Spontaneous Osteonecrosis of Knee After Arthroscopy Is Not Necessarily Related to the Procedure

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Abstract

Reports in the literature have suggested a causal relationship between knee arthroscopy and spontaneous osteonecrosis of the knee (SONK).

We conducted a study to determine if there are imaging characteristics associated with SONK and if there is a relationship between arthroscopy and SONK. In this retrospective review, we compared preoperative and postoperative findings in 11 patients (12 joints) who developed SONK after arthroscopy with findings in 11 age- and sex-matched controls who did not develop SONK after arthroscopy.

There were no significant preoperative radiologic differences between the SONK and control groups. All 12 SONK lesions seen on magnetic resonance imaging were in the medial femoral condyle. Six SONK knees developed the lesion after arthroscopy, and 6 had SONK lesions before arthroscopy. Eleven of the 12 SONK knees had a medial meniscal tear, compared with 8 medial meniscal tears and 3 lateral meniscal tears in the control group. Eight SONK knees and 5 control patients had medial meniscal extrusion of more than 3 mm.

A causal relationship between knee arthroscopy and SONK is questionable.

The term *spontaneous osteonecrosis of the knee* was first used by Ahlbäck[1] in 1968. This term, and the acronym *SONK* (sometimes *SPONK*[2]), has subsequently been used by other authors to refer to an apparent osteonecrosis of the knee, most commonly occurring within the medial femoral condyle. SONK typically occurs in older women who usually do not have the typical osteonecrosis risk factors, such as steroid use, sickle-cell anemia, and excessive alcohol intake. Furthermore, the radiologic appearance of SONK differs from the typical avascular necrosis findings seen with radiography and magnetic resonance imaging (MRI). In particular, on MRI, the abnormality of SONK does not have the typical serpiginous margin of bone infarction, or the double-line sign indicating both sclerosis and granulation tissue.[3] SONK is normally seen as a line of signal intensity on T1- and T2-weighted sequences; this line is adjacent to or parallels the subchondral bone with an adjacent area of extensive edema.

There is dispute over the cause of SONK. Yamamoto and Bullough[4] proposed the lesion is in part a subchondral insufficiency fracture and staged it into 4 parts. Histologic findings suggest at least some SONK lesions are subchondral insufficiency fractures.[5] Brahme and colleagues[6] were the first to describe SONK occurring after arthroscopy, and others have documented this finding. The condition has also been referred to as *osteonecrosis in the postoperative knee*.[7-13] An association of postoperative SONK with cartilage loss and meniscal tear has been proposed.[7-13]

We reviewed the clinical, radiologic, and MRI findings in 11 patients with evidence of postarthroscopy SONK to try to identify any risk factors that might predispose them to poor outcomes. Our study population consisted of 11 patients (12 knees) with SONK; 6 of the knees had the lesion before knee arthroscopy, and the other 6 developed the lesion after arthroscopy. We also considered MRI findings in a group of 11 age- and sex-matched patients who underwent knee arthroscopy and did not have or develop SONK. We reviewed the preoperative MRI findings of both groups for meniscal tear, meniscal extrusion, and cartilage loss. We had 2 hypotheses. First, patients with preoperative MRI findings of SONK would have articular cartilage changes, posterior root degeneration, and meniscal extrusion similar to those of patients who developed SONK after arthroscopy. Second, an age- and sex-matched group of patients who underwent arthroscopy and did not develop SONK would be similar in articular cartilage changes, posterior root degeneration or tear, and meniscal extrusion.

Materials and Methods

With institutional review board approval and waived informed consent, we reviewed all imaging studies, particularly the radiographs and MRI studies, of 11 patients (12 knees) who either had SONK before arthroscopy or developed it after ar-

Authors' Disclosure Statement: The authors report no actual or potential conflict of interest in relation to this article.

throscopy. In all these cases, arthroscopy was performed to alleviate mechanical symptoms associated with meniscal tear.

