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Late-diagnosed bilateral intertrochanteric femur fracture during an epileptic seizure

Epilepsi nöbeti sırasında gelişmiş geç tanı konmuş, iki taraflı intertrokanterik femur kırığı

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Although spontaneous and simultaneous bilateral hip fractures without trauma are seen rarely, epileptic seizures may lead to these fractures. We present an 82-year-old female patient with poor bone quality and a 20-year history of epilepsy. She had been using anticonvulsant drugs for almost 20 years. Following a convulsive epileptic attack, bilateral intertrochanteric femur fractures occurred (causing bilateral hip pain), which was diagnosed on the 12th day. An earlier pelvic anteroposterior roentgenogram would be helpful for early diagnosis. It should not be forgotten that bone fractures may be observed without trauma in epilepsy patients.

Key Words: Epileptic seizure; hip fracture/bilateral.

Travma olmadan iki taraflı kalça kırıkları nadirdir, fakat epileptik nöbetler bu tip kırıklara neden olabilir. Bu olgu sunumunda, 82 yaşında, kemik kalitesi kötü olan ve 20 yıllık epilepsi hastası olduğu bilinen bir kadın olgu sunuldu. Hasta yaklaşık 20 yıldır antikonvülzan ilaçlar kullanmaktaydı; bir epilepsi atağı sırasında her iki kalçasında intertrokanterik femur kırığı oluştuğu, fakat tanının 12. gün sonra konulabildiği öğrenildi. Daha erken çekilen bir pelvis ön-arka grafisi erken tanı için yardımcı olabilirdi. Epilepsi hastalarında travma olmaksızın kırıklar olabileceği akılda tutulmalıdır.

Anahtar Sözcükler: Epileptik nöbet; kalça kırığı/iki taraflı.

Although hip fractures are frequent in the elderly population, simultaneous and spontaneous (atraumatic) bilateral hip fractures are very rare. Fractures and dislocations of major joints are usually caused by severe external trauma,[1] or such cases may occur secondary to several metabolic disorders.[2] Seizures may cause significant muscular tension capable of fracturing bones.[3] Sudden forceful tonic muscular contractions of seizure activity are a lesser known cause of fractures and dislocations. Seizures caused by a wide variety of other disorders have been reported to cause skeletal lesions, predominantly fractures of the vertebrae and fractures and dislocations in the regions of the shoulder and hip.[1]

We present the case of an 82-year-old female epileptic patient with bilateral intertrochanteric femur fractures. She had been observed and under medical treatment for epilepsy for almost 20 years. Following her last epileptic convulsive attack, bilateral intertrochanteric femur fractures were not diagnosed until the 12th day, although she was taken to the emergency room several times. Why so late?

CASE REPORT

We present an 82-year-old epileptic female patient with bilateral intertrochanteric femur fractures. She had been referred to our clinic from another hospital due to her fractures and concomitant cardiac problems. She suffered from cardiac arrhythmias and had a 20-year history of epilepsy. She had been under medical treatment for epilepsy, although intermittently. During the last five years, she was able to move indoors with crutches, despite great difficulty.

On the day of the event, her sleep was interrupted by severe bilateral hip pain and general muscle pain in the morning, which she reportedly experienced once

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or twice a year due to a tonic-clonic seizure. She experienced difficulty moving her legs and suffered from generalized muscle pain, especially localized around both buttocks and knees. The seizure was followed by a postictal state that resolved spontaneously, and was reported to have lasted for approximately 1 minute. The patient was in her family's company during the entire seizure, and the family reported no fall from the couch or experience of any trauma.

