

**Case Report:****SCAPHOID OSTEONECROSIS: AN UNUSUAL SITE FOR OSTEONECROSIS**Günşah Şahin<sup>1</sup>, Özlem Bölgen Çimen<sup>1</sup>, Selda Bağış<sup>1</sup>, Hayal Güler<sup>1</sup>**SUMMARY**

Osteonecrosis is defined as the death of bone from repeated interruptions or a single massive interruption of the blood supply to the bone. In this article, we presented a 60 year old woman diagnosed as having polymyalgia rheumatica who developed scaphoid osteonecrosis because of taking corticosteroid for long period. The patient was reported because of unusual localisation for osteonecrotic bone involvement.

**Key words:** Osteonecrosis, corticosteroids

**ÖZET****SKAFOİD OSTEONEKROZ: OSTEONEKROZ İÇİN ATİPİK LOKALİZASYON**

Osteonekroz, kemiğe olan kan akımı temininin kesilmesinden dolayı kemiğin ölümü olarak tanımlanır. Bu makalede, polimiyaljiya-romatika tanısı almış ve uzun süreli kortizon kullanımına bağlı skafoid osteonekroz gelişmiş, 60 yaşında bir kadın hasta sunulmuştur. Osteonekroza bağlı kemik tutulumu açısından atipik lokalizasyonlu olduğu için yayınlanması uygun görülmüştür.

**Anahtar sözcükler:** Osteonekroz, kortikosteroidler.

**INTRODUCTION**

Osteonecrosis of bone is defined as death of osseous cellular components as a result of vascular compromise. Causes of osteonecrosis include trauma, alcoholism, pregnancy, obesity, hemoglobinopathies, dysbaric disorders, idiopathic osteonecrosis and corticosteroid administration.(1) Possible complications of osteonecrosis are secondary degenerative joint disease and cystic or sarcomatous transformation.(1) A predilection for the ends of the long bones such as femoral head and humeral head has been identified. After the femoral head, the most frequently affected site is humeral head (2).

We presented a female patient with scaphoid osteonecrosis on her right wrist, as a result of long term corticosteroid administration and as its atypic localisation for bone involvement we decided to report.

**CASE REPORT**

A 60-year old woman was admitted to the outpatient clinic with a complaint of right hand pain(dorsal) which lasted for one year. Three years ago she was diagnosed as having polymyalgia-rheumatica and administered corticosteroid(prednisolone) for three years. She was treated with 30 mg/day(single dose,p.o) tapering to 5mg/day gradually. She did not received any other drug. Six months, prior to assessment, she discontinued the drug because of side effects.

She had no history of trauma, alcohol abuse, cigarette smoking, drug abuse, irradiation and any systemic disease except polymyalgia –rheumatica.

On examination she was an obese woman(Body mass index=33.3). All vital signs were normal. She had tenderness on her right wrist with palpation. Results of examination of the locomotor

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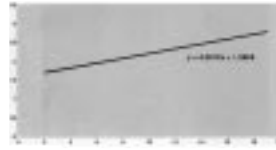
system, respiratory and the cardio-vascular system were unremarkable. Laboratory investigations revealed that white blood cell count was 11.600/mm<sup>3</sup>, hemoglobine was 13.3 gr/ dl, erythrocyte sedimentation rate (Westergren) was 36mm/h, C-reactive protein was 6.4mg/l and rheumatoid factor was <9.9IU/ml. Blood chemistry and urine analysis were normal. Chest radiograph was normal.

On plain film, there was irregular, sclerotic, round lesion at the proximal end of scaphoid bone (Figure 1). On tomography, medial region of scaphoid bone was sclerotic (radiodense) and it was consistent with a diagnosis of aseptic necrosis (Figure 2). On MRI, T1 and T2 weighted coronal and axial images displayed the characteristic of the lesion with loss of signal (hypointense) with diagnosis of osteonecrosis (Figure 3).

It was accepted that she has been in remission for polymyalgia-rheumatica and she was treated with non-steroidal-antiinflammatory drug (diflunisal- 1000mg daily ) for her pain. She was consulted with orthopedic surgeons but she refused operation. So she has been on follow-up for any complication.



**Figure 1:** On plain radiograph; irregular, sclerotic, round lesion at the proximal of the scaphoid.



**Figure 2:** On tomography; medial site of the proximal scaphoid is sclerotic (dense) and consistent with osteonecrosis.



**Figure 3:** On MRI ; T1, T2, weighted coronal and axial images display loss of signal that is consistent with osteonecrosis.

## DISCUSSION

The term osteonecrosis indicate the occurrence of ischemic death of the cellular constituents of bone and marrow. The necrotic bone is not only aseptic but also avascular. Causes of osteonecrosis include, trauma, alcoholism, use of corticosteroid, hemoglobinopathies, pregnancy, obesity, Gaucher's disease and dysbaric conditions(1). Corticosteroid administration is the most common aetiologic factor in patient with osteonecrosis who has not been exposed to trauma. Jones reported that corticostero-

id and alcoholism account for about 60 percent of all cases of non traumatic osteonecrosis. The development of osteonecrosis depends both on the dose and duration of corticosteroid exposure.(1,2) Analysis suggested that, for each 20 mg/day increase of prednisone, the risk of osteonecrosis increased 5 percent(1). The mechanism of corticosteroid osteonecrosis includes intravascular coagulation, intraosseous hypertension, microvascular destruction and direct cytotoxic effects on osteocytes. Corticosteroid induced alteration of lipid metabolism may cause fat embolism, intravascular coagulation or both. Fat embolism has been shown to produce fibrin thrombus and microvascular hemorrhage.It also affects the structure of vessels (3,4,5,6).

Obesity and advanced age are also independent risk factors for osteonecrosis as they cause decreased fibrinolytic activity(1). Although corticosteroid is the cornerstone of the treatment of Polymyalgia Rheumatica, we suggested that long term use of corticosteroid led to osteonecrosis of scaphoid bone in this patient and it was supposed that obesity with advanced age might facilitate it.

Certain laboratory tests may be useful for subjects suspected of having an associated condition. In our patient, there were no remarkable laboratory findings for osteonecrosis.

In early osteonecrosis, the radiographic examination is normal. Nuclear scintigraphy may show decreased uptake. Magnetic resonance imaging(MRI) is recognized as the most valuable imaging technique in the diagnosis and staging of osteonecrosis, surpassing scintigraphy in both specificity and sensitivity(1,2,4). Although the appearance was consistent with osteonecrosis on plain film, MRI played an essential role for differential diagnosis in our patient as the most sensitive means of detection of osteonecrosis.

In the treatment, there are various surgical procedures for scaphoid osteonecrosis in the literature such as performing bone graft, replacement arthroplasty and screw fixation(7,8,9,10,11). As the patient, refused surgical procedure, we recommended her taking nonsteroidal antiinflammatory drug for her pain and in order to observe for any complication developing in the future, such as, multifocal osteonecrosis, she has been on follow- up.

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