On subsequent review by a musculoskeletal radiologist, 6 patients with SONK had an identifiable lesion before surgery. All patients' symptoms had not improved with an earlier trial of conservative management. All preoperative and postoperative radiologic and MRI findings were reviewed. The patient group was assembled by writing to all the orthopedic surgeons who performed arthroscopy at our institution and asking for SONK cases seen in their practices. All but 2 cases were performed by a surgeon who treated a predominantly older, less active population. Clinical notes were reviewed for outcomes, and the musculoskeletal radiologist reviewed all radiologic studies. The 4 men and 7 women in the SONK group (1 woman had bilateral knee lesions) ranged in age from 43 to 74 years (mean, 63.8 years), and the 4 men and 7 women in the control group were age-matched to 43 to 75 years (mean, 63.6 years). The controls were chosen from a pool of patients who underwent knee arthroscopy at our institution.

MRI was performed using General Electric 1-T, 1.5-T, or 3-T magnets (GE Healthcare, Milwaukee, Wisconsin) or using Philips 1.5-T or open 0.7-T magnets (Philips Healthcare, Andover, Massachusetts). Imaging included sagittal and coronal proton density–weighted sequences and coronal and axial fat-suppressed T2-weighted sequences. SONK was diagnosed when a low signal line adjacent to the subchondral bone plate on the femoral or tibial condyles was present with an adjacent area of bone marrow edema in the respective condyle or when there was depression of the subchondral bone plate with adjacent edema. The MRI studies were reviewed for lesion location, and medial meniscus and lateral meniscus were reviewed for tear. Type of meniscal tear (horizontal cleavage, radial, complex degenerative) was documented, as was meniscal extrusion. The meniscus was regarded as extruded if the body extended more than 3 mm from the joint margin. Cartilage in the medial and lateral compartment was reviewed according to a modified Noyes scale listing 0 as normal, 1 as internal changes only, 2A as 1% to 49% cartilage loss, 2B as 50% to 90% loss of articular cartilage, 3A as 100% articular cartilage loss with subchondral bone plate intact, and 3B as 100% articular cartilage loss with ulcerated subchondral bone plate.[14] Osteoarthritic severity was similarly classified using the Kellgren-Lawrence scale,[15] where grade 0 is normal; grade 1 is unlikely to have narrowing of the joint space but potentially has osteophytic lipping; grade 2 has both definite narrowing of the joint space and osteophytes; grade 3 has narrowing of the joint space and multiple osteophytes, some sclerosis, and possible deformity of bone contour; and grade 4 has marked narrowing of the joint space, large osteophytes, severe sclerosis, and definite deformity of bone contour. Follow-up clinical notes and radiologic studies were reviewed in the assessment of patient outcomes.

All statistical analyses were performed with SAS 9.2 software (SAS Institute, Cary, North Carolina). Age data were evaluated with the Shapiro-Wilk test and graphical displays and were found to violate normality assumptions, so they are presented as medians and ranges; other variables are presented as count and column percentages. The Wilcoxon rank sum test was used to compare the 2 groups' age distributions. Fisher exact tests were used to compare proportions between the 2 groups for the other variables. Statistical significance was set at $P < .05$.

