Focal Spontaneous Osteonecrosis and Medial Meniscus Tear: Two Cases and a Literature Review

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Spontaneous or idiopathic osteonecrosis of the medial femoral condyle is a well-recognized cause of acute pain, tenderness over the joint line, effusion, and synovitis. Since the disease was first described by Ahlback and colleagues[1] in 1968, it has been the subject of several studies. Results from history, examination, and clinical studies suggest a complicated problem involving biomechanical and blood supply changes that tend to increase the susceptibility of the medial femoral condyle to avascular necrosis in the presence of meniscal pathology.[2-9] It remains unclear if the focal type of osteonecrosis arises from the insult of the arthroscopy or from an underappreciation of the disease in its early stages.

There are 2 types of avascular necrosis in the skeleton. The first, which occurs more commonly in patients aged 50 years or older, is *focal spontaneous osteonecrosis* (SO); the second, which tends to occur in patients aged 30 to 50, is *secondary osteonecrosis* (2nd ON). The clinical presentations of these types of osteonecrosis must be distinguished when diagnosing and treating patients. SO is associated with tenderness over the joint line, unilateral knee involvement, and acute pain exacerbated by increased weight-bearing.[4,8,10,11] With SO, most patients can remember the exact moment of symptom onset, and they describe pain increasing at night or at rest.[1,4,8,10-12] In contrast, 2nd ON generally shows bilateral involvement of multiple joints with gradual onset of diffuse pain.[8] Secondary osteonecrosis is commonly associated with such predisposing factors as corticosteroid therapy, alcoholism, and systemic lupus erythematosus.[2,5,8,13-15]

In patients with knee pain, early diagnosis requires a high index of suspicion for SO, as the diagnosis may not become apparent until months or years after symptom onset. Radiography, magnetic resonance imaging (MRI), and radionuclide bone scanning have proved capable of distinguishing SO from 2nd ON.[8] MR and bone scan images are commonly used to identify the disease in its early stages, and plain films are commonly used to rule out advanced disease when disease duration is not clear. Radionuclide techniques tend to confirm all 4 osteonecrosis stages.[8,10,11,16] Without early detection, SO treatment can become complicated by the treatment required for the apparent overlying pathologies (eg, degenerative medial meniscal tears).

When patients aged 50 years or older present with acute onset of medially-based knee pain, the most common differential diagnosis includes SO, medial compartment osteoarthritis, and degenerative medial meniscus pathology. Here we describe 2 cases of SO diagnosed and treated initially as degenerative medial meniscus tears, and we illustrate the clinical diagnostic features of the disease.

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Case Reports

Case 1

A woman in her early 50s presented to her local orthopedist with left knee pain and swelling that had progressively worsened over 3 weeks. The patient noted that the pain was exacerbated when walking and standing and partly relieved at rest. She also complained of sleeping problems, with an occasional increase in pain at night. The patient was 5 feet 6 inches tall, weighed 185 pounds, and was a nonsmoker of general good health, except for hypercholesterolemia. She had a completely benign medical history in regard to the knee, which to our knowledge included no prior injuries and no corticosteroid steroid treatments.

Physical examination revealed tenderness about the medial joint line but not about the medial distal femoral condyle. Range of motion was slightly restricted in flexion. The patient had mild patellofemoral crepitus, a small effusion, and diffuse synovitis. The collateral and cruciate ligaments were intact, and the hip and neurovascular examinations were normal. She had a positive McMurray sign.

At presentation, weight-bearing plain films showed mild narrowing of the medial joint line and early medial compartmental osteoarthritis, and T_2-weighted MR images showed a displaced degenerative tear involving the posterior horn of the medial meniscus with a Baker cyst measuring 5.0 cm posteromedially (Figures 1A, 1B) and moderate chondromalacia of the medial compartment with moderate joint effusion, but no evidence of SO. These images were obtained approximately 3 weeks before index surgery.

