Histologic Analysis of Postmeniscectomy Osteonecrosis

Hiroshi Higuchi, MD, PhD, Yasukazu Kobayashi, MD, PhD, Atsushi Kobayashi, MD, Kazuhisa Hatayama, MD, PhD, and Masashi Kimura, MD, PhD

Abstract

Bone marrow signal changes on magnetic resonance imaging (MRI) after meniscectomy have been reported as evidence of postmeniscectomy osteonecrosis, but this pathology is unclear.

We conducted a study to follow-up cases with bone marrow signal changes on MRI after meniscectomy and investigate the pathology of underlying lesions. Of 136 patients with no presurgical evidence of osteonecrosis, 29 had juxta-articular bone marrow signal changes on MRI after arthroscopic meniscectomy and subsequently underwent conservative therapy. In 6 of these 29 patients, clinical symptoms and radiographic changes began deteriorating. Based on the Koshino classification, 4 of the 6 patients had Stage-2 knee osteonecrosis and 2 had Stage-3. Arthroscopic and pathologic examinations were performed. Arthroscopic findings were fibrillation (all 6 cases), fissuring (4), ulceration (2), and eburnation (2). Histologic analysis confirmed subchondral bone fractures in all 6 cases, but osteonecrotic lesions were detected only in 2 cases with obvious radiologic deterioration.

Postmeniscectomy osteonecrosis might result from subchondral bone fractures. Fracture healing is worse in patients with comorbidities than in those without it; comorbidities might be a risk factor for osteonecrosis.

In 1991, Brahme and colleagues[1] were the first to report bone marrow signal changes on magnetic resonance imaging (MRI) after meniscectomy as evidence of postmeniscectomy osteonecrosis. They reported 7 patients with knee pain and meniscal tear without initial evidence of osteonecrosis at MRI and underwent arthroscopic meniscectomy. These patients returned within 2 to 14 months with recurrent pain in the treated knee. MRI then demonstrated abnormalities consistent with osteonecrosis, and coined the term *postmeniscectomy osteonecrosis*. Similar cases have since been reported,[2,3] but the diagnoses were made solely with MRI findings. Moreover, the natural history and clinical course of these cases were unclear. In other words, we can question whether bone marrow signal changes on MRI after meniscectomy signify idiopathic osteonecrosis as described by Ahlbäck.[4]

In 2002, we conducted a prospective study to determine the incidence of postmeniscectomy osteonecrosis as well as the sites and the sizes of bone marrow changes on MRI after arthroscopic meniscectomy.[5] The femoral or tibial condyles of one-third of the patients in that study showed bone marrow signal changes on postoperative MRI. If these changes had reflected spontaneous osteonecrosis, as Brahme and colleagues[1] proposed, the knee osteonecrosis rate would have been higher. However, Santori and colleagues[2] reported that, out of more than 2000 knee arthroscopy cases at their hospital over 10 years, only 2 had early osteonecrosis documented. In addition, arthroscopic meniscectomy follow-up reports indicated a low rate of postoperative knee osteonecrosis over the long term.[6,7] These reports suggest that bone marrow signal changes after meniscectomy do not reflect spontaneous osteonecrosis of the knee. The question then arises: What are the pathologic correlates of bone marrow signal changes after arthroscopic meniscectomy?

In the present study, we followed-up our postmeniscectomy osteonecrosis cases with clinical features and investigated underlying lesions both arthroscopically and histopathologically. Twenty-nine patients with the bone marrow signal changes on MRI after arthroscopic meniscectomy were followed-up. Conservative therapy was effective in 23 of these patients and their knee pain resolved; knee pain remained in the other 6 patients. We performed a second knee surgery in these 6 patients and investigated histology.

Materials and Methods

Between October 2007 and May 2009, we assigned 136 patients (78 men, 58 women) to our study group. These patients had an initial diagnosis of isolated meniscal tear but no evidence of osteonecrosis on MRI or as a clinical feature. All arthroscopic surgery was performed by the same surgeon (MK) with the patient under epidural or general anesthesia and with use of anteromedial and anterolateral portals. All operations were performed with hand instruments, and without contact Nd:YAG lasers. After surgery,