Results

Table 1 lists the demographics and imaging characteristics of the 11 patients—6 had SONK before arthroscopy and 6 developed it after arthroscopy. Comparison of the 11 patients with SONK and the 11 controls is summarized with P values in **Table 2**. Representative cases that either presented before surgery or developed after surgery are shown in **Figures 1 to 4**. There were 6 prearthroscopy lesions and 6 postarthroscopy lesions—all 12 in the medial femoral condyle. Eleven of the 12 knees had a medial meniscal tear, and 1 knee had both medial and lateral meniscal tears. In 8 of the 12 knees, the lateral meniscus was normal; in 2 knees, it had mild degeneration; and, in 1 knee, it had a complex tear. Assessment of hyaline cartilage revealed medial cartilage loss ranging from 2A to 3B (median, 2B) in the patients with SONK, and lateral cartilage loss ranging from 0 to 2A (median, 0). At surgery, all knees had a partial medial meniscectomy, and 6 had a partial lateral meniscectomy. Ten of the 12 knees had chondroplasty, 9 patellar and 5 of the medial femoral condyle. Only 4 of the 11 patients with follow-up of more than 1 year went on to joint replacement. Six of the 12 had follow-up of more than 2 years. Of the 6 patients without an identifiable SONK lesion on MRI before arthroscopy, 4 had mild to moderate knee pain 0.5, 2.4, 3.5, and 4 years after surgery. For the other 2 patients, knee replacement was performed 1.5 and 1.8 years after surgery. Of the 6 patients with prearthroscopy SONK, 4 had mild to moderate knee pain 1.5, 3.7, 6.5, and 6.8 years after surgery; the other 2 had knee replacement 0.5 and 1.8 years after surgery. Articular cartilage degeneration and meniscal extrusion were similar (**Table 1**). In the control

Table 1. **Comparison of Patients With Spontaneous Osteonecrosis of the Knee (SONK) Before Arthroscopy and Patients Who Developed SONK After Arthroscopy**

	SONK Before Arthroscopy (n = 6 knees)	SONK After Arthroscopy (n = 6 knees)
Age, y	46-71 (median, 69)	43-74 (median, 69)
Sex	2 men, 4 women	2 men, 4 women
Side	3 right, 3 left	4 right, 2 left
Medial meniscus tear	All 6 posterior	5 posterior
Articular cartilage grade	2A (3), 2B (1), 3A (1), 3B (1)	2A (5), 2B (1)
Extrusion	5	4

group, there was only 1 knee replacement, at 3 years, and the other 11 were functioning 2.6 to 5 years later. The longer follow-up resulted from selection of appropriate controls from the same year. Of the 6 SONK lesions found on preoperative MRI, 3 were read by the interpreting radiologist before surgery as possible SONK lesions, 2 were read as insufficiency fractures, and 1 was read as a possible insufficiency fracture.

Discussion

SONK is well described as a complication of arthroscopic knee surgery. However, this condition more commonly appears spontaneously in a population that has not had surgery. It has become clear that the term *SONK* may be misleading.[16] In a recent series of postoperative subchondral fractures reported by MacDessi and colleagues,[5] the average age of patients included in their study was 64 years. Pathologic analysis revealed subchondral fracture with callus formation in all cases. Only 2 knees had evidence of osteonecrosis, which appeared to be secondary to the fracture. Based on these findings, the authors concluded that "further investigation into the etiology of this condition is warranted." A prominent association with medial meniscal tear has been noted, with the medial femoral condyle predominantly affected. As already mentioned, SONK differs from classical avascular necrosis on several points, including lack of the typical avascular osteonecrosis risk factors and absence of the serpiginous margin and double-line sign seen with typical bone infarction. In addition, the SONK lesions seen on radiographs and MRIs of the knee typically are in the medial femoral condyle and are very different from the typical area of infarction seen in patients with known risk factors for secondary osteonecrosis.

The cause of SONK is not known. Of more importance from a medicolegal standpoint is that these lesions are not necessarily related to arthroscopy.[17] Interestingly, Pape and colleagues[17] noted that some of the lesions they studied may have been present before surgery, which is what we found in 6 (50%) of the SONK knees in our study. Our data thus support the proposition that some SONK lesions are present before arthroscopy, and some cases of so-called postarthroscopy SONK may in fact have been progressing before surgery.

Our data also reinforce the importance of radiologist–orthopedic surgeon communication regarding the presence of SONK. We emphasize the importance of communicating the MRI findings clearly, whether the lesion is called *SONK*, *SPONK*, or *insufficiency fracture*. The orthopedic surgeons in our series may have been unaware of the presence of these lesions before arthroscopic meniscectomy, given the wide variety of terms being used in radiologic reports.