Following the seizure, the patient, who lives with her daughter in a village, informed her daughter of her aches. Guided by her mother's previous experience with a convulsive attack that was followed by aches, the daughter offered analgesics and muscle relaxants to ease the pain. As the physical immobility and pain (the patient could not rise from the bed) continued for two days, she was taken to the nearest town hospital. Bilateral knee plain roentgenograms were taken for the lower extremity pain. As no emergent osseous pathology could be obtained, bed rest, analgesics and muscle relaxants were prescribed for general muscle aches. She used the prescribed medication for 10 days; however, no recovery was observed. As the pain had become unbearable, she was taken to the nearest city hospital. She reported pain in her entire lower extremity, which began after a convulsive attack, and that she had not been able to walk since the seizure. After physical examination, the doctor evaluated her pelvis anterior-posterior (AP) roentgenogram, which led to the diagnosis of bilateral intertrochanteric femur fractures 12 days after the generalized tonic-clonic seizure (Fig. 1a). Due to the risks related to anesthesia, she was not operated in the city hospital and was referred to our hospital, which has an intensive care unit if needed.

Carried in a litter to our emergency room, she was known to be epileptic for almost 20 years. Her seizures consisted of daily early morning myoclonus and occasional generalized tonic-clonic seizures. She was prescribed a twice-daily dose of phenytoin 100 mg; however, she was not compliant with the prescription. In her physical examination, both legs were in external rotation, she was unable to move her legs because of pain, and hip range of movements could not be examined. No neurovascular deficiency could be determined in the lower extremities.

Laboratory findings revealed the following: hemoglobin concentration 11.9 g/dl (normal: 12.2-17.2 g/dl), urea level 233 mg/dl (normal: 10-50 mg/dl), creatinine level 4.02 mg/dl (normal: 0.44-1.03 mg/dl), total protein level 5.6 g/dl (normal: 6.4-8.3 g/dl), albumin level 2.4 g/dl (normal: 3.5-4.8 g/dl), lactate dehydrogenase activity 541 U/L (normal: 98-192 U/L), alkaline phosphatase activity 218 U/L (normal: 32-91 U/L), creatinine kinase activity 1708 U/L (normal: 38-204 U/L), serum calcium level 3.9 mg/dl (normal: 8.9-10.3 mg/dl), intact parathormone level 757.5 pg/ml (normal: 12-88 pg/ml), homocysteine level 31.7 uMol/L (normal: 5-15 uMol/L), and serum phenytoin level 0.9 ug/ml (normal: 10-20 ug/ml).

Under general anesthesia, on the 14th day of the trauma, she was operated bilaterally in one session. First, for the right hip, bipolar hemiarthroplasty with cementation was applied in the lateral decubitus position, with lateral incision (by using the modified Hardinge approach). Then, the exact procedure was repeated for the left hip (Fig. 1b). After the operation, she was monitored in the intensive care unit for the first 24 hours. On the 2nd day of operation, she was mobilized and was able to walk with crutches. After consultation to the Neurology Department, her epilepsy treatment was re-organized. For hypocalcemia, a medical treatment was arranged following consultation to the Endocrinology Department. On the 5th day after the operation, she was discharged from the hospital.

DISCUSSION

Simultaneous bilateral hip injuries, including bilateral intertrochanteric femur fractures, are seen rarely. Most occur as a result of epileptic seizures, are electrically induced, or have hypocalcemic or uremic ori-

