JOURNAL OF CASE REPORTS 2015;5(1):84-87

Bone Alterations in Painful Osteoarthritic Shoulder

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Abstract:

This report describes shoulder osteoarthritis due to bone alterations which were confirmed radiographically. We report an 85 year old Japanese female with right shoulder pain. Radiographic findings showed bone signal changes using magnetic resonance imaging (MRI) and accumulation by bone scintigraphy in the affected area. The screening of other joint (left shoulder) showed no such changes. So, bone alterations may be involved in pathogenesis of shoulder osteoarthritis presenting with pain, which we want to emphasize in this case.

Key words: Shoulder, Osteoarthritis, Joints, Magnetic Resonance Imaging, Bone, Pain, Humans.

Introduction

Shoulder osteoarthritis (OA) is characterized by painful and restricted motion caused by the gleno-humeral joint degeneration and destruction. The pathophysiology of OA is generally considered to be degeneration and destruction of articular cartilage. Also, repetitive mechanical stress over the overlying cartilage during physiological movement, especially loading stress, causes OA in weight bearing joints such as in knee or hip joint [1]. However, shoulder joint is usually considered as non-weight bearing joint.

Recent evidences suggest not only the articular cartilage degeneration, but also disintegrity of multiple joint tissues in pathogenesis of OA [2]. Some authors have reported that bone alterations might be the primary pathophysiology of OA in hip joint [3,4]. In addition, there have been reports suggesting signal alterations of bones detected by magnetic resonance imaging (MRI) in the patients with joint pain, suggestive of microfractures which might be contributory to osteoarthritis [3-6]. To best of our knowledge, we have not come across any literature showing correlation between shoulder pain and bone alterations.

Case Report

An 85-year-old Japanese woman presented at our institution with severe pain while raising her right

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Received: October 9, 2014 | **Accepted:** December 29, 2014 | **Published Online:** February 25, 2015
This is an Open Access article distributed under the terms of the Creative Commons Attribution License (creativecommons.org/licenses/by/3.0)
Conflict of interest: None declared | **Source of funding:** Nil | **DOI:** http://dx.doi.org/10.17659/01.2015.0021

shoulder. She had no job history of overuse of shoulder joint. Her right shoulder had an apparent joint contracture while left shoulder had a slight joint contracture. Plain radiograph showed bilateral huge osteophytes in the humeral heads, joint space narrowing, and osteosclerosis in shoulders [Fig.1.a, b: both circled areas, KL grading scale IV]. MRI examination showed that heterogeneous low intensity by T1-weighted imaging (T1W) (TR: 530 (SI), FA: 90 (SI), TE: 14 (SI) and high intensity by short τ inversion recovery T2-weighted imaging (T2-STIR) in the only right proximal humerous head and acetabulum, but not in the left shoulder joint [Fig.1c-f]. Bone scintigraphy also revealed that the piled up accumulation was seen in the right shoulder while a little accumulation in the left shoulder. The contracture in the right shoulder improved to some extent by ROM exercise and the right shoulder pain substantially improved. Bone mineral density (BMD) was measured using a Dual-energy X-ray Absorption fan-beam bone densitometer (Lunar Prodigy; GE Healthcare, Waukesha, WI, USA) at the L1-4 levels of the postero-anterior spine and bilateral hip. The BMD values at L1-4 or bilateral hip were 1.077 (T-score was -0.6) or -.996 (T-score was 0.5), respectively. She has taken non-steroidal anti-inflammatory drugs (NSAIDs), celecoxib (200 mg per day), and tramadol (25 mg every night) to reduce her shoulder pain.

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

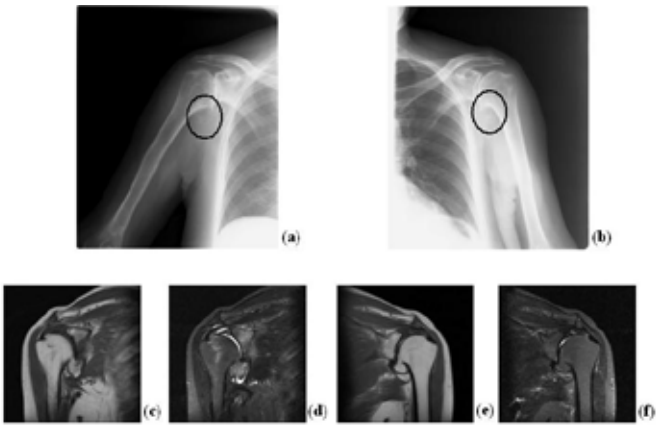

Fig.1: Plain radiograph showed KL grading IV in the bilateral shoulder joints. Huge osteophytes below the humeral heads (circled areas), joint space narrowing, and osteosclerosis were observed by plain radiograph **(a,b)**. Within a month after the onset, low intensity by T1W and high intensity by T2-STIR by MRI were observed only in the right shoulder, although no signal change was observed in the left shoulder **(c-f)**.