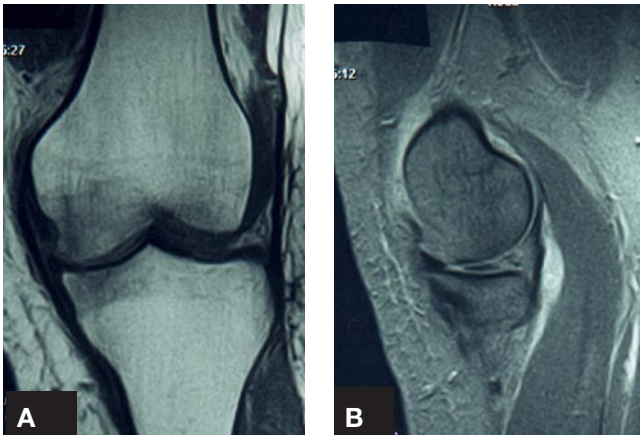

Figure 1. Case 1—preoperative fat-suppressed T_2-weighted coronal **(A)** and sagittal **(B)** magnetic resonance images show posterior horn medial meniscus pathology and no recognizable evidence of focal spontaneous osteonecrosis of the medial femoral condyle.

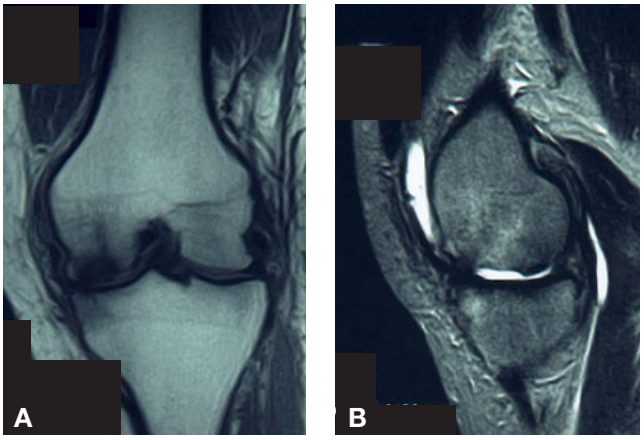

Figure 2. Case 1—postoperative fat-suppressed T_2-weighted coronal **(A)** and sagittal **(B)** magnetic resonance images show osteonecrosis and collapse of the medial femoral condyle, stage IV.

All conservative measures, including physical therapy, use of nonsteroidal anti-inflammatory drugs, and intra-articular corticosteroid injections, failed to relieve the patient's symptoms. Arthroscopy of the knee was then scheduled for approximately 6 weeks after symptom onset. With the patient under general anesthesia and in the supine position, the surgery was performed with a 30° videoarthroscopy system. The principal pathology was located in the medial compartment, where there was an irreparable complex tear of the posterior horn of the medial meniscus—consistent with the patient's symptoms. In addition, there was a radial and longitudinal component along with mild chondromalacia of the undersurface of the patella, without significant arthritis. The anterior cruciate ligament (ACL) and the lateral meniscus were both intact. The patient did not have a chondroplasty, nor were any electrothermal therapies applied. The procedure proceeded without complications.

By 9 weeks after surgery, the patient's knee findings and pain complaints had not improved but worsened. The patient presented with gradually increasing recurrent medially based knee pain exacerbated by use and relieved at rest. Despite prior aspiration and an intra-articular steroid injection (triamcinolone acetonide), symptoms persisted. Additional physical therapy had not been effective. There was no interval trauma.

Physical examination revealed thickening and swelling around the medial joint space (1+ effusion). There was no pes anserinus tenderness or neurovascular change. The patient did not have any restricted hip motion or gait abnormalities that would suggest the knee pain was referred from the hip. Range of motion was -5° to +120°.

Plain films obtained approximately 9 weeks after surgery showed mild to moderate osteoarthritis of the patellofemoral joint and mild arthritis of the medial joint space with mild joint space narrowing in the knee. There was no significant osteophyte formation. Compared with the right knee joint space, the left showed a 50% reduction. Technetium bone scans, also obtained approximately 9 weeks after surgery, revealed increased uptake at the medial joint space. Cultures were negative at this time.

Plain films obtained 6 to 8 months after surgery showed a large osteochondral defect (1.5x1.0 cm) in the medial femoral condyle involving the weight-bearing portion, consistent with SO. There were secondary degenerative changes, medially of the distal femur and proximal tibia, with progressive varus deformity. MR images obtained around the same time showed osteonecrosis and collapse of the medial femoral condyle, which fit the clinical pattern of a stage IV lesion (Figures 2A, 2B). In addition, the patient still had a Baker cyst (4.0x1.0 cm) in the medial aspect of the knee and severe complete secondary chondromalacia of both the medial femoral condyle and the medial proximal tibial plateau. No new medial meniscal tear was seen. The patient was offered an elective left medial total knee arthroplasty. The patient declined the procedure and progressed to advanced medial compartment osteoarthritis with varus deformity and no resolution of pain.

