

Manifest Osteoporosis and Bilateral Shoulder Avascular Necrosis Coexistence Due to the Long Term Glucocorticoid Use: A Case Report

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Abstract: Glucocorticoids have many side effects. Osteoporosis is relatively common due to long-term glucocorticoid use, but humerus avascular necrosis is a less encountered condition. Present case is a female patient with a diagnosis of bronchial asthma who is receiving medical treatment consist of high dose corticosteroids for nearly 10 years. She was considered as steroid-induced osteoporosis, and aseptic necrosis of bilateral humeral head.

Keywords: Glucocorticoids, osteoporosis, avascular necrosis.

INTRODUCTION

Glucocorticoids acts as immunosuppressive and antiinflammatory drugs in appropriate therapeutic doses. Glucocorticoids may cause many side effects [1]. Osteoporosis is a commonly seen side effect of corticosteroids on bone metabolism, however avascular necrosis is a rare side effect. Steroids directly inhibit bone formation and cause a decrease in the number and activity of osteoblasts [2, 3].

Osteonecrosis is defined as a death of the bone marrow and bone elements due to recurring or complete cessation of blood supply to the bone. Osteonecrosis may be idiopathic or secondary, post-traumatic or atraumatic. Although osteonecrosis may be seen in any joint, but wrist joints, femoral head, femoral condyle and humeral head are the most affected sites. Colletaral circulation at the femoral head is usually limited. Impaired blood supply results in necrosis of the epiphysis. In large groups of patients with atraumatic osteonecrosis of the hip, rate of alcohol and/ or glucocorticoid use is 60-90%. Risk of glucocorticoids depends on the dose and the recruitment period. It is considered that total dose of 2000- 4000 mg of prednisone is the threshold for the development of osteonecrosis. However, after the low-dose glucocorticoid use osteonecrosis of the femoral head have been reported [4-6].

CASE REPORT

61-year-old female patient with a diagnosis of bronchial asthma was admitted to the orthopedic clinic with complaints of pain and difficulty in lifting her arms on the right and left shoulders. She had bronchial asthma for 10 years and have 5-6 times asthma attacks per year. She was treated with theophylline, antihistamines and different doses of corticosteroids. The patient's history emerged increasing shoulder pain and weakness for at least 2 years. These findings of patient was considered as malignancy, secondary osteoporosis (steroid-induced), right and left shoulder avascular necrosis in preliminary diagnosis.

The patient's plain films were revealed lumbar compression fractures. Bone mineral density measurement of L1-L4 spines were as follows; T score: -2.64, Z-score: -3.20. PTH, ALP, SGOT, SGPT, sedimentation, CRP was detected in the normal ranges. Other laboratory findings were as follows; 25(OH) vitamin D: 13 mcg, serum Ca⁺⁺: 9.1 mg/ dL, cortisol: 4.32mg/ dL. Tumor markers for malignancy screening were negative. In blood count; WBC: 11600 with neutrophil predominance (76.6%) and lymphopenia (11.2%), eosinopenia (1.0%). This blood table was in accordance with corticosteroid use. Atelectasis changes were detected on chest radiograph. Abdominal ultrasonography was normal. Direct radiography revealed previous fractures and possible avascular necrosis (Fig. 1). Magnetic Resonance Imaging (MRI) was interpreted as avascular necrosis (Fig. 2, 3).

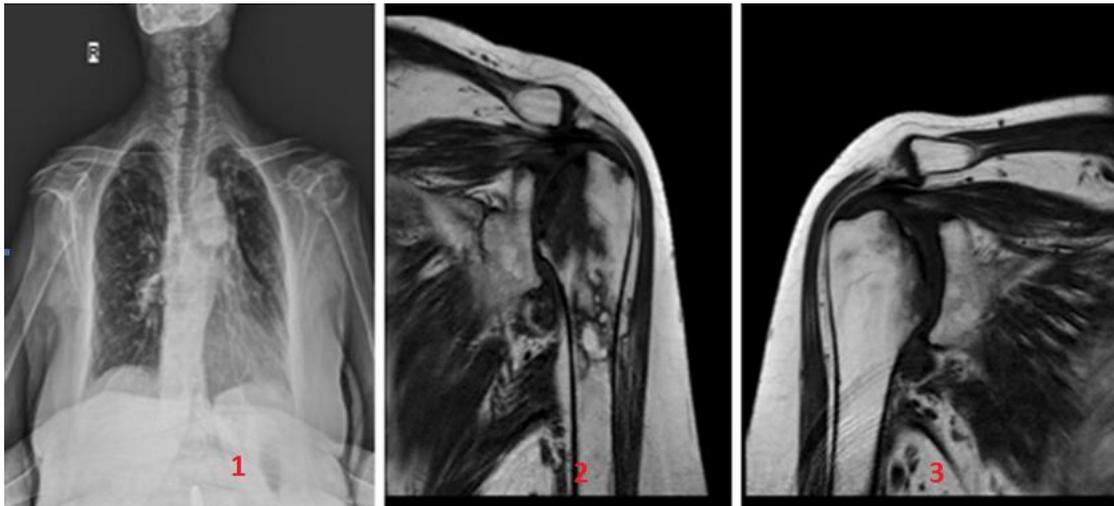


Fig. 1: Direct chest radiogram of the patient
Fig. 2& 3: Bilateral humeral avascular necrosis on MRI

According to physical examination, laboratory and radiological findings; she was diagnosed as manifest osteoporosis and bilateral humerus avascular necrosis due to irregular and uncontrolled use of steroids. Treatment schedule performed for the patient was as follows; alendronate 70 mg once a week, active vitamin D: 0.5mcg/ day and elemental calcium: 1g/ day. Prosthesis to both shoulders were planned by orthopedicians.

