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Case Report

# Osteonecrosis of the Knee: The Unintended Consequence of Steroid Abuse

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The anti-inflammatory and immunosuppressive properties of steroids allow their use in a wide variety of rheumatological diseases, asthma, inflammatory bowel disease, cancer therapy, and severe viral infections. Though life-saving or organ-saving, long-term clinical use leads to a vast array of complications. Osteoporosis is the most common orthopedic side effect of steroid abuse, while osteonecrosis is a rare occurrence. The risk of osteonecrosis appears to be dose and duration dependent, but several patient factors also play a major role and usually affect the femoral head followed by the knee joint. The long-term effects of steroids must be explained to all patients on therapy, but this risk is missed in individuals who abuse steroids for recreational or performance-enhancing purposes. We describe a male, aged 29 years, who presented with dull aching bilateral knee pain of 2-years' duration after a long-term steroid abuse for weight and muscle mass gain. Radiological and magnetic resonance imaging studies confirmed osteonecrosis of femoral and tibial condyles and secondary degenerative arthritis of the knee joint. Prompt suspicion, early diagnosis, and intervention in osteonecrosis of knee joints, and termination of steroids may reverse the pathology and prevent progression of disease.

**Keywords:** Avascular necrosis of bone; Glucocorticoids; Knee osteoarthritis; Osteonecrosis; Performance-enhancing substances

Glucocorticoids are commonly used drugs owing to their anti-inflammatory and immunosuppressive properties.[1] Their use remains vast in various rheumatological diseases, asthma, inflammatory bowel disease, post-transplant chemotherapy, cancer therapy, and treatment of severe viral infections.[2] The recent COVID-19 pandemic has surged the use of oral corticosteroids in patients with fatigue and myalgia to improve survival and reduce the hospital stay.[3] Although steroids are landmark drugs that can be life-saving or organ-saving, they are not without side effects and long-term complications, and their use must be judicial. Osteoporosis is a common complication of long-term steroid use, while osteonecrosis (avascular necrosis) is rare.[4] Endothelial damage, genetic predisposition, dysbarism, coagulation defects, and local anatomical tendency are theories postulated for steroid-induced osteonecrosis, but the cause remains largely unknown.[5] The risk of osteonecrosis development appears to be dose and duration-dependent, but several patient factors such as genetic susceptibility and pre-existing disease states also play a major role.[6] The femoral head is the most common site involved in osteonecrosis followed by femoral condyles, tibial condyles, talus, and bones of feet and hands. The abuse of anabolic androgenic steroids for muscle building and performance enhancement is a well-documented public health hazard with a significant effect on various organ systems.[7] However, there is limited evidence of glucocorticoid abuse for the same reasons, hence the risk of osteonecrosis can be attributed to non-

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pharmacological abuse of steroids. Reports from Middle Eastern countries suggest nearly 20% of the population consumed glucocorticoids for weight gain.[8] Consumption of oral corticosteroids for weight gain or recreational use has not been documented in India. We describe such a case of bilateral osteonecrosis of the knee in an adult male after the recreational use of oral corticosteroid.

## Case Report

A male patient, age 29 years, (body mass index: 23.5) presented to the orthopedic clinic with complaints of bilateral knee pain for 2 years. His pain was initially intermittent and exertional but slowly progressed to a contiguous dull aching type (visual analog scale = 8). He also complained of a visible swelling around his right knee for one year, associated with difficulty squatting and sitting cross-legged. The patient had a history of consuming oral steroid tablets (dexamethasone 7.5 mg) daily for about a year before the onset of his symptoms. He claimed to have consumed them to build his muscle mass. Following the development of knee swelling, he gave a history of four intra-articular injections of an unknown drug and aspirations from the right knee over the past year. The patient's pain and swelling subsided transiently following each articular injection, but the swelling eventually increased with the persistence of the same dull aching pain. The patient gave no history of any medical illnesses nor did he report being a smoker or alcoholic. There was no history of trauma to either knee before the onset of his symptoms.