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Table. **Patient Data**

Case	Sex	Age, y	Time Between 1st Meniscectomy & 2nd Operation, wk	Complications	Osteonecrosis Stage	Arthroscopic Findings	Pathologic Findings
1	F	62	30	HT, DM	3	FIB, FIS, EBU	FR, NE
2	M	70	24	HT, HL	2	FIB, FIS	FR
3	F	59	35	HT, HL	2	FIB, FIS	FR
4	F	67	21	HT, HL	2	FIB, FIS	FR
5	M	64	28	HT, DM	3	FIB, ULC, EBU	FR, NE
6	F	72	30	HL	2	FIB, ULC	FR

Abbreviations: DM, diabetes mellitus; EBU, eburnation; FIB, fibrillation; FIS, fissuring; FR, subchondral bone fracture; HL, hyperlipidemia; HT, hypertension; NE, osteonecrotic lesions; ULC, ulceration.

weight-bearing and full range of motion were allowed as tolerated. Further rehabilitation consisted of muscle strength training and bicycling.

One to 4 months after arthroscopic meniscectomy, some patients reported recurrent sharp knee pain on daily activity or on performing the rehabilitation protocol. These patients underwent conservative therapy including weight-bearing control, muscle strength reeducation, and activity level reduction; radiography and MRI were used to determine the reasons for the recurrent pain.

Figure 1. Radiographs show oval radiolucent shadow in subchondral area of weight-bearing portion of femoral condyle with increasing osteosclerosis in cortex distal area.

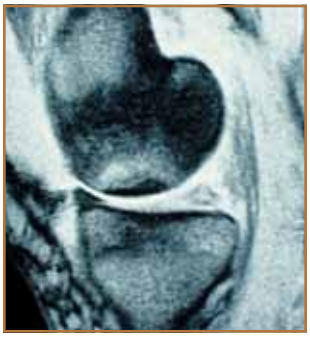

Figure 2. T_2-weighted sagittal magnetic resonance imaging after meniscectomy shows low and high signal intensity areas in subchondral lesion of medial femoral condyle.

Figure 3. Photomicrograph of tissue beneath articular cartilage shows fractured bone trabeculae and vascular granulation tissue but no evidence of osteonecrosis.

In 29 patients, MRI showed the bone marrow signal changes that, according to some authors,[1-3] reflect so-called postmeniscectomy osteonecrosis. Conservative therapy was effective in 23 of these patients and their knee pain resolved. The other 6 patients (2 men, 4 women; mean age, 66 years; age range, 59-72 years) still had severe pain after 5 to 8 months.

We used the Koshino radiography-based classification[8] of knee osteonecrosis to characterize these 6 cases by developmental stage. In Stage-1 (incipient stage), the patient reported pain in the medial side of the knee, but the radiograph shows no abnormalities. In Stage-2 (avascular stage), an oval radiolucent shadow appears in the subchondral area of the weight-bearing portion of the femoral condyle, and osteosclerosis increases in the cortex distal to the radiolucent area. By Stage-3 (collapsed stage), the radiolucent area is expanded, a sclerotic halo surrounds it, and the subchondral bone plate distal to the radiolucency is collapsed and is visible as a calcified plate. In Stage-4 (degenerative stage), osteophytes and osteosclerosis are visible in the ipsilateral tibial condyle and in the involved femoral condyle.

Of the 6 patients, 4 had Stage-2 osteonecrosis and 2 patients, Stage-3. All 6 patients had general disease complications. Regarding past history, 5 patients had hypertension, 4 had hyperlipidemia, and 2 had diabetes mellitus (**Table**). Three of the cases were managed with high tibial osteotomy, and 3 cases were managed with unilateral knee arthroplasty, all as second-stage surgical procedures. Arthroscopic examinations were performed to obtain macrohistological information regarding areas of signal change. In all cases, the biopsy technique was used, or harvesting was done during surgery, to obtain the samples needed for pathologic analysis for osteonecrotic lesions.

Results

The Table lists 4 arthroscopic findings for areas of bone marrow signal changes on MRI: fibrillation (6 cases), fissuring (4), ulceration (2), and eburnation (2). There were no cases of subchondral bone dropping from marrow, as occurs in severe cases of osteonecrosis. Thus, osteoarthritic changes caused the majority of the arthroscopic findings.

The pathologic study revealed vascular granulation around subchondral bone in all cases and trabecular bone formation between subchondral bone and bone marrow in 5 cases. However, osteonecrotic lesions were detected in only 2 cases, both of which were Stage-3 (**Table**).