Case 2

A man in his mid 50s presented with acute and severe medially-based right knee pain that had progressively worsened over a week. The patient was 5 feet 5 inches tall, weighed approximately 250 pounds, and was a nonsmoker with hypertension, generalized osteoarthritis, and severe degenerative disc disease. The disabling pain made weight-bearing impossible. There was no recent history of trauma, and the patient denied popping, snapping, effusion, bleeding, and ecchymosis. To our knowledge, he had not undergone any previous knee injury or any form of corticosteroid treatment other than the single intra-articular cortisone injection (triamcinolone acetonide) given at presentation, and to which he had not responded.

Physical examination revealed that the right knee pain was localized along the medial collateral ligament (MCL). Tenderness was evident medially along the collat-

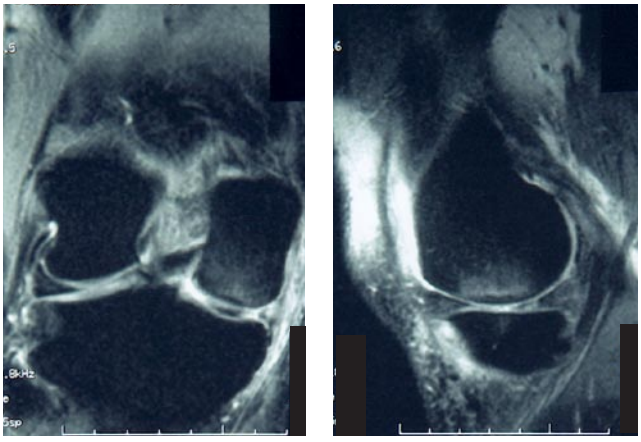

Figure 3. Case 2—12 days before surgery, fat-suppressed T_2-weighted coronal **(A)** and sagittal **(B)** magnetic resonance images show posterior horn medial meniscus pathology. There is no definitive evidence of focal spontaneous osteonecrosis of the medial femoral condyle, but there may be, in retrospect, some subtle changes at the articular surface medially. A surgeon who notices these changes should follow the patient and consider bone scanning.

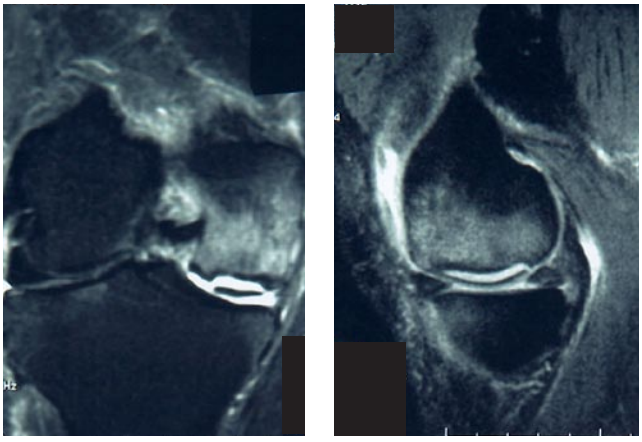

Figure 4. Case 2—22 days after surgery, fat-suppressed T_2-weighted coronal **(A)** and sagittal **(B)** magnetic resonance images show a frank osteochondral fracture of the medial femoral condyle and associated avascular necrosis of a large, detached, nondisplaced osteochondral fragment.

eral ligament and pes anserinus but mostly the joint line. The MCL was intact, as were the ACL and the posterior cruciate ligament (PCL). The patellofemoral joint was asymptomatic, and the patient was neurovascularly intact. Initial plain films obtained at 1 week were negative and showed no loose bodies. MR images obtained 1.5 and 3 weeks before the index surgery showed a degenerative complex tear of the posterior horn and midbody of the medial meniscus with moderate to marked adjacent medial compartment chondromalacia (Figures 3A, 3B). There was also a small effusion with slight extension of fluid into the semimembranosus and gastrocnemius bursa. Diagnostic and surgical videoarthroscopy of the right knee was scheduled.