The natural history of spontaneous osteonecrosis of the medial tibial plateau has also been studied.[18] There were 3 outcome patterns—acute extensive collapse of the medial tibial plateau, rapid progression to varying degrees of osteoarthritis, and complete resolution. It has been shown that resolution of SONK can occur in the early stages of the disease, within several months, but often the changes progress

Table 2. Univariate Test Results: Comparison of Arthroscopy Controls and Arthroscopy Patients With Spontaneous Osteonecrosis of the Knee (SONK)

	Median (Range) or N (%)		
	Controls (n = 11)	SONK (n = 11)	*P*
Age, y	67.0 (32.0)	66.0 (31.0)	>.99
Sex			>.99
Female	7 (63.6%)	8 (66.7%)	
Male	4 (36.4%)	4 (33.3%)	
Side			>.99
Left	4 (40.0%)	5 (62.5%)	
Right	6 (60.0%)	7 (58.3%)	
Medial meniscus tear			.3168
No	3 (27.3%)	1 (8.3%)	
Yes	8 (72.7%)	11 (91.7%)	
Lateral meniscus tear			.3168
No	8 (72.7%)	11 (91.7%)	
Yes	3 (27.3%)	1 (8.3%)	
Extrusion			.4136
Medial	5 (45.5%)	8 (66.7%)	
None	6 (54.6%)	4 (33.3%)	
Kellgren-Lawrence			.4261
0	3 (27.3%)	1 (8.3%)	
1	1 (9.1%)	3 (25.0%)	
2	0	2 (16.7%)	
3	4 (36.4%)	2 (16.7%)	
4	3 (27.3%)	3 (25.0%)	
5	0	1 (8.3%)	
Medial cartilage			.2292
0	2 (18.2%)	0	
2A	1 (9.1%)	4 (33.3%)	
2B	5 (45.5%)	6 (50.0%)	
3A	3 (27.3%)	1 (8.3%)	
3B	0	1 (8.3%)	
Lateral cartilage			.1346
0	5 (45.5%)	10 (83.3%)	
1	1 (9.1%)	1 (8.3%)	
2A	1 (9.1%)	1 (8.3%)	
2B	1 (9.1%)	0	
3A	3 (27.3%)	0	

to bone destruction and articular cartilage collapse.[19]

In our series of patients, there was a female predominance, and mean age was 64 years. We investigated cartilage loss, meniscal tear, and meniscal extrusion to see if we could predict outcomes in patients who had the lesion before arthroscopy and if we could predict who might be at risk for developing the lesion after arthroscopy. Type of surgical procedure was also reviewed. For the sake of simplicity, we divided the follow-up patients into 2 groups: those managed with conservative treatment, which we deemed a reasonable outcome, and those who subsequently required knee joint replacement, which we deemed a poor outcome. As seen from our representative cases, both groups had patients with cartilage loss, meniscal tear, and meniscal extrusion to varying degrees. There were no risk factors pointing to a reasonable or poor outcome. In the group of patients with prearthroscopy lesions, we found the same problem. We were unable to identify a risk factor that might suggest a poor rather than a reasonable outcome. We must also emphasize that, in our review of patient charts, we could find no other causes for osteonecrosis. In particular, arthroscopic causes of acute chondral loss (eg, thermal wash, laser, bupivacaine pain pumps, epinephrine in irrigant) were not identified.

This study consisted of a series of cases managed at our institution over the past 8 years. Our data and this study had several limitations:

- We may have been unable to identify other SONK cases that belonged in the group from our institution. In addition, we had only 11 patients for comparison with patients without SONK. Likewise, there were only 6 knees each in the prearthroscopy and postarthroscopy SONK groups. We also used images obtained from 1-T, 1.5-T, and 3-T closed MRI devices and one 0.7-T open device. These were, however, at the same institution.