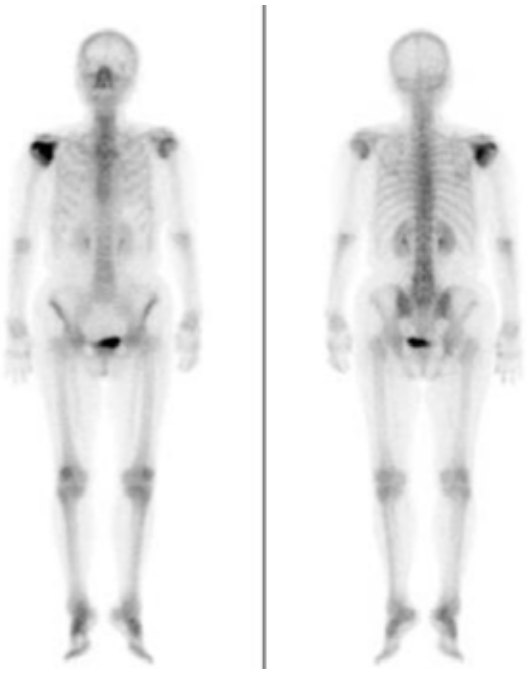

Fig.2: Bone scintigraphy showing accumulation in right shoulder.

Discussion

There have been a few reports on the prevalence of shoulder OA. Schaardenburg *et al*. report 4% prevalence of shoulder OA in the population aged older than 85 years old [7]. Oh *et al*. report that radiographic primary shoulder OA was 16.1%

and was relatively lower than that of knee OA (37.7%) [8]. In addition, according to the Research on Osteoarthritis Against Disability (ROAD) study in the Japanese elderly population (over 60 years), 47.0% in men and 70.2% in women had knee OA [9]. These results show that the incidence of shoulder OA is much less than that of knee joint, which is a loading joint. It is generally thought that the weight bearing stress is much less in shoulder joint than that in hip or knee joint. Therefore, it is explainable that the frequency of shoulder OA is much less than that of knee OA.

Recently, Sandell has reviewed the etiology of OA regarding genetic effects on joint structure [10]. Oh *et al*. also report that age and knee OA could be the risk factors of shoulder OA. In addition, bilateral shoulder OA was 7.7% in the population study of South Korea. The bilateral shoulder OA was present in 47.7% of those with primary shoulder OA [8]. Since shoulder OA occurs frequently in both shoulders compared to other joints, genetic effects might be involved in pathophysiology of shoulder OA.

In the Glucosamine/chondroitin Arthritis Intervention Trial (GAIT) study, joint pain significantly improved after a half year observation even in the placebo group [11]. According to the ROAD study, the incident rate of knee pain among knee OA patients was only 21.2% and 27.3% in men and women, respectively [12]. These results suggest that, in most OA cases, joint pain naturally may heal and that joint degeneration may not necessarily cause joint pain. Thus, the cause of joint pain in OA might not only be primarily cartilage affection.

Millett *et al*. very recently reported on the advanced shoulder OA cases, most of which were successfully treated by comprehensive arthroscopic management. The authors also introduce a case with huge humeral head osteophyte (goat's beard deformity) like in our case [13]. However, they did not mention the etiology of end-staged shoulder OA.

The osteophyte accompanying with OA is a bony outgrowth formed around the margins of the affected joint. However, the mechanism of osteophyte formation is largely unknown. Kaneko *et al*. recently reported that Perlecan (Hspg2), a heparan sulfate proteoglycan, plays an important role in osteophyte development in OA using genetically-modified mice [14].

We have previously reported that one of the pathophysiology of OA is bone alterations with probable microfractures [5,6]. It was also stipulated in our previous observations that bone alterations using MRI are observed at early staged-OA. Thereafter joint pain is improved but OA progresses radiographically. Bone alterations using MRI with joint pain were observed in most advanced staged-OA cases, while no signal change is seen when the patients have no joint pain [5,6]. Based on these findings, we have also proposed that OA can be classified into the following 3 stages using radiographic and MRI findings: i) MRI-based signal change stage, ii) Remission-staged OA, iii) End-staged OA. It is suggested that this right shoulder was at MRI-based signal change stage and this left shoulder was remission-staged OA [6].

