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A case of bilateral hip and knee osteonecrosis in a patient with ankylosing spondylitis who used steroids due to immune thrombocytopenic purpura

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Abstract

We aim to present a case of bilateral hip and knee osteonecrosis due to steroid use in an ankylosing spondylitis (AS) patient who developed immune thrombocytopenic purpura (ITP) after the use of nonsteroidal anti-inflammatory drugs (NSAID). A 60-year-old male patient was diagnosed with AS with bilateral sacroiliitis and inflammatory low back pain on sacroiliac radiography 10 years ago. He was followed up with NSAIDs. Intravenous steroid treatment was started in the hematology clinic to the patient who had developed ITP due to NSAID usage. The patient presented to our clinic with bilateral hip pain that developed after steroid therapy. He had limited hip joint movement and bilateral destructive hip joints in pelvic radiography. Bilateral total hip arthroplasty (THA) was performed by the Orthopedics and Traumatology Department at 1-year intervals because of osteonecrosis associated with steroid use. When he presented to our clinic 3 years later with bilateral knee pain, we detected osteonecrosis in the distal segments of bilateral femurs and proximal tibial diaphysis on knee MRI. We implemented a rehabilitation program with the conservative treatment recommendation of orthopedics. Comorbidities or drug side effects should be examined more carefully while treating peripheral joint involvement in patients with AS.

Keywords: Osteonecrosis, Meloxicam, Immune thrombocytopenic purpura, Ankylosing spondylitis

Introduction

Osteonecrosis (ON) is a bone disorder that results with the demolition of the bone and bone marrow cells [1]. Immune Thrombocytopenic Purpura (ITP) is a hematological disease associated with autoantibody activity against glycoprotein structures on platelets due to primary or secondary causes [2]. We present a case with bilateral hip and knee osteonecrosis due to steroid use of patients with ankylosing spondylitis (AS) who developed ITP after nonsteroidal anti-inflammatory drug (NSAID) use. The patient was 60 years old. After the first diagnosis of AS, he presented with bilateral hip ON first, then bilateral knee ON over the next 4 years. Orthopedics was consulted to learn whether if surgery was an option, but conservative treatment was recommended. This is the first case report to present ON in the lower extremities in 4 different regions in the patient who has AS.

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Case presentation

A 60-year-old male patient was admitted to our clinic with inflammatory back pain 8 years ago. He also had morning stiffness which lasted more than 30 minutes due to thoracolumbar limitation and bilateral heel pain. He had no disease other than insulin-dependent diabetes mellitus for the last 13 years, hyperlipidemia for the last 12 years and he was operated on for meniscopathy. There were bilateral sacroiliitis in sacroiliac magnetic resonance imaging (MRI), syndesmophytes in the lateral thoracic radiography (Figure 1), and bilateral enthesitis on the lateral foot x-ray (Figure 2). He was HLA B27 negative and had a CRP value of <0.5mg/dl. Other laboratory values were also within normal ranges.

Figure 1: Lateral thoracic radiography

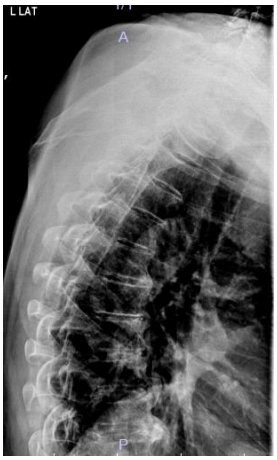
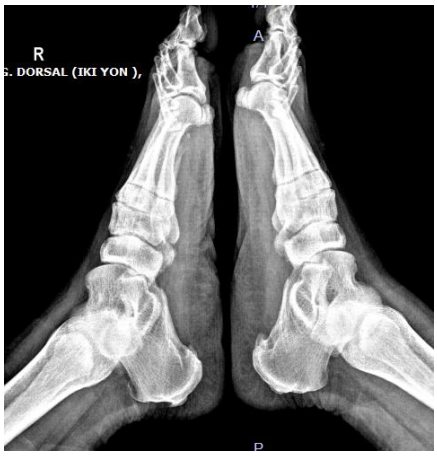
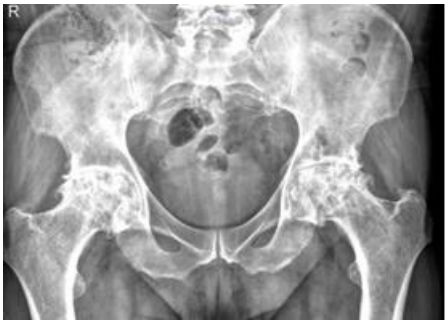