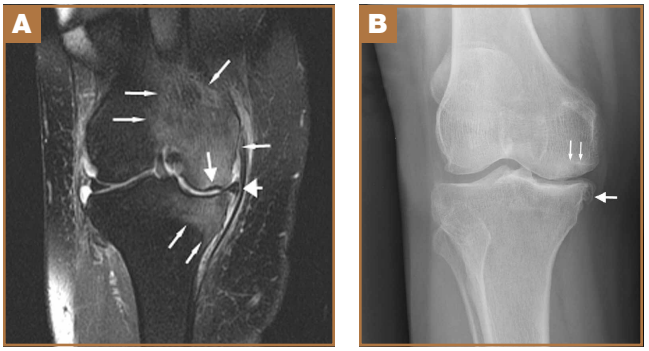

Figure 1. Patient: 68-year-old woman. (A) Fifteen days before arthroscopy, coronal fat-suppressed T2-weighted magnetic resonance imaging (repetition time [TR] 3000 ms, echo time [TE] 56 ms, echo train length 4) of right knee shows extrusion of medial meniscus (short arrow) secondary to radial tear in posterior horn. Note fracture of subchondral bone plate of medial femoral condyle (large arrow), extensive edema of medial femoral condyle (thin arrows), and some edema of medial tibial plateau (thin arrows). Appearance is consistent with preoperative spontaneous osteonecrosis of knee. (B) Four years 9 months (1756 days) after arthroscopy, coned-down standing anteroposterior radiograph shows small sclerotic area in medial femoral condyle (thin arrows). Note mild narrowing of medial joint space and marginal spurring of medial tibial plateau (large arrow). Outcome was reasonably good, with no total knee arthroplasty more than 6 years after initial arthroscopy.

