



CASE SERIES

Algorithm for Treatment of Hip and Knee Osteonecrosis: Review and a Presentation of Three Example Cases

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Abstract

Osteonecrosis (ON) of the hip and knee can be a source of severe morbidity for affected individuals. Although several risk factors and explanations for the pathogenesis of ON have been recognized, there exists little consensus in the literature as to the appropriate clinical management. We present three patients treated at our institution and a treatment algorithm for hip and knee osteonecrosis.

Keywords

Osteonecrosis, Spontaneous Osteonecrosis of the knee, Avascular necrosis, Algorithm

Abbreviations

ON: Osteonecrosis; THA: Total Hip Arthroplasty; TKA: Total Knee Arthroplasty; AVN: Avascular Necrosis; CSI: Corticosteroid Injection.

Introduction

Osteonecrosis (ON) of the hip most often occurs in the third to fifth decade of life. It has a predictable progression from asymptomatic lesion visual only on advanced imaging to eventual bone collapse requiring surgical intervention. Risk factors commonly associated with ON of the hip include trauma, excessive alcohol use, prolonged corticosteroid use and any number of diseases that can lead to ischemia of the femoral head. Even with exposure to these risk factors, however, people infrequently develop ON and even fewer become symptomatic. In a study by Lieberman, et al. looking at a cohort of liver transplant patients with extensive corticosteroid exposures only 2-3% went on to develop ON [1,2]. Overall, hip ON is a multifactorial disease process

with a poorly understood genetic contribution and dose response mechanism.

Staging

The primary measurement tools used in the discussion and treatment of hip ON are the Ficat staging system and the Kerboul angle for assessing the degree of femoral head involved in a lesion. The original Ficat system was based on clinical and radiographic signs as well as scintigram and core biopsy findings [3]. Table 1 shows a simplified explanation of the Ficat system.

The Ficat classification was later modified by Steinberg to be based solely on clinical symptoms and radiographic changes [4]. These changes are described briefly in Table 2.

The combined necrotic angle, originally described by Kerboul, is an estimate of the extent of involvement of the femoral head. This was originally described as the sum of the angles of the involvement on AP and lateral radiographs, and later on MRI [5-7]. A study by Ha, et al. showed that if the combined necrotic angle was < 190, the risk of progression to femoral collapse was significantly less than an angle of > 240 [8]. Intuitively, this makes sense as one would expect lesions with greater involvement of the femoral head to have a higher rate of collapse.

Both surgical and non-surgical options exist for hip osteonecrosis. Medical treatment as a monotherapy is generally only used in early ON with pre-collapse lesions in an attempt to slow or stop disease progression and mitigate the need for surgical intervention [9-11]. One study by Wang, et al. demonstrated extracorpore-

Table 1: Ficat and Arlet classification for hip osteonecrosis.

Classification and staging systems for osteonecrosis	
Ficat and Arlet	
Stage I	Normal
Stage II	Sclerotic or cystic lesions
	A) No crescent sign B) Subchondral collapse (crescent sign) without flattening of the femoral head
Stage III	Flattening of femoral head
Stage IV	Osteoarthritis with decreased joint space with articular collapse

Table 2: Steinberg classification for hip osteonecrosis.

Stage	Radiographs	MRI
0	Normal	Normal MRI and bone scan
1	Normal	Abnormal MRI and/or bone scan
2	Cystic/sclerotic changes	Abnormal MRI and/or bone scan
3	Crescent sign (subchondral collapse)	Abnormal MRI and/or bone scan
4	Flattening of femoral head	Abnormal MRI and/or bone scan
5	Narrowing of joint	Abnormal MRI and/or bone scan
6	Advanced degenerative changes	Abnormal MRI and/or bone scan

Table 3: Two year survival rate for surgical treatment options in hip ON.

Treatment option	Two-year survival rate (%)	Author
Core decompression	74	Rajagopal, et al. [16]
Core decompression with allograft	81	Wei, et al. [17]
Core decompression with autograft	78	Keizer, et al. [18]
Core decompression with vascularized autograft	67	Berend, et al. [19]
Rotational osteotomy	92	Biswal, et al. [20]

al shock wave treatments to be effective as compared to core decompression with autograft when using pain and Harris hip scores as outcome measures [12]. There is limited evidence that enoxaparin and alendronate have been shown to reduce disease progression. However, enoxaparin has not been shown to work in steroid induced ON, and the data on alendronate has not been reproducible in further studies [13-15]. One of the most important things to consider is that patients who develop femoral head ON generally have had a long history of insult to that region of bone, either from steroids, alcohol, or coagulopathy. In this setting, progression of disease is expected and attempt at bone preserving interventions have worse outcomes overall [11].