**Figure 1.** Gross wasting of the thigh and gluteal muscles of the patient is seen from lateral [A] and anterior [B] profiles with multiple scars over the knee joint and hyperpigmentation of the skin.


**Figure 2.** Radiographs of both knees showing diffuse osteopenia and multiple radiolucent lesions. The white arrow represents an osteolytic lesion in the medial femoral condyle of the right knee with sclerotic margins and flattening of the condyle.

There was no history of any bleeding episodes, blood transfusions, radiation or chemotherapy received, abdominal pain, or skin lesions.

A physical examination of the patient revealed a painful and swollen right knee with supra and parapatellar fullness and restricted joint motion. Both knees were tender on palpation of femoral and tibial condyles with marked tenderness of the right medial joint line and diffuse synovial thickening. Gross wasting of his thigh and calf muscles was noted along with multiple scar marks on the right knee (Figure 1). Both hips and ankles were normal. Abdominal examination revealed no hepatomegaly or splenomegaly. Blood parameters were unremarkable with normal hemoglobin, white blood cell count, erythrocyte sedimentation, C-reactive protein, liver function, and kidney function tests. Rheumatoid factor, anti-cyclic citrullinated antibody (anti-CCP), and anti-nuclear antibody (ANA) were negative. The sickling test was negative, and hemoglobin electrophoresis was normal. Synovial fluid was aspirated (80 ml of straw-colored fluid), and analysis showed normal glucose and protein levels. Cell count was

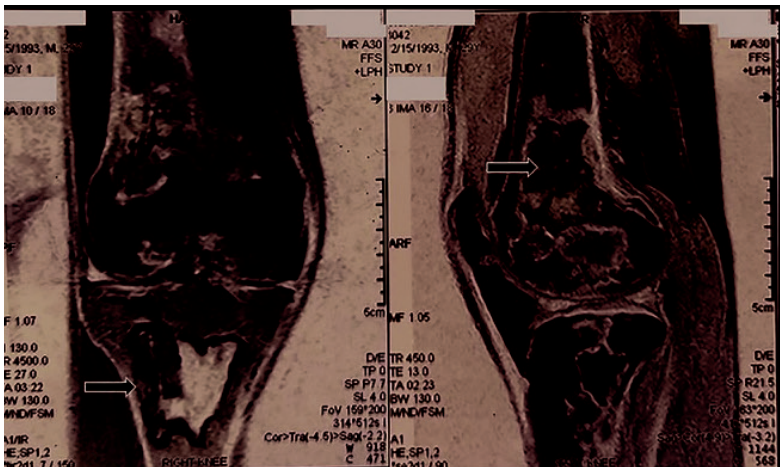

**Figure 3.** Magnetic resonance imaging of the right knee showed multiple medullary meta-diaphyseal lesions in the proximal tibia and distal femur with serpiginous border and the classical double line sign (white arrows) and subchondral necrosis of condyles.

normal, and the gram stain and Zeihl-Neelsen (ZN) stain were negative for any organisms.

A radiological assessment was done, which narrowed the diagnosis. Radiographs of both knees showed multiple radiolucent lesions surrounded by sclerotic margins. An osteolytic lesion in the right medial femoral condyle with subchondral collapse and reduced joint space was evident, representing Koshino stage 3 osteonecrosis as depicted in Figure 2.[9] Magnetic resonance imaging (MRI) revealed the presence of medullary meta-diaphyseal lesions in the proximal tibia and distal femur with a serpiginous border and double line sign (Figure 3). Subchondral necrosis of femoral and tibial condyles was evident. Synovial thickening and moderate joint effusion were seen. All features were representative of osteonecrosis of the knee.