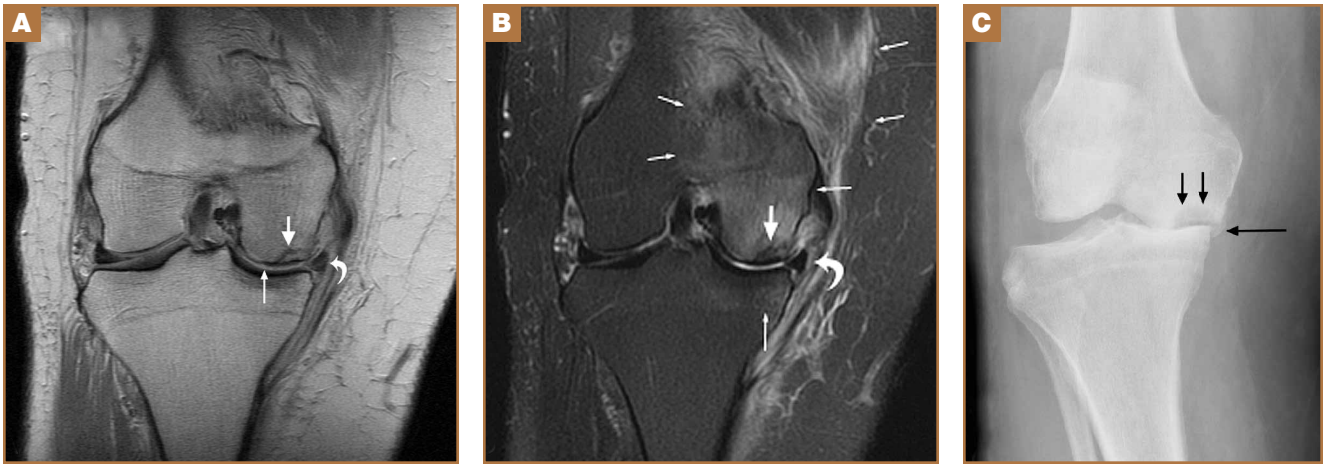

Figure 2. Patient: 64-year-old woman. (A) Thirty-two days before arthroscopy, coronal proton density–weighted magnetic resonance imaging (MRI) (repetition time [TR] 3000 ms, echo time [TE] 12.416 ms, echo train length 6) of right knee shows spontaneous osteonecrosis of knee (SONK) lesion with low signal line adjacent to weight-bearing surface of medial femoral condyle (thick arrow). Note some loss of cartilage on medial femoral condyle (thin arrow) and prominent extrusion of medial meniscus (curved arrow) secondary to radial tear in posterior horn of meniscus. Appearances are consistent with preoperative SONK. (B) Coronal fat-suppressed T2-weighted MRI (TR 3250 ms, TE 87.04 ms, echo train length 12) shows low signal line (thick arrow) and extensive edema in medial femoral condyle and vastus medialis (thin arrows) and minimal edema in medial tibial plateau (thin arrow). Note extrusion of medial meniscus (curved arrow). (C) Three months (97 days) after arthroscopy, coned-down standing anteroposterior radiograph shows complete loss of joint space in right medial compartment (long arrow) and subchondral collapse of medial femoral condyle in area of SONK shown on MRI (short arrows). Note tibia in mild varus as well as mild lateral subluxation of right tibia on femur. Outcome was poor, with total knee arthroplasty required 5.5 months after arthroscopy.

