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Knee Joint Pain Potentially Due to Bone Alterations in a Knee Osteoarthritis Patient

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 83
Final Diagnosis: Osteoarthritis
Symptoms: Knee joint pain
Medication: —
Clinical Procedure: Resting
Specialty: Orthopedics and Traumatology

Objective: Challenging differential diagnosis
Background: Osteoarthritis (OA) is the leading cause of musculoskeletal pain and functional disability worldwide. However, the etiology of this condition is still largely unknown.
Case Report: We report the clinical course of an elderly man with knee OA.
Conclusions: Plain radiographs and MRI examinations performed during follow-up suggested that the pathophysiology of the patient's knee OA and joint pain may have been primarily due to bone alterations.

MeSH Keywords: Arthralgia • Knee Joint • Magnetic Resonance Imaging

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/891233>

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Background

Osteoarthritis (OA) is characterized by degeneration and destruction of articular cartilage that results in pathological bone changes [1]. In recent studies on the relationship between OA progression and bone lesions, alterations were found to occur ahead of obvious cartilage degeneration and OA. Joint pain also frequently indicates the presence of microfractures, as do signal alterations in bones in magnetic resonance imaging (MRI) of patients with hip joint pain [2,3]. Although we have reported that the pathophysiology of hip OA presumably includes bone alteration, the pathogenesis of bone changes in this context is still largely unknown.

There exist numerous studies on the relationship between OA and MRI findings [4,5]. In this report, we describe a patient with knee OA and the associations among the pathophysiologicals of OA, joint pain, and MRI findings.

Case Report

An 86-year-old man presented at our institution with severe right knee joint pain in May 2011. The patient complained of right lower leg edema that had coincided with the occurrence of joint pain. The edema persisted until the knee pain stopped. Alendronate administration was begun in October 2011 immediately after he was diagnosed as having osteoporosis. His knee pain had become greatly improved by May 2012. At the first visit, plain radiographs revealed a bone cyst and joint space narrowing (Figure 1A, 1B) (Kellgren and Lawrence grading [KL] grading II) [6]. Two years later, his joint

pain had disappeared. At that time, plain radiographs showed increased joint space narrowing (KL grading II) and localized bone formation (Figure 2A, black arrow) at the proximal lateral side of the tibia and MRI analysis depicted a cyst in the proximal medial tibia (Figure 2B, 2C). Three months later, his knee pain returned. MRI examination disclosed broad signal changes in the distal medial femur and in the proximal medial tibia by T1-weighted imaging (T1W) (TR: 530 [SI], FA: 90 [SI], and TE: 14 [SI]) and short τ inversion recovery (STIR) (TR: 5010 [SI], FA: 180 [SI], TE: 71 [SI], and TI: 150 [SI]) (Figure 3A, 3B). Plain radiographs showed no OA progression or localized bone formation at the proximal lateral side of the tibia (data not shown). Four months afterwards, his knee joint pain was improved, but plain radiographs revealed further joint space narrowing and localized bone formation (Figure 4B, black arrow) at the proximal lateral side of the tibia (KL grading II) (Figure 4A, 4B).

The patient was informed that data from the case was desired for publication and he gave his consent.

Discussion

We encountered an aged man with knee OA. Plain radiographs at his first visit showed mild OA-like features. MRI analysis revealed a medial tibial cyst during the pain-free period and broad bone signal changes in the joint at the time of severe knee pain. The OA later progressed radiographically to a more severe stage, suggesting that the cause of OA and joint pain may have been bone alterations in the knee, as also seen in a hip joint we described previously [2,3].

Figure 1. Plain radiograph displaying a bone cyst and joint space narrowing, but no localized bone formation at the proximal lateral side of the tibia (KL grading II). (A) frontal view, (B) lateral view.

Figure 2. Plain radiographs revealing progressed OA and localized bone formation (**A**; black arrow) at the proximal lateral side of the tibia (KL grading II) (**A**; frontal view). Osteonecrosis in the proximal medial tibia was seen by T1W (**B**; white arrow) and STIR (**C**; white arrow).

Figure 3. MRI indicating broad signal changes in the distal medial femur and in the proximal medial tibia by T1W (**A**) and STIR (**B**).

Muraki et al. have reported that in a sample of Japanese individuals over 60 years of age, 47.0% of men and 70.2% of women had radiographic knee OA. However, the incidence of knee pain among the patients was only 21.2% and 27.3% respectively [7]. Clegg et al. have observed that joint pain had spontaneously improved 6 months after onset in a non-medicated group [8]. These results suggest that many OA patients do not have joint pain. Furthermore, it seems that knee joint pain in most OA patients improves naturally.

There exist numerous reports on the relationship between knee OA and bone marrow lesions (BMLs) in knee MRI proposing that BMLs change over time and contribute to the pathophysiology and progression of radiographic knee OA [4,5]. In one of the largest studies of its kind, Sofat et al. reported that knee joint pain was found in 77.5% of individuals with knee OA and BMLs [9]. In our patient, broad bone alterations detected by MRI were observed in the medial knee joint. These findings strongly suggest that the pathophysiology of knee OA

Figure 4. Plain radiograph demonstrating progressed joint space narrowing and localized bone formation (**B**; black arrow) at the proximal lateral side of the tibia (KL grading II). (**A**) frontal view, (**B**) lateral view.

and joint pain are BMLs. Our data also showed no apparent relationship between radiographic OA features and joint pain, but rather that OA progressed after bone signal changes were detected by MRI. This evidence implies that the bone is the primary region of OA occurrence and progression.

Taljanovic et al. have compared MRI findings with histology in 19 hip OA subjects who had undergone hip surgery. The authors concluded that bone marrow edema detected by MRI in hip OA correlated with the severity of pain, radiographic findings, and microfractures [10]. Other groups have recently described that such MRI signal changes in joints frequently suggest microfractures [4,9,10]. We also have reported that bone changes could be the primary cause of hip OA and that the pathophysiology of hip OA could be microfractures [2,3]. Collectively, this body of evidence strongly implicates microfractures with knee OA pathophysiology.

Lastly, we detected localized bone formation at the proximal lateral tibia in a follow-up plain radiograph that had not been radiographically evident at the first visit, indicating that bone formation had apparently increased over time. This finding resembled the callus observed in the process of general fracture healing. It is currently not known why or how localized bone formation is present in OA. However, because imaging reflected OA progression in this case, localized bone formation might also be a hallmark of bone microfractures and knee OA progression.

Conclusions

We encountered the case of an aged man with knee OA whose plain radiograph and MRI findings revealed OA progression that was potentially due to bone alterations. Specifically, microfractures may be implicated with OA pathogenesis.

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