Surgical Treatment

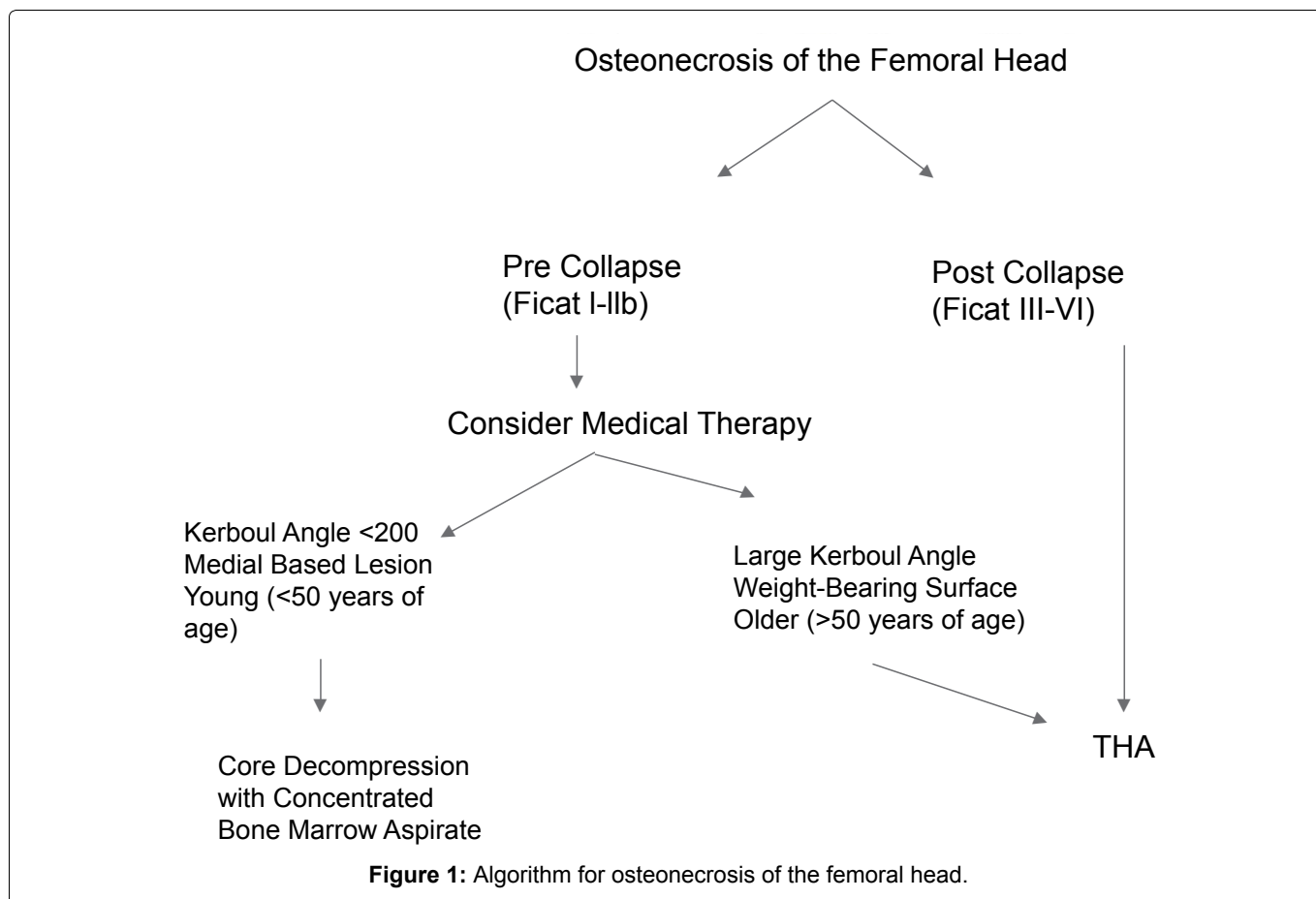
Options for surgical management of hip ON can largely be classified as either femoral head sparing or femoral head replacing. Femoral head sparing techniques include any number of methods for core decompression with or without the use of grafting material. Grafting substrates can then be further divided into synthetic, allogenic and autogenic. The gold standard in grafting material is to use a vascularized autograft, typically taken from the fibula, to fill in the defect left behind by the core decompression [16,17]. This technique has been associated with improved outcomes in some patient populations, but is a technically demanding surgery. The various treatment options with the 2-year survival rate, as defined as no further intervention over that time, is shown in Table 3 [18-20].