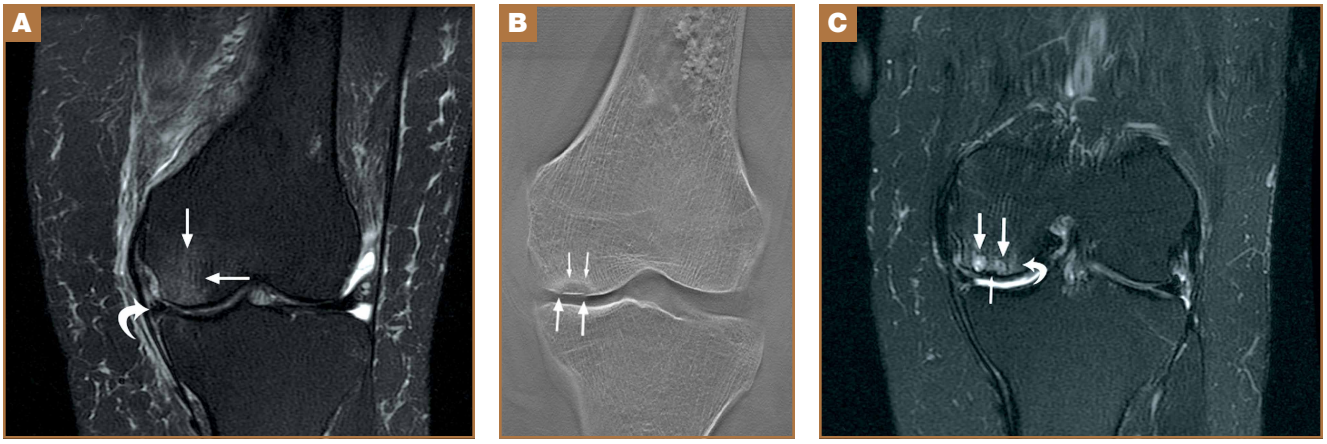

Figure 3. Patient: 64-year-old woman. (A) Seventy days before arthroscopy, coronal fat-suppressed T2-weighted magnetic resonance imaging (MRI) (repetition time [TR] 5000 ms, echo time [TE] 82.94 ms, echo train length 10) of left knee shows mild edema in medial femoral condyle (straight arrows) with no evidence of spontaneous osteonecrosis of knee (SONK). Note mild extrusion of medial meniscus (curved arrow) secondary to diffuse degeneration and tear of posterior horn and body. (B) Six months (181 days) after arthroscopy, dedicated coronal tomosynthesis section shows SONK lesion on weight-bearing surface of medial femoral condyle. Note fractured subchondral bone plate (thick arrows) and marginal sclerosis of lesion (thin arrows). (C) Ten months (321 days) after surgery, coronal fat-suppressed T2-weighted MRI (TR 4500 ms, TE 81.98 ms, echo train length 10) shows minimal edema in medial femoral condyle (curved arrow), minimal irregularity of subchondral bone plate (thin arrow), and suggestion of 2 small geodes (thick arrows). Although SONK developed after surgery, outcome was reasonable, with no total knee arthroplasty almost 2.5 years after arthroscopy.

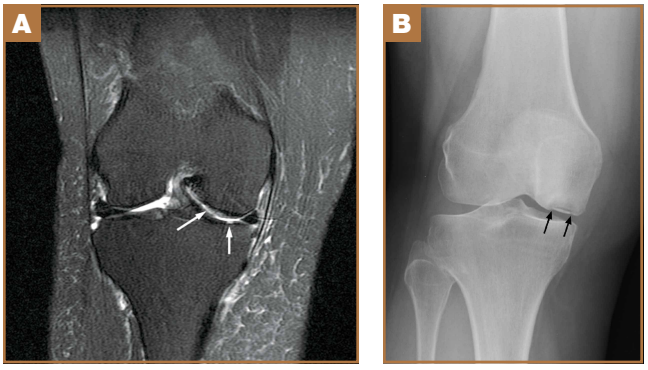

Figure 4. Patient: 68-year-old woman. (A) Twenty-five days before arthroscopy, coronal fat-suppressed T2-weighted magnetic resonance imaging (repetition time [TR] 4000 ms, echo time [TE] 87.68 ms, echo train length 12) of right knee shows no evidence of spontaneous osteonecrosis of knee (SONK). Note irregularity of hyaline cartilage (arrows). (B) One year 1 month (404 days) after arthroscopy, coned-down standing anteroposterior radiograph shows SONK lesion on weight-bearing surface of medial femoral condyle with fracture through subchondral bone plate (arrows). Three months (111 days) after arthroscopy, radiographs (not illustrated) showed a knee effusion with clear narrowing of medial joint space but no evidence of SONK. Outcome was poor, with unicompartmental knee arthroplasty required 1 year 10 months after initial arthroscopy.

- Timing of our imaging was not uniform. In particular, in 3 of the patients who developed SONK after arthroscopy, preoperative MRI studies were performed quite some time before surgery. However, in these patients, more recent preoperative radiographs did not show any evidence of lesions. It can also be seen that postarthroscopy follow-up of patients varied. It is possible that, on longer follow-up, some of the cases we classified as having a reasonable outcome may have gone on to require total knee arthroplasty. One could argue that, in the patient who developed SONK within 1 year after surgery (**Figure 4**), the lesion was not related to the surgery. However, this patient's radiographs 3 months after surgery did not show the SONK lesion but clearly showed prominent medial joint space narrowing—a new finding.
- Only 1 musculoskeletal radiologist evaluated the radiographs, MRIs, and tomosynthesis (similar to computed tomography) studies for this investigation.
- This lesion is not common, thus giving us a small group to analyze.

Despite our data limitations and the retrospective nature of this study, we compiled a reasonably representative sample of surgical SONK patients that matches other samples reported in the literature. Unfortunately, we could not identify any risk factors pointing to the likelihood of developing SONK or any risk factors pointing to either a reasonable or a poor prognosis in these patients. The etiology of the lesion remains an enigma. Our finding 6 cases of prearthroscopy lesions that did not necessarily result in a poor outcome, combined with our inability to identify any risk factors for SONK, points to the lack of a causal relationship with arthroscopy.

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Acknowledgments: The authors thank Meredith Mahan, MS (Division of Biostatistics, Department of Public Health Sciences, Henry Ford Hospital, Detroit, Michigan) for statistical selection and analysis.