At videoarthroscopy, with the patient under general anesthesia, partial medial meniscectomy and chondroplasty of the medial femoral chondral were performed. The partial meniscectomy involved basket resection of the majority of the posterior horn of the medial meniscus, which was unstable because of radial and horizontal components of the degenerative medial meniscus tear. The chondroplasty involved the articular weight-bearing surface of the medial femoral condyle. A mechanical shaver was used in this procedure, but drilling and electrothermal therapy were not required. The unstable osteochondral flap and the fraying of the articular cartilage, consistent with Outerbridge grade III changes, were addressed over an area of approximately 3 to 4 cm, but electrothermal therapies were not applied.

The patient's condition worsened rapidly after surgery. The patient did not want to bear weight because of the severe pain. He continued having severe recurrent medially based pain and effusions (1+/2+), and a mild varus deformity was rapidly developing. Additional physical therapy, an additional intra-articular injection of steroids (triamcinolone acetonide), and rest all failed to relieve symptoms 3 weeks after surgery. Physical examination revealed normal motion and no cruciate or collateral ligament instability. There was medial tenderness, particularly over the femoral condyle.

Plain films obtained approximately 3 weeks after surgery now clearly showed an osteochondral defect to the medial femoral condyle and a 75% collapse in the medial compartment. Standard sagittal, axial, and coronal MR images obtained around the same time showed intermediate grade II to III patellofemoral chondromalacia with minimal joint effusion. A frank osteochondral fracture had developed on the weight-bearing aspect of the medial femoral condyle, with associated osteonecrosis of a large, detached, but nondisplaced osteochondral fragment (3x1.5 cm x 4 mm in depth) (Figures 4A, 4B).

As unrelenting severe pain and varus deformity continued over several months, a cemented total knee arthroplasty (using a PCL-sacrificing technique) was performed on the right knee. The avascular medial femoral condyle was resected and grafted from resected bone as needed. A cystic lesion (1.5x1.0 cm x 8 mm) was aggressively curettaged and locally grafted. No complications occurred during the procedure, and surgical bone pathology was consistent with avascular necrosis. The patient had good pain relief, but range of motion was slightly decreased in flexion (0°-100°). At 2-year follow-up, there were no clinical or radiographic signs of implant loosening and no residual pain.

Discussion

Since Ahlback and colleagues[1] first described the symptoms commonly associated with SO, several cases have been documented, illustrating a high incidence rate of SO of the medial femoral condyle.[2,4-9] The exact etiology of SO after meniscectomy is unclear, but a literature search turned up 3 theories for development of osteonecrosis of the knee (Figure 5).

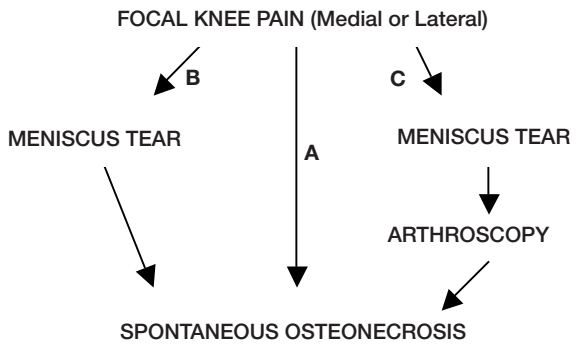

Figure 5. Theories for the development of focal spontaneous osteonecrosis (SO) of the knee. **Natural-history-of-SO theory (A):** Knee pain results from idiopathic SO, which is subclinical (patient in early stage of disease, diagnosis not apparent). **Altered-stress-biomechanics theory (B):** Knee pain results from meniscus tearing, which secondarily causes abnormal loading leading to SO. **Iatrogenic theory (C):** Knee pain results from medial meniscus tear, and subsequent arthroscopy may contribute to SO.

