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Case Report

Bone betrayal: avascular necrosis in psoriatic arthritis

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ABSTRACT

Psoriatic arthritis is a multifaceted inflammatory condition with varied musculoskeletal manifestations. Although hip joint involvement remains uncommon, but may lead to severe sequelae such as avascular necrosis (AVN), especially in the presence of steroid exposure. We report the case study of a young male with history of chronic plaque psoriasis who presented with pain of lower back and right hip. There were no records of any corticosteroid exposure, trauma, alcohol consumption or any metabolic disorders. On examination, the patient was found to have enthesitis and nail changes, suggesting possibility of psoriatic arthritis. Magnetic resonance imaging revealed grade 2 AVN of the right femoral head without sacroiliitis. This case study highlights the possible occurrence of AVN in patient with psoriatic arthritis. This could be attributable to the mechanisms causing systemic inflammation and disruption of the vascular integrity. It emphasizes the need to screen for atypical manifestations in PsA so as to enable timely management.

Keywords: Psoriatic arthritis, Avascular necrosis, Hip joint, Enthesitis

INTRODUCTION

Psoriatic arthritis represents a chronic autoimmune arthropathy affecting 30% of the individuals diagnosed with psoriasis.[1] Although peripheral joint involvement have diverse clinical presentations, involvement of the axial skeleton, particularly affecting the hip region-is relatively infrequent. Nonetheless, the presentation of hip involvement has an aggressive course with substantial compromise of mobility.[2]

The disease mechanisms involve intricate interactions between genetic predisposition, compromise of hosts' immunity and environmental triggers. The outcomes of this result in inflammation of the synovium and loss of vascular integrity.[3] Consequently, individuals with psoriatic arthritis have increased susceptibility to develop coagulation abnormalities, thromboembolic phenomenon, hyperuricemia and homocystinemia.[4,5]

Avascular necrosis (AVN) (osteonecrosis) is a severely debilitating condition characterized by necrosis and death of bone tissue due to ischemia. Load bearing joints, particularly the hip (acetabulofemoral) joint is the predominant site affected, which is associated with destruction of head of femur and disintegration of surface of the joint.[6]

Patients present with excruciating pain in hip joint during load bearing activities and marked deterioration while performing daily activities.

Prolonged use of corticosteroids and chronic alcohol abuse are the predominant causes for non-traumatic AVN

in autoimmune disorders such as systemic lupus erythematosus, myositis and rheumatoid arthritis.

However, occurrence of osteonecrosis in individuals with psoriatic arthritis is remarkably infrequent and very few cases have been reported worldwide.[2,4]

This case report describes the unusual presentation of osteonecrosis in a young male diagnosed with chronic plaque psoriasis.

In this case report, we describe the atypical presentation of AVN in a young male with chronic plaque psoriasis.

CASE REPORT

A young male of 27 years with a history of chronic plaque psoriasis presented with complaints of chronic pain of 2 years duration in the lower back and right hip. 5 years ago, he had scaly, pruritic plaques involving the occipital area of the scalp which had gradually progressed over to the cheeks, glabella and over to ears. The patient had used multiple treatments such as topical agents and homeopathic therapy, which failed to provide him any relief, thus resulting in cessation of all treatments.

Two years ago, the patient started to experience right sided dragging pain in the lumbar region, which was radiating upto the heel. The patient had no early morning stiffness, neurological symptoms, or any difficulties in turning in bed.

In addition to this, 6 months ago, the patient also experienced discomfort in the right metatarsophalangeal joint and transient pain in the left fifth toe. The patient did not have other systemic symptoms or other articular involvement.

On physical examination, patient was found to have multiple erythematous scaly plaques on bilateral cheeks, auricular areas and over the scalp. The PASI score was 0.8. The nail examination revealed pitting and onycholysis.

On musculoskeletal examination, the patient had right sacro-iliac joint tenderness along with bilateral enthesitis of Achilles tendon. Leed's Enthesitis index was 2/6. There was no restriction of hip and knee movements. Patrick's test was negative. There were no spinal tenderness or cervical abnormalities. All the above clinical features were suggestive of Psoriatic arthritis.

Laboratory investigations revealed normal complete blood picture, renal function tests and inflammatory markers (CRP, ESR) and HLA-B27. MRI of the sacroiliac joints and spine revealed grade 2 osteonecrosis of the right femoral head along with anterior wedging and changes in the endplates of lower thoracic vertebra, consistent with Scheurmann's disease.

Further evaluation of risk factors of AVN, including sickle cell disease and antiphospholipid antibody syndrome were done which were negative.