The novelty of this case was that however clinically OA occurred in the both shoulders, but radiologically high signal intensity by STIR and low intensity by T1W detected by MRI and the obvious accumulation by bone scintigraphy were seen only in his right shoulder by bone scintigraphy. There was obvious contracture in the right shoulder and joint pain was reduced after improvement in contracture. These findings indicate that the joint pain in her right shoulder might have been caused by bone alterations in addition to contracture and not by cartilage degeneration.

Conclusion

This case describes an elderly lady who had bilateral shoulder OA. The shoulder with joint pain showed bone alterations on MRI. While non-painful shoulder did not. These results suggest bone alterations as one of the factors in the disease progressions of painful shoulder OA.

References

1. Roach HI, Aigner T, Soder S, Haag J, Welkerling H. Pathobiology of osteoarthritis: pathomechanisms and potential therapeutic targets. Curr Drug Targets. 2007;8:271-282.
2. Scanzello CR, Goldring SR. The role of synovitis in osteoarthritis pathogenesis. Bone. 2012;51:249-257.
3. Guermazi A, Roemer FW, Haugen IK, Crema MD, Hayashi D. MRI-based semiquantitative scoring of joint pathology in osteoarthritis. Nat Rev Rheumatol. 2013;9:236-251.
4. Taljanovic MS, Graham AR, Benjamin JB, Gmitro AF, Krupinski EA, Schwartz SA, *et al*. Bone marrow edema pattern in advanced hip osteoarthritis: quantitative assessment with magnetic resonance imaging and correlation with clinical examination, radiographic findings, and histopathology. Skeletal Radiol. 2008;37:423-431.
5. Kamimura M, Nakamura Y, Uchiyama S, Ikegami S, Kato H. Joint pain undergoes a transition in accordance with signal changes of bones detected by MRI in hip osteoarthritis. Open Rheumatol J. 2013;7:67-74.
6. Kamimura M, Nakamura Y, Mukaiyama K, Ikegami S, Uchiyama S, Kato H. The pathophysiology of primary hip osteoarthritis may originate from bone alterations. Open Rheumatol J. 2013;7:101-107.
7. Van Schaardenburg D, Van den rande KJ, Ligthart GJ, Breedveld FC, Hazes JM. Musculoskeletal disorders and disability in persons aged 85 and over: a community survey. Ann Rheum Dis. 1994;53:807-811.
8. Oh JH, Chung SW, Oh CH, Kim SH, Park SJ, Kim KW, *et al*. The prevalence of shoulder osteoarthritis in the elderly Korean population: association with risk factors and function. J Shoulder Elbow Surg. 2011;20:756-763.
9. Muraki S, Oka H, Akune T, Mabuchi A, En-yo Y, Yoshida M, *et al*. Prevalence of radiographic knee osteoarthritis and its association with knee pain in the elderly of Japanese population-based cohorts: the ROAD study. Osteoarthritis Cartilage. 2009;17:1137-1143.
10. Sandell LJ. Etiology of osteoarthritis: genetics and synovial joint development. Nat Rev Rheumatol. 2012;8:77-89.
11. Clegg DO, Reda DJ, Harris CL, Klein MA, O'Dell JR, Hooper MM, *et al*. Glucosamine, chondroitin sulfate, and the two in combination for painful knee osteoarthritis. N Engl J Med. 2006;354:795-808.
12. Muraki S, Akune T, Oka H, Ishimoto Y, Nagata K, Yoshida M, *et al*. Incidence and risk factors for radiographic knee osteoarthritis and knee pain in Japanese men and women: a longitudinal population-based cohort study. Arthritis Rheum. 2012;64:1447-1156.
13. Millett PJ, Horan MP, Pennock AT, Rios D. Comprehensive Arthroscopic Management (CAM) procedure: clinical results of a joint-preserving arthroscopic treatment for young, active patients with advanced shoulder osteoarthritis. Arthroscopy. 2013;29:440-448.
14. Kaneko H, Ishijima M, Futami I, Tomikawa-Ichikawa N, Kosaki K, Sadatsuki R, *et al*. Synovial perlecan is required for osteophyte formation in knee osteoarthritis. Matrix Biol. 2013;32:178-187.