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Am J Orthop. 2015;44(6):E184-E189.

References

1. Ahlbäck S. Osteoarthritis of the knee. A radiographic investigation. *Acta Radiol Diagn*. 1968;(suppl 277):7-72.
2. Juréus J, Lindstrand A, Geijer M, Robertsson O, Tägil M. The natural course of spontaneous osteonecrosis of the knee (SPONK): a 1- to 27-year follow-up of 40 patients. *Acta Orthop*. 2013;84(4):410-414.
3. Zurlo JV. The double-line sign. *Radiology*. 1999;212(2):541-542.
4. Yamamoto T, Bullough PG. Spontaneous osteonecrosis of the knee: the result of subchondral insufficiency fracture. *J Bone Joint Surg Am*. 2000;82(6):858-866.
5. MacDessi SJ, Brophy RH, Bullough PG, Windsor RE, Sculco TP. Subchondral fracture following arthroscopic knee surgery. A series of eight cases. *J Bone Joint Surg Am*. 2008;90(5):1007-1012.
6. Brahme SK, Fox JM, Ferkel RD, Friedman MJ, Flannigan BD, Resnick DL. Osteonecrosis of the knee after arthroscopic surgery: diagnosis with MR imaging. *Radiology*. 1991;178(3):851-853.
7. Faletti C, Robba T, de Petro P. Postmeniscectomy osteonecrosis. *Arthroscopy*. 2002;18(1):91-94.
8. Johnson TC, Evans JA, Gilley JA, DeLee JC. Osteonecrosis of the knee after arthroscopic surgery for meniscal tears and chondral lesions. *Arthroscopy*. 2000;16(3):254-261.
9. al-Kaar M, Garcia J, Fritschy D, Bonvin JC. Aseptic osteonecrosis of the femoral condyle after meniscectomy by the arthroscopic approach. *J Radiol*. 1997;78(4):283-288.
10. DeFalco RA, Ricci AR, Balduini FC. Osteonecrosis of the knee after arthroscopic meniscectomy and chondroplasty: a case report and literature review. *Am J Sports Med*. 2003;31(6):1013-1016.
11. Kusayama T. Idiopathic osteonecrosis of the femoral condyle after meniscectomy. *Tokai J Exp Clin Med*. 2003;28(4):145-150.
12. Prues-Latour V, Bonvin JC, Fritschy D. Nine cases of osteonecrosis in elderly patients following arthroscopic meniscectomy. *Knee Surg Sports Traumatol Arthrosc*. 1998;6(3):142-147.
13. Santori N, Condello V, Adriani E, Mariani PP. Osteonecrosis after arthroscopic medial meniscectomy. *Arthroscopy*. 1995;11(2):220-224.
14. Noyes FR, Stabler CL. A system for grading articular cartilage lesions at arthroscopy. *Am J Sports Med*. 1989;17(4):505-513.
15. Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. *Ann Rheum Dis*. 1957;16(4):494-502.
16. Kidwai AS, Hemphill SD, Griffiths HJ. Radiologic case study. Spontaneous osteonecrosis of the knee reclassified as insufficiency fracture. *Orthopedics*. 2005;28(3):236, 333-236.
17. Pape D, Lorbach O, Anagnostakos K, Kohn D. Osteonecrosis in the post-arthroscopic knee. *Orthopade*. 2008;37(11):1099-1107.
18. Satku K, Kumar VP, Chacha PB. Stress fractures around the knee in elderly patients. A cause of acute pain in the knee. *J Bone Joint Surg Am*. 1990;72(6):918-922.
19. Soucacos PN, Xenakis TH, Beris AE, Soucacos PK, Georgoulis A. Idiopathic osteonecrosis of the medial femoral condyle. Classification and treatment. *Clin Orthop*. 1997;(341):82-89.

This paper will be judged for the Resident Writer's Award.