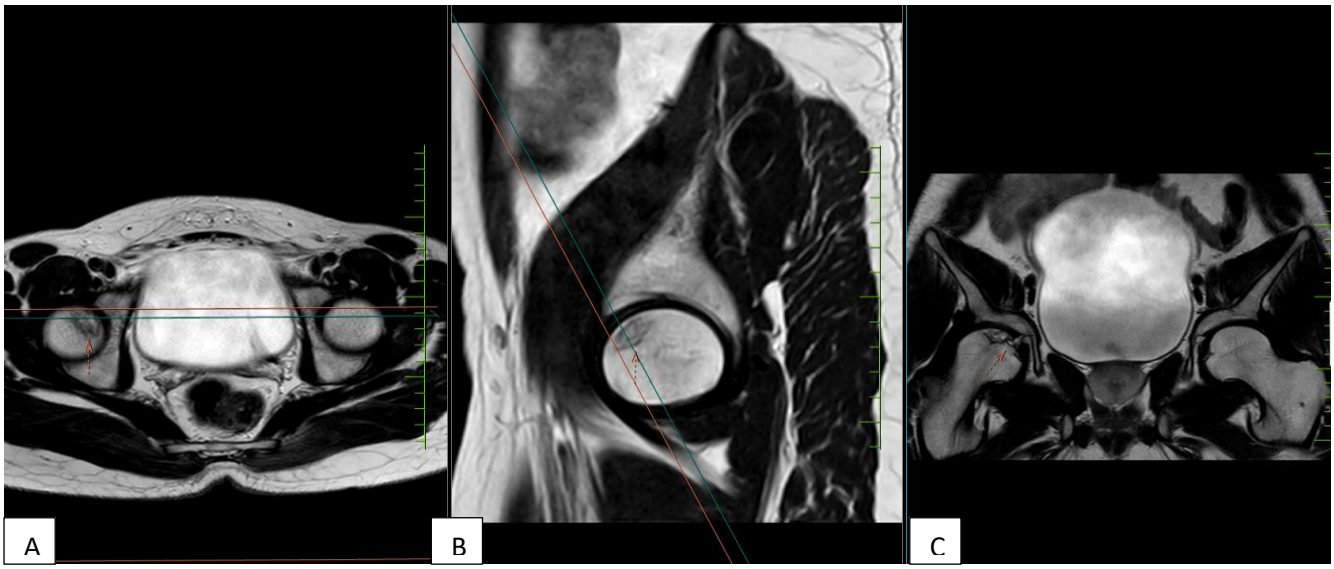

Figure 1 (A-C): MRI images of right hip joint demonstrating joint space narrowing and osteonecrotic changes in right femoral head (yellow circles).

DISCUSSION

The presence of AVN in conjunction with PsA is a rare but clinically significant presentation. PsA is a chronic immune mediated condition wherein the immune mediated destruction of articular surfaces is mediated by pro-inflammatory proteins like TNF and the interleukins (IL-6). These cytokines in addition to the promoting inflammation in the synovial tissue, also damage the vascular integrity leading to the sequelae of

inflammation, occlusion and ischemia. Endothelial dysfunction in conjunction with hypercoaguable state predisposes the individual to the development of AVN, despite of lack of traditional risk factors like chronic use of corticosteroids, alcohol consumption, and the trauma.[6,7]

In this case report, we describe a 27-year-old male with history of chronic plaque psoriasis, with enthesitis, cutaneous and ungual changes, who presents with grade 2 AVN of right head of femur. The absence of conventional causes of AVN such as corticosteroid use, alcohol intake and hematological conditions, make this case study a unique occurrence of idiopathic osteonecrosis. Absence of the typical arthropathy features of PsA such as sacroiliitis, or involvement of axial joints on imaging, along with presence of abnormalities of vertebral endplates which are consistent with Scheurmann's disease, indicates the complex nature of musculoskeletal involvement in this patient population.

Hickman et al were the first ones to report the presentation of non-traumatic AVN in PsA patient.[7] They reported a series of 10 patients with PsA, 5 out of whom developed AVN in absence of corticosteroid treatment, suggesting involvement of intrinsic inflammation as a possible mechanism in the pathogenesis of PsA. The present case closely aligns with this pattern, thus demonstrating that even in the absence of pharmacological or metabolic risk factors, vascular compromise and its sequelae in form of ischemia of bone can occur.

Michet et al reported that 6.3% of patients with PsA had involvement of the hip joint, with most of them requiring early hip replacement due to accelerated damage of the hip joint and functional deterioration.[8] They also observed varied patterns of arthropathy such as sacroiliitis and spondylitis along with hip joint involvement. In present case, there was no sacroiliitis, but there was evidence of vertebral endplate destruction which indicates the diverse presentations of PsA.

In a nationwide analysis of PsA by Chiu et al they further reinforced the connection between severe PsA and increased risk of AVN.[2] Their comprehensive study revealed a higher inflammatory burden and thus higher risk of AVN, especially in the young male population. The clinical profile of our patients-age below 30 years, presence of enthesitis and nail involvement, mirrors the population at risk of their study.

Recent study by Tang et al demonstrated that TNF enhances osteocyte mediated RANKL expression and actively promotes osteoclast development.[9] This highlights the crucial mechanism of bone destruction in absence of steroid use mediated bone damage, thus proving that presence of chronic inflammation itself is sufficient to trigger AVN in susceptible individuals.

Lechner et al reported that TNF plays a critical role by not only up regulating RANKL expression, but also increases bone resorption significantly along with simultaneous inhibition of osteoblast activity.[10] They also reported that TNF suppresses osteoprotegerin production and induces osteoblast apoptosis, thereby creating a favorable environment for bone necrosis.

Park et al reported the occurrence of AVN in an elderly female patient with PsA.[6] However, unlike the present patient, the patient in Park's study underwent surgical intervention due to rapid joint destruction.

CONCLUSION

The occurrence of AVN in a young male patient with PsA, in the absence of traditional risk factors of AVN highlights the existence of pathological processes in which systemic inflammation, endothelial injury and cytokine mediated bone destruction. Periodical screening of PsA patients for development of potential AVN, especially in the high risk cohorts such as younger males, is necessary to improve early detection, improve patient's quality of life and avert surgical interventions.

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