Figure 2: Lateral view of foot in x-ray, enthesitis is observed on bilateral calcaneal areas



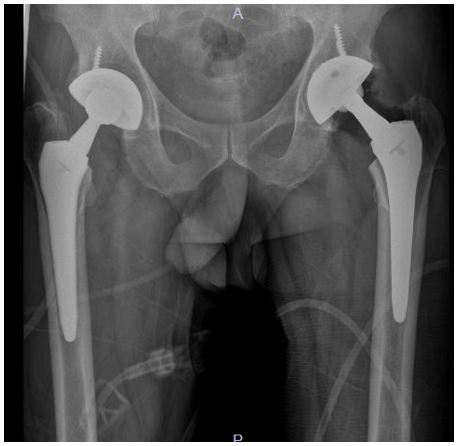
The patient was diagnosed with AS. The initial treatment was meloxicam at a daily dose of 15 mg. The patient was invited for follow-up 14 days later, but he missed the appointment. During this time, he went to another health institution and was diagnosed with thrombocytopenia. He received intravenous (IV) methylprednisolone at 10 mg/kg/dose for 3 days. Steroid treatment was tapered and terminated within 4 weeks. One year later, after the ITP treatment finished, the patient presented to the clinic with bilateral hip pain. On physical examination, limitation of movement was observed within the hip joint. Pelvic radiographs (Figure 3) showing bilateral destruction of the hip joints prompted us to consult with the orthopedics and traumatology clinic regarding surgical treatment for avascular necrosis.

Figure 3: Pelvic radiography, destructive hip appearance due to the avascular necrosis



Bilateral total hip arthroplasty (THA) had been performed 1 year apart (Figure 4).

Figure 4: Pelvic radiography, Bilateral total hip replacement, postoperative view.



During the follow-up of AS and bilateral THA, the patient presented to our clinic with more severe bilateral knee pain in the left knee after 4 years. Bilateral knee radiography showed lesions in the distal femur and proximal tibia (Figure 5). The bone infarct areas of 2x1 cm in the femur and 2.5x1.5 cm in the tibia were detected in the left knee MRI (Figure 6).

The patient with AS who developed ITP after NSAID usage had ON in four joints due to the use of high-dose steroids for ITP treatment. He is currently under treatment in our clinic.

Figure 5: A two-way bilateral knee X-ray of the distal femur and proximal tibia on the border of irregular sclerotic lesions.

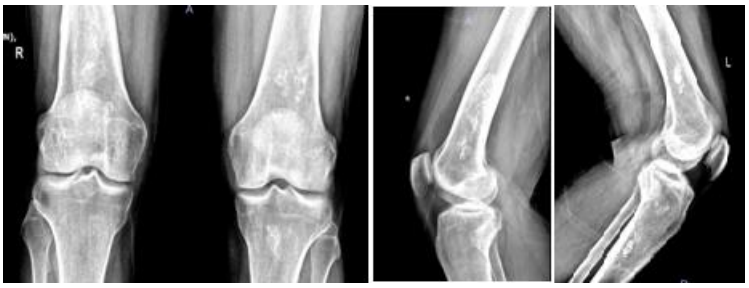
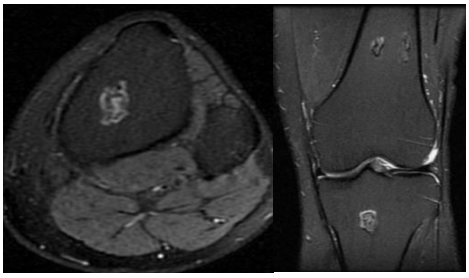


Figure 6: MRI of the left knee, two lesions of 2x1 cm in the femur and one lesion of 2.5x1.5 cm in the tibia.