Due to the significant joint swelling, an arthroscopic synovectomy was performed on the right knee. Diagnostic arthroscopy revealed inflamed and hypertrophied synovium and Grade 4 cartilage changes and subchondral fracture with necrosis of the subchondral bone of the medial femoral condyle. The articular cartilage surface of the patella and lateral femoral condyle was found to be healthy along with intact ligaments. Biopsy from the medial bone defect was suggestive of bony fragmentation with fibro collagenous tissue and areas of hemorrhage and necrosis without signs of infection. Based on clinical, radiological, histologic, and arthroscopic features, the patient was diagnosed with osteonecrosis of the bilateral knee, possibly due to chronic steroid abuse.

The patient was kept non-weight bearing after the arthroscopic surgery and is planned for a total replacement of both knees in the future. Oral bisphosphonate (alendronate 70 mg weekly), calcium (calcium citrate maleate 500 mg daily), and vitamin D (60,000 IU weekly) therapy was initiated. Due to the chronic consumption of low-dose oral corticosteroids, a skeletal survey of both his hips was performed despite no hip symptoms, and it revealed avascular necrosis of both his femoral heads. MRI scan was done and Steinberg stage II/III avascular necrosis was seen in both femoral heads (Figure 4).

## Discussion

Ahlbäck initially described osteonecrosis of the knee, which can lead to end-stage arthritis of the knee joint.[10] Osteonecrosis of the knee is the second most common affected part after the hips. Knee osteonecrosis can be divided into three categories: spontaneous (SONK/ also called primary or osteonecrosis of the knee), secondary (ischemic/atraumatic/ idiopathic osteonecrosis), and post-arthroscopy osteonecrosis.[11] The most common type of knee osteonecrosis is SONK, which mainly affects patients above age 50 years. This is followed by secondary osteonecrosis, which can be due to multiple medical conditions such as sickle cell disease, alcohol consumption, corticosteroid abuse, myeloproliferative disorders, and tobacco abuse.[12]

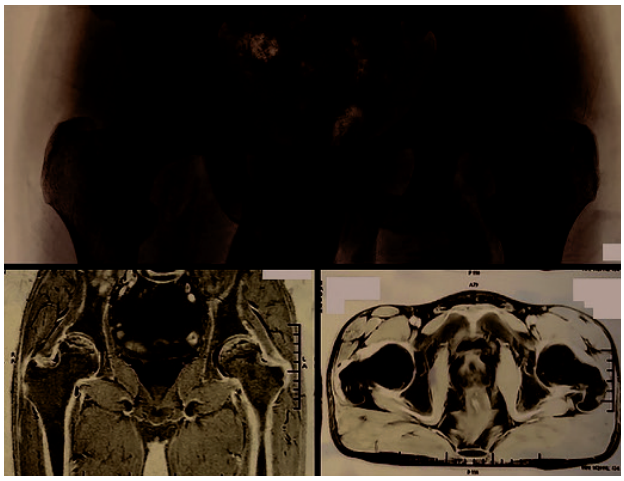

**Figure 4.** Upper panel: Radiograph of both hips showing sclerosis of both femoral heads and subchondral crescent sign. Lower panels: MRI images of both hips confirming Steinberg stage II/III osteonecrosis of femoral heads.