Three Theories for the Development of Knee Osteonecrosis

The first may be referred to as the **"natural-history-of-SO theory,"** according to which the patient is in the early stage of the disease process and is symptomatic, but the diagnosis is not yet apparent. The disease itself has been described as following a 4-stage course: I, generally no observational findings; II, small flattening of the medial femoral condyle; III, increased flattening with an apparent lesion indicated by a sclerotic halo; IV, articular cartilage collapse and additional degenerative changes that cause varus or valgus deformities.[8,11,17] Soucacos and colleagues[11] reported that the interval for stage I after symptom onset was 6 to 8 weeks; stage II, 2 to 4 months; stage III, 3 to 6 months; stage IV, 9 months to 1 year. With an evolving disease like osteonecrosis, it is difficult to establish whether positive meniscal pathology is a predisposing factor or simply an overlying pathology that may be present during the course of the disease. Reports have emphasized that preexisting meniscal pathology correlates well with the later overlying diagnosis of SO and indicates a high rate of SO of the medial femoral condyle after arthroscopy.[2,4-7,9] The Table illustrates a series of patients who had concurrent meniscus pathology and were later diagnosed with SO after surgery.

The second theory may be referred to as the **"altered-stress-biomechanics theory,"** according to which meniscus tearing causes altered knee loading leading to SO. Knee overloading has been suggested as possibly causing microfractures and later ischemic conditions.[7-10,18] In a study of 33 patients (mean age, 60 years), Norman and Baker[4] speculated that meniscal pathology may play a crucial role in the disease evolution by adding weight-bearing stress to the surrounding articular surface. Muscolo and colleagues[7] similarly suggested that abnormal loading may be involved, and they indicated that 50% of the force was transferred through the meniscus in extension and 85% when the knee is flexed at 90°.

Altered Blood Supply: A Common Thread?

In all 3 theories, development of SO in the medial femoral condyle appears to be a compound problem involving an altered blood supply.[2-5] In particular, vascular compromise of the subchondral bone has been described as contributing to the advance of the disease. The resulting insufficient blood flow produces edema and increased pressure within the problem area, and these changes further impede the vascular supply.[5,10] Examining histopathologic studies of 7 patients with SO in the medial femoral condyle, Yamamoto and Bullough[18] indicated that the primary event leading to later stages of the disease may have been a subchondral fracture resulting from underlying osteoporosis. Thrombosis, atherosclerosis, occlusion of venous flow, and marrow changes have been suggested as possible factors leading to ischemic conditions.[8]

With regard to blood supply, Reddy and Fredrick3 provided a plausible explanation for a high incidence of avascular necrosis of the medial condyle relative to the lateral femoral condyle. The vasculature of 12 adult cadaver legs showed a differential pattern of blood flow between the medial and lateral femoral condyles. The medial femoral condyle received vascular flow predominantly from the superior medial geniculate artery, with small contributions from branches of the popliteal artery. More important, the vascular supply was primarily to the middle third of the condyle and in effect created an anterior "watershed" zone. The medial condyle collateral system was lacking compared with the lateral femoral condyle system, which was supplied by both the superior lateral geniculate and anastomotic branches of the inferior lateral geniculate. The finding of an anterior "watershed" zone and diminished collateral system may also help explain the high incidence rate of SO of the medial femoral condyle.

The last theory may be referred to as the **"iatrogenic theory,"** according to which arthroscopy after a medial meniscus tear may contribute to SO. Muscolo and colleagues[7] claimed that removal of more than 15% to 34% of the medial meniscus can increase the contact pressure such that the contact area may be reduced by as much as 50% after meniscectomy. Kobayashi and colleagues[2] reported that bone marrow changes in 32 patients after meniscectomy were evident only on the surgical side for both lateral and medial meniscal procedures. They concluded that the degree of meniscectomy affected prevalence of postmeniscectomy bone changes.

Progression of SO on Scans

Plain films, MR images, and bone scans are all used to verify changes in the structure of the skeleton. With the progression of SO through 4 stages, it appears that there is an interval after symptom onset in which the disease in its earlier stages may not be readily distinguished with MRI and radiography. In a study of 6 patients with

meniscus pathology (4 medial and 2 lateral meniscal tears), Johnson and colleagues[6] found that the SO diagnosis was unidentifiable 6 weeks before surgery, after symptom onset, but was made after partial meniscectomy at a mean of 4 months. Kobayashi and colleagues[2] reported that bone marrow changes were most often visible on MR images within 8 months after surgery. In an animal study, Nakamura and colleagues[19] observed that it may take up to 4 weeks after symptom onset to confirm SO by MRI. Muscolo and colleagues[7] reported on 8 patients who had medial meniscus tears within a mean of 18 weeks before MRI diagnosis and showed evidence of SO after arthroscopy. Examining plain films, Norman and Baker[4] reported the earliest evidence of flattening of the medial condyle (3 months after symptom onset). Also using plain films, al-Rowaih and colleagues[20] reported that the typical SO lesion in 14 knees was observed at a mean of 28 weeks. These cases also seem to emphasize the evolution of the disease process.

