of fractures but Stienbuch et al believed that higher dose and longer duration is needed to increase the risk of fractures[4,5]. The guidelines for the prevention of Glucocorticoid induced osteoporosis have been proposed few years ago[6-9]. Devogelaer et al reported that supplemental calcium and vitamin D should be the first-line therapy in patients receiving ≥ 7.5 milligrams/daily[10]. Compston suggested that Bisphosphonates should be the treatment of choice, with supplementation of calcium and vitamin D[11]. Recent

studies suggest Teriparatide as a replacement of Alandronate[12]. Our patient was on 15 mg of prednisolone with no supplementation or anti-resorptive therapy.

Skeletal trauma due to generalized tonic and clonic seizures is not uncommon. Vertebral fractures, femoral neck, skull, shoulder and humeral head had been reported[13-15]. Joshy reported a case of bilateral femoral neck fractures due to an epileptic fit[16]. The patient we are reporting sustained bilateral femoral neck fractures, bilateral shoulder surgical neck fracture dislocations and a Smith's fracture. It has been reported that fractures are 2-6 times more common in epileptic patients than the general population[17-19]. This has been blamed on low bone mass which is caused by anti-epilepsy drugs, but the patient we are reporting sustained all the injuries during the first known attack of the epileptic fit and had steroid induced low bone mass[20].

Bilateral hip fractures are not uncommon and it occurs due to high energy trauma, early diagnosis reduces the incidence of permanent disability. Skeletal injuries due to post epileptic fit could be of serious consequences as the diagnosis is often delayed which causes long-term functional disability, which in our patient ended in bilateral avascular necrosis of head of humerus[21]. Delay in the diagnosis was thought to be due to the fact that patients remain in the intensive care unit, often unconscious and ventilated. Our patient was not ventilated but was complaining of severe arthralgia and difficulty in moving the limbs, which should have alerted the treating physicians.

CONCLUSION

This is a case of aplastic anemia, which was treated with long-term steroid. The case demonstrates that patients treated with long term steroids, need to be supplemented with anti-resorptive, otherwise an episode of epileptic fit could lead to major skeletal trauma, multiple fractures could not be excluded. Clinicians should be aware of the complication of such treatment.

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