In one case, a 67-year-old woman had hypertension and hyperlipidemia. Severe knee pain and joint swelling recurred suddenly 3 months after arthroscopic meniscectomy and it became impossible for her to walk. Conservative therapy (weight-bear-

ing control) did not resolve the pain, and radiographs showed a Stage-2 osteonecrotic lesion in the medial femoral condyle 5 months after surgery (**Figure 1**). T_2-weighted MRI showed an area of diffuse low signal intensity in the medial femoral condyle, where radiographic change had been observed (**Figure 2**). As conservative therapy was not effective, high tibial osteotomy was used for the second treatment. Arthroscopy at time of second surgery revealed a high degree of fibrillation and fissuring of cartilage, correlating with signal change on MRI. Fractured bone trabeculae and vascular granulation tissue beneath the subchondral bone were also recognized on pathology, but osteonecrosis was not apparent (**Figure 3**).

Discussion

One-third of the patients in the study by Kobayashi and colleagues[5] showed bone marrow signal changes in femoral or tibial condyles on postoperative MRI after arthroscopic partial meniscectomy. Moreover, the authors reported that age, sex, and articular cartilage condition at meniscectomy were not risk factors for these juxta-articular bone marrow changes, and they mentioned that the sizes and the pattern of these MRI changes varied. If these changes had reflected spontaneous osteonecrosis, as Brahme and colleagues[1] proposed, the knee osteonecrosis rate would have been higher. However, the incidence of knee osteonecrosis after meniscectomy is very rare, as Santori and colleagues[2] found. In addition, reports of follow-up of arthroscopic meniscectomy have indicated a low rate of postoperative knee osteonecrosis over the long term.[6,7] Our results showed that most bone marrow signal changes after meniscectomy are reversible and that clinical symptoms are transient. It may be that the cases that do not respond to conservative treatment are a different entity from osteonecrosis and thus have a worse prognosis.

Nakamura and colleagues[9] reported a case of subchondral bone microfracture of the knee without osteonecrosis after arthroscopic meniscectomy and concluded that histologic analysis of osteonecrosis by radiography and MRI is incorrect. Yamamoto and Bullough[10] described the histology of spontaneous osteonecrosis of the knee and concluded that subchondral insufficiency fractures lead to early osteonecrosis. This hypothesis for the etiology of osteonecrosis might help explain the incidence of juxta-articular bone marrow signal changes after arthroscopic meniscectomy. Meniscectomy, which causes increased contact pressure at operated joint surfaces,[11,12] can lead to subchondral microfracture, and MRI detects this reaction. This finding would explain why most juxta-articular bone marrow signal changes on MRI after meniscectomy are reversible and why clinical symptoms are transient.

On the other hand, fracture healing is thought to be worse in patients with comorbidities (eg, hyperlipidemia, hypertension, diabetes mellitus) than in patients without it, and therefore background disease might be a risk factor for osteonecrosis.[13-16] Indeed, the results of the present study showed that postmeniscectomy osteonecrosis cases, which resist conservative therapy, including weight-bearing control, were complicated by general disease. As older patients with other ailments may develop osteonecrosis after meniscectomy, surgeons should consider this phenomenon.

Postmeniscectomy osteonecrosis might result from subchondral bone fractures. These insufficiency fractures with osteoarthritic change do not always lead to osteonecrosis. However, surgeons should be aware that the healing of subchondral bone fractures is worse in patients with background disease than in those without it.

Dr. Higuchi is Director, and Dr. A. Kobayashi is Orthopedic Doctor, Department of Orthopaedic Surgery, Asakura Sports Rehabilitation Clinic, Maebashi, Gunma, Japan. Dr. Y. Kobayashi is Senior Director, and Dr. Kimura is Director, Gunma Sports Medicine Research Center, Zenshukai Hospital, Maebashi, Gunma, Japan. Dr. Hatayama is Orthopedic Doctor, Department of Orthopaedic Surgery, Gunma University Faculty of Medicine, Maebashi, Gunma, Japan.

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Address correspondence to: Hiroshi Higuchi, MD, PhD, Department of Orthopaedic Surgery, Asakura Sports Rehabilitation Clinic, 249-1 Asakura, Maebashi, Gunma, 371-0811 Japan (tel, 81-27265-6522; fax, 81-27265-6527; e-mail, jaguar156z@yahoo.co.jp).

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