Discussion

Osteonecrosis (ON, avascular necrosis) is caused by ischemic injury of bone and bone marrow cellular components. It is more common in the hip joint (90%) but can be detected in the knee joint with a lower incidence (10%) [1]. Steroid use is the most common reason for ON. Many osteonecrosis cases related to steroid use have been reported in the literature. Even though there are many publications, there is no case of avascular necrosis observed in 4 joints in a patient with AS.

The side effects of steroids depend on the dose and time. ON can usually be detected by imaging 1-6 months after high-dose corticosteroid (>0.5 mg/kg/day) treatment [3]. However, some publications report osteonecrosis cases after low-dose steroid intake [4]. In 1996, Usui et al. [5] reported that 4 women who received steroids, one for ITP and 3 for lupus, were diagnosed with femoral head fractures due to femoral head osteonecrosis. In in a case report published by Yildiz et al. [6] in 2008, a 64-year-old female patient reportedly received pulse steroid therapy for 3 weeks, starting at 1000 mg/day, reducing the dose every 3 days. Two weeks after starting the treatment, there was unilateral pain in his left knee and hip, and after 4 weeks he was unable to walk without support. A diagnosis of ON was made with unilateral right knee and hip MRI. After rehabilitation, hip and knee arthroplasty was recommended. In 2015, a 17-year-old patient diagnosed with ITP received pulse steroid therapy for 4 days, at a dose of 40 mg/day every 4 months. After 2 years of treatment, he had severe hip pain during heavy lifting. MRI showed ON in the right femoral head at the second year of treatment and in the left femoral head at the third year. The patient's age was appropriate for core decompression therapy rather than bilateral total hip replacement surgery [7].

ITP is a hematologic disease caused by autoantibodies that target glycoprotein structures on the platelets for primary or secondary reasons. Secondary reasons include autoimmune diseases (lupus, antiphospholipid syndromes), infections (HBV, HCV, HIV, CMV), and medical treatments (NSAIDs, heparin, quinidine). Secondary thrombocytopenia results from immune-mediated platelet destruction associated with medical therapy. The reduction in the platelet count occurs within a few hours to 1-2 weeks after drug use [2]. The most common side effects of meloxicam in the NSAID group are gastrointestinal complaints such as diarrhea and dyspepsia, neurological symptoms such as dizziness and headache, and dermatological complaints such as rash. Thrombocytopenia is a rare side effect that occurs in less than %2 of the patients [8]. The first case of meloxicam-induced thrombocytopenia was verified in India. According to the case report, a 57-year-old woman who took 75 mg/day meloxicam for arthralgia had ITP symptoms in 24 hours after taking two doses. The patient’s platelet count reached basal levels during a year of observation. In another case, reported by Ranieri MM in Philadelphia in 2014 [9], the platelet count of the patient was 2000/mm^3 with the clinical symptoms of ecchymosis and gastrointestinal bleeding after one week of meloxicam use. According to the Hill criteria for causality, the patient was diagnosed with immune thrombocytopenia secondary to meloxicam.

There were no data on the development of ON after steroid treatment in both above-mentioned cases. In our case, ITP developed after the use of NSAIDs, as in the previous 2 cases, and also, ON occurred in the late period after steroid use for ITP treatment. In the literature, there are no cases of ONs in 4 joints due to steroid intake. In this case report, while treating AS, we detected ITP, which is a rare side effect of meloxicam. We also found ON which developed in 4 joints in the late period of steroid treatment after ITP.

Comorbidities or drug side effects should be examined more carefully while treating peripheral joint involvement in patients with AS.

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