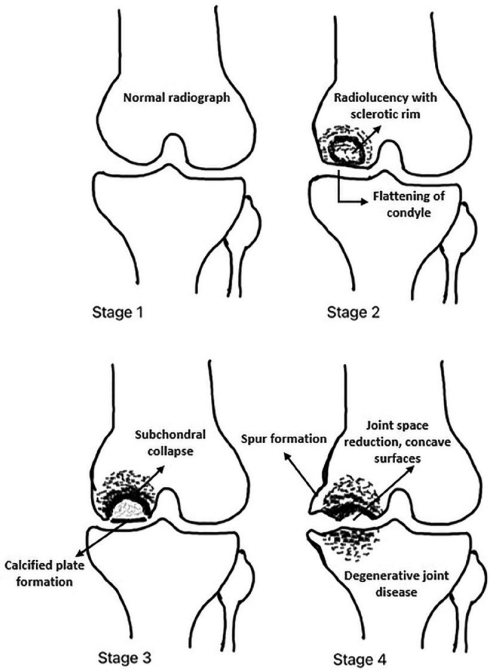


**Figure 5.** Koshino staging of spontaneous osteonecrosis of the knee.

Steroid-induced avascular necrosis (AVN) accounts for 10%–30% of secondary osteonecrosis, which is the most common cause.[13] In a study conducted by Shigemura et al.,[14] alcoholism was found to be less commonly associated (18.3%) with osteonecrosis as compared to corticosteroid use (54.9%) in 131 patients with femoral head osteonecrosis. There have been multiple studies reporting the association of steroid use in various conditions such as rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease, asthma, organ transplantation, and the occurrence of osteonecrosis.[1,3,5,13,15–24]

The pathophysiology of steroid-induced osteonecrosis is debated, and multiple mechanisms have been described. The development of bone necrosis is not caused by a single event but by multiple effects of steroids on the normal homeostasis of bone which influence each other. Steroids lead to reduced bone marrow stem cell proliferation, reduced vascular endothelial growth factor (VEGF) proliferation, adipogenesis, hyperlipidemia, decreased clotting factors, direct cell apoptosis, and oxidative injury to the endothelial cells. This results in vascular damage, lipid deposition, ischemic stress, and increased intra-osseous pressure, finally resulting in osteonecrosis of the bone.[25] Even though the natural effects of steroid use on the bone are well documented, controversy remains on the dose of steroid use, the duration of therapy, the route of administration, and possibly the genetic traits in certain individuals that lead to an increased risk of osteonecrosis. Recent cross-sectional data also supports the association of weight gain supplements containing herbal products with the development of bone osteonecrosis.[26]

Aaron et al.[13] studied the relationship between dose and duration of steroid use with the development of femoral head AVN and found a relative risk of 5.8 with the use of steroids for more than 700 days ($P$ = 0.10) and a risk of 8.8 for a cumulative dose of 9000 mg ($P$ = 0.02). In a case-controlled study on patients receiving high-dose corticosteroids after hematopoietic cell transplantation (HCT), the risk of development of AVN is 4 times, 5.6 times, and 8 times normal controls as compared to patients receiving <3870 mg, 3870-9735 mg, and >9735 mg, respectively.[19] It is observable that with higher cumulative doses and longer duration of steroid use, the risk of development of osteonecrosis rises. However, there must be a low degree of tolerance in screening patients for AVN even in patients receiving a pulse therapy of high-dose steroids.[16] This drastic complication, which is extensively reported on the oral abuse of steroids, can also develop with a local dose of intra-articular steroid injection as well.[17] Çalapkulu and Dharmshaktu have reported cases of bilateral femoral AVN in patients even on low-dose steroid therapy.[1,18] Osteonecrosis can also occur in the knee following an intra-articular corticosteroid injection, affecting the medial tibial

**Table 1.** Koshino classification of osteonecrosis of the knee (1979)

| Koshino stage [9] | Description | Age (years) | Treatment |
|---|---|---|---|
| 1 | No changes in radiographs | Any | Conservative |
| 2 | Subchondral lucency surrounded by osteosclerosis and flattening of weight-bearing area | Any | Conservative/core decompression & bone grafting |
| 3 | Extension of radiolucencies and subchondral collapse | < 60 | UKA |
| | | ≥ 60 | TKA |
| 4 | Osteosclerosis, osteophytosis, and degenerative disease | < 60 | UKA |
| | | ≥ 60 | TKA |

UKA, Unicompartmental knee arthroplasty; TKA, Total knee arthroplasty

condyle.[22] Yousef et al.[20] reported a case of bilateral knee osteonecrosis following long-term intra-nasal steroid therapy. Various genes also contribute to osteonecrosis, which include eNOS, PAI-1, VEGF, and ApoA. Mi-RNA, which are non-coding RNAs of 21-23 base pairs, have also been associated with the development of AVN.[5]