Treatment and Prognosis

The prognosis and treatment options for patients with SO seem to vary with the extent of the lesion. Ecker and Lotke[10] noted that lesions that make up more than 50% of the condyle have a poor prognosis. Lotke and colleagues[21] found a poor prognosis for lesions when the ratio of its transverse width to the affected condyle was larger than 0.4. Muheim and Bohne[22] found that a ratio of lesion width to lesion length indicated a poor prognosis with lesions larger than 5 cm^2. Finding small lesions early on tends to result in good prognoses with conservative treatments of therapy and limited weight-bearing activity.[5,10,11] Larger lesions tend to require surgical interventions ranging from débridement, core decompression, and osteotomy to prosthetic replacement.[8,10,11] Many factors (eg, extent and location of bone necrosis) ultimately determine the prognosis and can be difficult to quantify in a patient.

"When patients...present with medially based knee pain and have normal plain films and MR images, scintigraphy should be performed...."

The majority of cases show early signs of SO with MRI during the initial diagnosis.[2,7-10] However, discrepancies have been reported in the specificity of MRI when used to detect early changes in an evolving disease, such as avascular necrosis, and bone scans have shown increased uptake in patients who develop SO when MR images and plain films do not show initial abnormalities in the subchondral bone.[7,8,11] For example, in 1997, after reviewing 101 patients with SO not related to a degenerative meniscus, Soucacos and colleagues[11] found normal plain films at 6 to 8 weeks and generally no T_2-weighted abnormalities, but positive scintigraphy. At 2 to 4 months, some abnormalities appeared on plain films and MR images, but diagnoses were mainly on the basis of technetium 99 bone scans. After 3 to 6 months, significant abnormalities were apparent on plain films, MR images, and bone scans. Pollack and colleagues[16] reported on 10 knees with SO symptoms and positive scintigraphy; 2 of the 10 knees appeared normal on MRI. Similarly, Bjorkengren and colleagues,[17] in a study of 16 patients with SO and positive radionuclide bone scans, found 2 patients with normal T_1-weighted MR images and 4 patients with normal T_2-weighted MR images.

MRI and radiography are better tools for diagnosing SO in its later stages, while radionuclide bone scans may provide evidence of early changes suggestive of an underlying pathology of SO.[6,8-11] With there being variation in the reported appearance of SO both before and after surgery, we should not underestimate the importance of ruling out SO in its early stages, especially when a meniscal pathology could parallel these stages.

Conclusions

We presented 2 cases of SO of the medial femoral condyle after arthroscopy in 2 patients with no history of trauma or identifiable predisposing SO factors other than age. Similarly, no predisposing 2nd ON factors were identified. Initial preoperative MR images and plain films showed medial meniscal tears and no definitive evidence of SO. Radionuclide bone scans were not used in preoperative diagnosis because both patients had evident medial pathology consistent with symptoms.

Failure to improve with rest and continued pain at night and/or at rest should suggest the diagnosis of SO. SO of the medial femoral condyle is a significant finding in conjunction with a degenerative medial meniscus tear. When patients, particularly older patients, present with medially based knee pain and have normal plain films and MR images, scintigraphy should be performed so that misdiagnosis and unneeded arthroscopy can be avoided. Early recognition of SO may help prevent collapse, deformity, and secondary osteoarthritis of the involved joint space. SO is relatively uncommon compared with medial meniscus tears, and this should be kept in mind when the patient's knee pain is nonmechanical, worsens with inactivity or at night, fails to improve after arthroscopy, or is out of proportion to what might be expected from meniscal pathology alone.