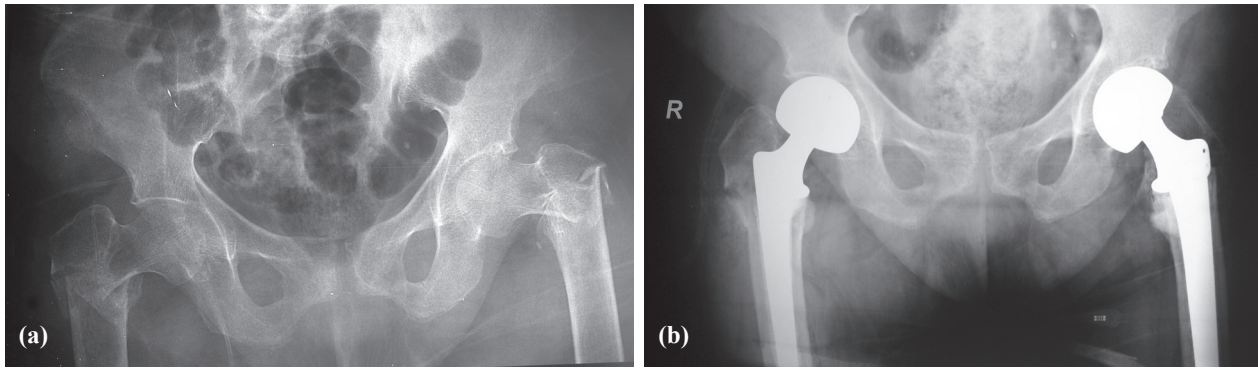

Fig. 1. **(a)** Preoperative roentgenogram. **(b)** Postoperative roentgenogram.

gin.[5] Several factors may contribute to the increased fracture risk in seizure patients. Muscular contraction generated by seizures may directly fracture bone; however, indirect mechanisms may also elevate fracture risk. Several reports emphasize bone disease as a major precipitating factor, and there is an increased incidence of fracture in chronic epilepsy.[1]

Antiepileptic medications may affect intestinal calcium absorption and can induce anticonvulsant osteopathy.[3] When used for long periods, anticonvulsant drugs cause osteomalacia. Anticonvulsant drugs block mineralization of the bone matrix, decrease peripheral response to the active vitamin D, help to degrade vitamin D by inducing hepatic enzymes, and decrease calcium intake from the gastrointestinal tract. For these reasons, anticonvulsant drug usage may cause osteomalacia.[2]

Postmenopausal osteoporosis and immobilization for long durations are other causes of osteomalacia. Active mobilization results in rapid return of blood supply to both the bone and soft tissues and improves articular cartilage nutrition. Further, when combined with weight-bearing, active mobilization greatly decreases post-traumatic osteoporosis and enhances bone formation.[4] Our case concerns an 82-year-old osteoporotic female who had been on anticonvulsant drugs for almost 20 years.

Thirty to 35% of seizure patients have experienced a secondary injury as a result of seizure during their lifetime. These observations support the importance of the evaluation of secondary injury in patients presenting to the emergency department. Fractures are less common complications in seizure patients who experience seizure; however, they have been reported to occur in 0.25% to 2.4% of this group of patients. Patients with epilepsy are 33% more likely to sustain a fracture in their lifetime than those without epilepsy.[3] The proximal humerus was the most common site of fractures in the atraumatic group.[3,6]

Some seizure-induced fractures, such as compression fracture of the vertebrae and fractures of the humerus or the head or neck of the femur, resemble more common fractures caused by external trauma. If a patient is not known to have epilepsy or if the seizure was not witnessed, the unexpected finding of such a fracture may lead to a suspicion of assault, particularly if the patient is not in a condition to give a clinical history.[1] There are some reported cases of acute fractures of the acetabulum secondary to a convulsive seizure. Seizures could also lead to acute periprosthetic fractures of the acetabulum in patients with osteopenia after total hip arthroplasty.[7]

Seizure-related fractures, which most frequently involve the head and neck of the femur and the proximal humerus, may sometimes present diagnostic difficulties, but usually are evident due to pain or deformity and the history of seizure.[1] Physicians should be alert to the possibility of fractures in patients with epileptic seizures. Pain in any part of the body should signal the need for immediate radiographic examination.[5] To avoid unrecognized injuries in postconvulsive patients, a thorough evaluation must be performed prior to dismissal of the injury as a ligament sprain or muscle strain.[5] Certain reports indicate that recognition of the injury may be delayed or found incidentally on imaging for unrelated reasons.[1] Bilateral simultaneous trochanteric fractures are rare and potentially life-threatening injuries, associated with high morbidity. They should be diagnosed and treated as soon as possible.

In conclusion, evaluation of extremity pain, deformity, ecchymosis, and crepitus should help in the identification of bony injury following a seizure and should always be tracked by radiographs of the affected area. Likewise, an A-P radiograph of the pelvis should be obtained for any seizure patient suffering from hip or groin pain.

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