DISCUSSION

Osteoporosis is a disease of elder people, especially postmenopausal women. In general, osteoporosis is caused by secondary reasons in 30-60% rates, and this rate may increase to 64 % in male population [7, 8]. The secondary causes of osteoporosis in men are alcoholism, glucocorticoid use and hypogonadism. Glucocorticoids leads to rapid bone loss even during early treatment periods; treatment time shorter than even 3 months, bone mineral density may reduce 10-20%. Low dose (<7.5 mg / day) or alternate day glucocorticoid therapy has been identified to cause clinically significant bone loss [9, 10]. Hansen M *et al.* determined reduction in bone mineral density in rheumatoid arthritis patients with corticosteroid dose of 6 mg daily with 2160 mg cumulative dose [11]. Although osteoporosis is a common side effect of corticosteroids on bone metabolism, however avascular necrosis is rare. Humeral head osteonecrosis is less frequent and difficult to diagnose because of its silent clinical survey, in comparison with femoral head osteonecrosis as we mentioned in the present case. Especially in elderly patients, differential diagnosis of osteoporosis, osteoarthritis and rheumatic diseases should be made thoroughly.

In comparison with femoral head osteonecrosis, the symptoms and loss of function caused by necrosis of the humeral head is relatively less

severe. No doubt that, this situation is a result of exposure to less load of arms than lower extremities. Another consequence of less load exposure lead a slower progression of pathology on the shoulders [12,13].

CONCLUSION

This case showed us that the irregular use of corticosteroids without medical supervision is not safe. Especially corticosteroid therapy in patients with diseases that affect the immune system should be more careful and necessitates regular monitoring. The lowest dose and short- period treatment should be executed, if possible. Side effects of corticosteroids on bone metabolism should always be considered and prophylactic calcium and vitamin D supplementation should be provided; and patients should be taken into strengthening exercise program for dorsal region muscles. Regular physical examination and tests for bone metabolism should be made in patients using corticosteroids longer than six months. In patients with steroid- induced osteonecrosis, extremity function loss is relatively more common due to their systemic disorders, corticosteroid use and immobility.

REFERENCES

1. Fardet L, Kassar A, Cabane J, Flahault A; Corticosteroid-induced adverse events in adults: frequency, screening and prevention. *Drug Saf.*, 2007; 30(10): 861-881.
2. Canalis E, Mazziotti G, Guistina A, Bilezikian JP; Glucocorticoid-induced osteoporosis: pathophysiology and therapy. *Osteoporosis Int.* 2007; 18(10): 1319-1328.
3. Deogelaer JP; Glucocorticoid-induced osteoporosis: mechanisms and therapeutic approach. *Rheum Dis Clin North Am.*, 2006; 32(4): 733-57.
4. McKee MD, Waddell JP, Kudo PA, Schemitsch EH, Richards RR; Osteonecrosis

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- of the femoral head in men following short-course corticosteroid therapy: a report of 15 cases. *CMAJ*, 2001; 164(2): 205-206.
5. Bekler H, Uygur AM, Gökçe A, Beyzadeoğlu T; Steroid kullanımının femur başı avasküler nekrozu patogenezindeki yeri: Deneysel hayvan modeli. *Acta Orthop Traumatol Turc.*, 2007; 41(1):58-63.
 6. Yalçın P. Glukokortikoid osteoporozu. *Romatizma*, 2000; 15(2):145-150.
 7. De Vries F, Bracke M, Leufkens HG, Lammers JW, Cooper C, Van Staa TP; Fracture risk with intermittent high-dose oral glucocorticoid therapy. *Arthritis Rheum.*, 2007; 56(1): 2008-2014.
 8. Woolf AD; An update on glucocorticoid induced osteoporosis. *Curr Opin Rheumatol.*, 2007; 19(4): 370-375.
 9. Shah SK, Gecys GT; Prednisone-induced osteoporosis: an overlooked an undertreated adverse Effect. *J Am Osteopath Assoc.*, 2006; 106(11): 653-657.
 10. Karkoulias K, Charokopos N, Kaparianos A, Sampsonas F, Tsiamita M, Spiropoulos K; Aseptic femoral head necrosis in a patient receiving long term courses of inhaled and intranasal corticosteroids. *Tuberk Toraks*, 2007; 55(2): 182-185.
 11. Hansena M, Pødenphantb J, Florescuc A, Stoltenberga M, Borchb A, Kluger E *et al.*; A randomised trial of differentiated prednisolone treatment in active rheumatoid arthritis. Clinical benefits and skeletal side effects. *Ann Rheum Dis.*, 1999; 58(11): 713-718.
 12. David HG, Bridgman SA, Davies SC, Hine AL, Emery RJ; The shoulder in sickle-cell disease. *J Bone Joint Surg Br.*, 1993;75(4): 538-545.
 13. Milner PF, Kraus AP, Sebes JI, Sleeper LA, Dukes KA, Embury SH *et al.*; Osteonecrosis of the humeral head in sickle cell disease. *Clin Orthop Relat Res.*, 1993;289: 136-143.