Whether the development of SO after arthroscopy is due to the procedure, the unrecognizable early-stage degenerative joint disease, or the meniscal tear itself has not yet been established, but meniscal tears have been reported as existing before the disease develops (Figure 5). Most likely, SO is a separate entity, and factors such as medial meniscus tears and arthroscopy do not cause SO but may

TABLE. SUMMARY OF REPORTED CASES OF FOCAL SPONTANEOUS OSTEONECROSIS (SO) WITH MENISCUS PATHOLOGY*

Study	Sex	Mean Age (y)	Steroid Use	MMT	LMT	Chondro-malacia	Arthroscopy	Arthroscopic Meniscectomy	Time	SO	Area SO or Change
Muscolo et al[7]	M	54	No	Flap	—	FC	Yes	Partial	16 wk	Focal	SO MFC
	F	57	No	Flap	—	—	Yes	Total	20 wk	Focal	SO MFC
	F	62	No	Flap	—	FC	Yes	Partial	6 wk	Focal	SO MFC
	F	65	No	BH	—	—	Yes	Total	36 wk	Focal	SO MFC
	F	65	No	LT	—	FC	Yes	Total	8 wk	Focal	SO MFC
	M	69	No	HT	—	—	Yes	Total	7 wk	Focal	SO MFC
	M	70	No	HT	—	FC	Yes	Partial	32 wk	Focal	SO MFC
	M	75	No	LT	—	—	Yes	Total	17 wk	Focal	SO MFC
Brahme et al[9]	4M/3F	60.5	—	4 HT		6FC	Yes	7 partial with debridge	2-14 mo	6 focal	5 SO MFC
			—	2 BH							1 SO MFC/TP
			—		1 AT	1 both	Yes			1 focal	1 SO LFC/TP
Johnson et al[6]	F	79	No	MMT	—	FC†	Yes	Partial, CP	3 mo	Focal	SO MFC
	M	58	No	MMT	—	Both†	Yes	Partial, CP	5 mo	Focal	SO MFC
	F	75	No	—	LMT	—	Yes	Partial	4 mo	Focal	SO LFC
	M	41	No	MMT	—	Both†	Yes	Partial	6 mo	Focal	SO MFC
	M	54	No	MMT	—	FC†	Yes	Partial, CP	3 mo	Focal	SO TP
	F	53	No	MMT	—	FC†	Yes	Partial, CP	3 mo	Focal	SO MFC
	F	62	No	—	LMT	FC†	Yes	Partial, CP	4 mo	Focal	SO TP
Santori et al[5]	—	21	—	Radial/ posterior horn	—	—	Yes	Partial	<1 mo	Focal	SO MFC
	F	47	—	BH	—	—	Yes	Partial	1 mo	Focal	SO MFC
Norman & Baker[4]	14M/19F	60	45% intra-articular	21 MMT 6 no tear 6 N/A	—	—	No	—	—	—	31 SO MFC 1 SO LFC 1 SO MFC/LFC
Kobayashi et al[2]	51M/42F	36.6	—	44 MMT	49 LMT	17 medial/ 10 lateral†	Yes	57 partial 36 total	8 mo	—	Marrow changes in 16 FC/TP 6 FC 10 TP
Narvaez et al[8]	5M/8F with 2nd ON	49	2 gluco-corticoid 2 alcoholism 2 renal transplant 1 thalassemia 1 leukemia 3 systemic steroid	3 MMT	—	—	No	— ±	105 d 9 focal 120 d	4 polytopic	4 bilateral FC/TP 4 MFC/TP 2 unilateral FC/TP 2 LFC 1 MFC
	7M/17F	66	No	22 MMT	—	—	No	—	24 focal		19 SO MFC 3 SO TP 2 SO MFC/LFC

*MMT indicates medial meniscus tear; LMT, lateral meniscus tear; BH, bucket handle; LT, longitudinal tear; HT, horizontal tear; AT, anterior tear; CP, chondroplasty; Time, postoperative time until SO diagnosis; MFC, medial femoral condyle; TP, tibial plateau; LFC, lateral femoral condyle; FC, femoral condyle; 2nd ON, secondary osteonecrosis.
†Outerbridge[23] classification grade II/III.

hasten SO symptom onset because of secondary collapse or osteoarthritis. This scenario seems consistent with the cases presented here and the literature reviewed.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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