Rapid bone destruction in a patient with knee osteoarthritis. A case report and review of the literature

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Summary

We report the clinical outcome of an elderly man with knee osteoarthritis (OA) accompanied with recurring severe joint pain. He had no history of trauma to the affected knee. Plain radiographs and magnetic resonance imaging uncovered rapid and severe bone deformity, which likely led to the patient’s progressed radiographic OA. These findings indicate that a pathophysiology of OA may be bone alterations.

KEY WORDS: knee; osteoarthritis; rapid bone destruction.

Introduction

Osteoarthritis (OA) is the most common disease of the joints, affecting more than 60% of the elderly population. The pathogenesis of primary OA suggests an intrinsic disease of the cartilage in which biochemical and metabolic alterations result in its breakdown (1). Sulzbacher recently reported that changes in subchondral bone preceded cartilage degeneration, which suggested primary alterations of the subchondral region (1). Although OA is a multifactorial disease process, early cartilage damage and ultimate loss of articular cartilage are considered as central features and significant contributors to OA progression (2).

Bone alterations detected by magnetic resonance imaging (MRI) may be one of the main etio-pathophysiologies of OA (3, 4). In this disorder, affected bone regions appear as low intensity areas by T1-weighted imaging (T1W) and high intensity areas by short-tau inversion recovery imaging (STIR), which indicate the presence of water or bone marrow edema (6). Taljanovic et al. suggested that the pathophysiology of bone marrow edema in OA hips may be microfractures based on patho-histological findings (7). It has been also reported that bone alterations detected by MRI in patients with hip OA might have been microfractures that led to OA (3, 4).

In addition to cartilage degeneration (8), bone or joint loss is another major pathophysiologically OA (9). Although there have been several accounts of joint deformity, such as osteophytes, subchondral osteosclerosis, and bone cysts, being important pathological hallmarks for OA (10), the mechanism by which bone deformity, such as varus or valgus, occurs has not yet been reported.

Plain radiographs are generally used to identify the specific features predictive of OA progression (2). It was recently described that one of the causes of OA was bone alteration as detected by MRI (3, 4). However, the association between knee deformity and MRI-detected lesions in patients with OA remains unaddressed.

In clinical practice, we often encounter cases of knee deformity accompanying OA that rapidly deteriorate in spite of no apparent history of knee trauma. To the best of our knowledge, the precise mechanism of rapid and severe OA progression apart from traumatic event, severe osteoporosis, or overuse has not been described.

In this study, we report the clinical outcome of an elderly man with right knee OA. His knee deformity progressed rapidly in plain radiographs as well as in MRI examinations, possibly due to bone alterations.

Case report

A 73-year-old man had been visiting our institution for 3 years since April 2011 for bilateral knee pain. His height was 157.4 cm his body weight was 59.0 kg. He had no serious past medical history. Clinical examination revealed the range of motion (ROM) of the right and left knee were 120 and -20 degrees and 130 and -20 degrees, respectively. Plain radiographs showed Kellgren and Lawrence (KL) grading II in the right knee joint, localized bone formation at the proximal lateral tibia, and a bone cyst in the middle of the proximal tibia plateau at the initial visit (Figure 1a). As conservative treatment, non-steroidal anti-inflammatory drugs (NSAIDs) were prescribed and right knee puncture was performed periodically for pain management.

At the first presentation, MRI of his right knee revealed broad bone signal changes by T1W and STIR in the proximal tibia plateau and the initial visit (1a). As conservative treatment, non-steroidal anti-inflammatory drugs (NSAIDs) were prescribed and right knee puncture was performed periodically for pain management. At the first presentation, MRI of his right knee revealed broad bone signal changes by T1W and STIR in the distal-medial femur and proximal-medial central tibia (Figure 1b, c). As described previously (3, 4), bone signal changes were judged to be present only when they were detected by both T1W and STIR.

Clinical Cases in Mineral and Bone Metabolism 2014; 11(3): 232-235
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During the observation period, the size of the patient’s localized bone formation at the proximal lateral tibia changed constantly (Figures 1a, 2a, 3a), but his severe knee pain soon subsided following therapeutic measures. In March 2013, the patient’s right knee pain recurred. Plain radiographs revealed progressed OA (KL grading IV) (Figure 2a) compared with the first visit. MRI of the joint showed worsened broad bone signal changes by T1W and STIR in the distal-medial femur and proximal-mediocentral tibia and larger bone defects in the medial knee joint (Figure 2b, c). The conditions of bone mineral density (BMD) examination at the L1-4 levels of the posteroanterior spine and bilateral proximal femur were as reported previously (3, 4). The patient’s BMD at the time for the L1-4 levels was 1.331 g/cm², YAM: 113%, T-score: 1.8, and Z-score: 3.2, and that for the total hip levels was 1.071 g/cm², YAM: 114%, T-score: 1.0, and Z-score: 2.3. Conservative treatment again improved his pain.