Patients with osteonecrosis of the knee generally present with spontaneous onset of medial-sided knee pain. Pain may worsen during the night and on bearing weight on the affected knee, and tenderness may be classically elicited over the femoral or tibia condyles.[12] Investigations include radiographs, bone scan, and MRI. Radiographs are usually negative during the early stages of the disease, with radiolucency, subchondral collapse, condylar flattening, and osteochondral defects seen in later stages. Koshino et al.[9] first described the radiographic stages of SONK in 1979 (Table 1). Progression of disease eventually leads to reduced medial joint space and secondary degenerative arthritis (Figure 5).[12]

Scintigraphy studies show an initially reduced uptake of the radionucleotide followed by an increased uptake at later stages.[27] The most sensitive tool for detecting bone osteonecrosis is MRI.[28] Features of early osteonecrosis include low signal intensities on T2W images in the subchondral bone and focal epiphyseal depressions. The classical appearance of a serpiginous band of low signal intensity on the T1W image and two rims, one low signal outer line and a high signal inner line on the T2W image ("*double line sign*") is diagnostic of osteonecrosis. The outer low signal line corresponds to the junction between normal and ischemic bone, comprising fibrous and sclerotic tissue, whereas the inner high signal depicts the reparative zone comprising fibro-vascular tissue.[5]

Treatment can be broadly divided into non-operative and operative options. Non-operative options are reserved for the early stages of osteonecrosis and are limited in number. The primary aim is pain management and is by using NSAIDS and local non-pharmacological agents such as extracorporeal shock wave therapy and ultrasonography. Protected weight bearing to prevent progression into medial compartment erosion. Oral therapy using bisphosphonates has been recommended in a few case series but concrete evidence of its role in reversing osteonecrosis is yet to be established.[29] A randomized control trial by Meier et al.[30] showed no benefits of oral bisphosphonates (ibandronate) over NSAIDS.

Operative techniques are generally reserved for larger defects or higher grades of necrosis. Defects <3.5 $cm^2$ or <20% condyle involvement can be treated without surgery. Condyle involvement >5 $cm^2$ or >50% warrants surgery, as these large lesions can lead to collapse.[11,31,32] Surgical management is broadly divided into joint preserving and joint replacing surgeries. Joint preserving techniques include arthroscopic debridement, synovectomy, core decompression, osteochondral autograft, and high tibial osteotomy.[33–36] Core decompression of the femoral condyle and artificial bone grafting using calcium hydroxyapatite may also be done.[37] High tibial osteotomy shows a reduction in synovial inflammation and cartilage destruction when performed on patients with <4 $cm^2$ defects.[38] Successful osteochondral autologous transfer has been shown to reduce joint replacement surgery by at least a decade.[35]

Since all joint preserving surgeries only aim at delaying the replacement surgeries, the latter is the mainstay of treatment in osteonecrosis involving more than half the condyle. Unicondylar knee arthroplasty (UKA) can be performed in lesions involving only the medial compartment, whereas total knee arthroplasty (TKA) is reserved for patients with a diffuse involvement. In a long-term cohort study by Flury et al.,[39] both UKA and TKA had excellent outcomes in patients with osteonecrosis of the knee and were not dependent on the size/location of the defect.

## Conclusion

Osteonecrosis due to steroid intake is a debilitating complication that leads to pain, limitation of motion, and restricted activities. Due to the multifactorial effects of steroids on the bone, it is difficult to predict the occurrence of necrosis and the degree of involvement. Hence, all patients on steroid therapy must be counselled regarding its complications, particularly osteonecrosis. Early diagnosis and treatment by non-surgical and joint-preserving surgeries may add life to the affected joint and can delay the inevitable replacement surgeries.

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