Figure 1 a-c - a) Plain radiograph at the first visit shows KL grading II in the right knee. Subchondral sclerosis, osteophytes, and joint space narrowing at the medial joint are evident. A large bone cyst (black arrow) is observed on the middle of the proximal tibial plateau. Localized bone formation in the proximal lateral tibia is apparent (white arrow). b, c) Broad low-intensity area by T1W and high intensity area by STIR in the medio-central femur and tibia are observed.

Figure 2 a-c - a) Plain radiograph shows KL grading IV. Bone destruction in the medial tibia and varus deformity at the medial knee joint are seen. Localized bone formation on the proximal lateral tibia has become larger than at the first visit (white arrow). b, c) Broad low-intensity area by T1W and high intensity area by STIR in the medio-central femur and tibia are observed. Increased joint effusion in the medial joint is noted.

Figure 3 a-c - a) Plain radiograph shows KL grading IV. Severe bone destruction in the medial tibia and progressed varus deformity at the medial knee joint are seen. Localized bone formation on the proximal lateral tibia is evident, but decreased (white arrow). b, c) Broad low-intensity area by T1W and high intensity area by STIR in the medio-central femur and tibia. Markedly increased joint effusion in the medial joint is observed.
In January 2014, the patient returned to our institution complaining of right knee pain. The joint deformity had rapidly progressed; plain radiographs uncovered bone destruction in the medial distal tibia of the right knee (KL grading IV) (Figure 3a). MRI confirmed broad bone signal changes by T1W and STIR in the distal-medial femur and proximal-medial-central tibia. The bone defect in the medial knee joint was again larger than that of the second visit (Figure 3b, c).

Discussion

We encountered an elderly man whose right knee radiographic deformity worsened rapidly without obvious trauma or past history. Bone alterations were observed in MRI as the bone deformity progressed in this patient with OA accompanied with joint pain.

In a population-based study in Canada, the incidence of OA in women aged over 50 was substantially higher than that in men. OA in the knee and hand is also more common in women (1, 11). A recent study has shown that individuals with OA have a higher risk of death compared with the general population (12). Therefore, preventing the risk factors of OA is important, especially in women.

Walker et al. have shown that the rate of OA progression varies substantially among patients (2). A clinical challenge in managing patients with OA is identification of the individuals who are at risk for the rapidly progressive form of the disease. Mechanical factors such as obesity are a major contributor to the etiopathogenesis of OA (13). Meissier et al. have also demonstrated that overloading and/or repeated microtrauma produce high mechanical forces that lead to radiographically proven knee OA (14).

It is generally considered that joint deformity and remodeling occur according to Wolff's law, which states that mechanical loads affect bone architecture (15). The femoral neck includes two trabecular columns, one vertical and the other horizontal. Wolff's law defines that cancellous structures forming on the femoral neck are due to compression forces and the effect of physiological forces acting through living bone (16). Hammer later reported that Wolff's law accounted for his observations in a series of pathologically healed fractures (17).

The progression of OA appears to involve new bone structure alterations in the osteochondral junction (18). Harold described how load-bearing joints can be affected according to Wolff's law, which states that mechanical loads affect bone architecture (15). The femoral neck includes two trabecular columns, one vertical and the other horizontal. Wolff's law defines that cancellous structures forming on the femoral neck are due to compression forces and the effect of physiological forces acting through living bone (16). However, the best of our knowledge, there has been no report of radiographic OA knee deformity classification, which may include such features as proximal medial tibial collapse and recessed joint line aligned with the shape of the distal femoral cortical bone. Several reports have recently emerged on microfractures assessed by MRI in OA (3, 4).

We have proposed a radiographic disease staging system for OA (26) that evaluates the presence of osteophytes, narrowing of joint space (excluding inter-osteophyte bridges), subchondral sclerosis, subchondral cysts, collapse of the central joint cortical bone, and deformity in the frontal plane (27). However, to the best of our knowledge, there has been no report of radiographic OA knee deformity classification, which may include such features as proximal medial tibial collapse and recessed joint line aligned with the shape of the distal femoral cortical bone. Several reports have recently emerged on microfractures assessed by MRI in OA (3, 4).

We have proposed a radiographic disease staging system for OA progression based on plain radiographs and MRI of hip joints as well (3, 4). Therefore, OA classification may optimally be based not only on plain radiographs, but also on MRI results.

In conclusion, earlier findings along with our patient's clinical course suggest that bone alterations are a major feature of OA and that the progression of OA might depend on the degree of radiographic bone alterations. MRI may also be useful in the diagnosis and follow-up of OA.

References

   2011;19:283-94.
   H. The Pathophysiology of Primary Hip Osteoarthritis may Originate from
   undergoes a transition in accordance with signal changes of bones detected
Rapid bone destruction in a patient with knee osteoarthritis. A case report and review of the literature


15. Wolff J. Das Gesetz der Transformation der Knochen. Berlin: A Hirschwald (Springer-Verlag published an excellent English translation of this monograph in 1898); 1892.