Rotational osteotomies also fall into the category of femoral head sparing treatment, but unlike decompression alone, are more technically demanding [20]. The goal is to offload the pre-collapse lesion through rotation of the femoral head, thus allowing continued weight bearing through an unaffected region [21,22]. Unfortunately, rotational osteotomies may create proximal femoral anatomical challenges for any subsequent arthroplasty procedure.

Total Hip Arthroplasty (THA) is the definitive treatment and the likely final pathway for most patients with femoral head osteonecrosis. Hemiarthroplasty is less often performed due the more predictable outcome and durability with THA as well as the often younger, higher demand patient who presents with this pathology. THA is also preferred over hip resurfacing due to the potential complication of femoral neck fracture or poor fixation in compromised proximal femoral bone stock. Previously, it was thought that patients with ON treated with THA had a greater inherent risk of dislocation. This risk of dislocation is more likely the result of greater demand placed on the Total Hip Arthroplasty (THA) by a younger patient population [23]. Improvement in THA materials and surgical techniques has led to improved outcomes. Recent studies have quoted 83-93% survivorship at > 15 years follow-up for patients with ON treated with THA [24-29].

Our Algorithm for Osteonecrosis of the Hip

All patients with radiographic and clinical evidence



of osteonecrosis undergo a routine hypercoagulation screen, including blood test for Lupus anticoagulant, Cardiolipin IgG and IgM, Protein S activity, Protein C activity, Antithrombin III activity, and serum Homocysteine. This standard set of labs was developed through conjunction with our Hematology partners at the treating institution (Kaiser Permanente West LA Medical Center). These results help us determine appropriate Deep Vein Thrombosis (DVT) prophylaxis as well as seek appropriate medical consultation.

While the Ficat Staging system is the primary classification tool used in research settings, we generally determine treatment based on whether the lesion is pre-collapse or post-collapse. Our algorithm is shown in brief in [Figure 1](#). Once a patient has progressed to post-collapse stage (we include patients with subchondral collapse (crescent sign) as post-collapse lesions), there are few if any treatments short of THA that can offer patients predictable outcomes. It is the patients who are pre-collapse and symptomatic in which we try our hardest to preserve the hip joint and delay arthroplasty. We have found that in young patients with a pre-collapse lesion, a small Kerboul angle (< 200), and a medially based lesion (closer to the fovea) have the best chance of achieving acceptable outcomes without arthroplasty. When the lateral pillar is maintained, as in medially-based focal lesions, the support of the femoral head remains adequate and there is a slower disease progression overall. In these select patients we

offer core decompression in an attempt to preserve the native joint if possible. Our technique is to use a more proximal start site for core decompression, as Colwell, et al. showed that there is an increased risk of immediate postoperative fracture in cores that were taken distal to the vastus ridge [30]. We use a series of 3.2 mm Steinman pins percutaneously introduced into the femoral head to make 3 small diameter core decompression sites as originally described by Mont, et al. [31]. We augment Mont's described procedure by over drilling the center most pin with a 4.5 mm drill and arthroscopic long-handle curette. Additionally, we have begun augmenting our core decompression with the use of concentrated iliac crest bone aspirate, which has recently been shown to significantly improve the clinical outcomes of this procedure [32]. Postoperatively, we instruct our patients to be toe touch weight-bearing for 6 weeks and 6 weeks of partial weight-bearing to allow for bone deposition and healing.

Elderly patients (over age 50) who have symptomatic hip osteonecrosis, however, should be considered for THA regardless of the stage of their lesions. THA provides reliable pain relief in this population just as it does in the setting of osteonecrosis.

Case 1

MG is a 31 yo F who presented with left hip pain for over a decade. MRI and radiographs had been obtained prior to presentation to our office which showed ante-

rior quadrant cystic changes in the femoral head consistent with AVN in the pre-collapse state. On physical exam, the patient had excellent range of motion. The patient had already tried and failed conservative measures such as oral pain control, CSI, and protected weight bearing. She elected to undergo core decompression of the affected femoral head. At one year postoperatively she has limited improvement in her pain and continues to rehabilitate [Figure 1](#), [Figure 2](#), [Figure 3](#), [Figure 4](#), and [Figure 5](#).

Case 2

ID is an 18-year-old female, with Systemic Lupus Erythematosus (SLE) treated with long term steroids, collect and plaquenil and a history of right fibular osteosarcoma, who presented February 2015 with a one-year history of progressive activity-related bilateral hip pain. The patient described deep aching pain in the groin with radiation into the buttock and down the medial thigh on

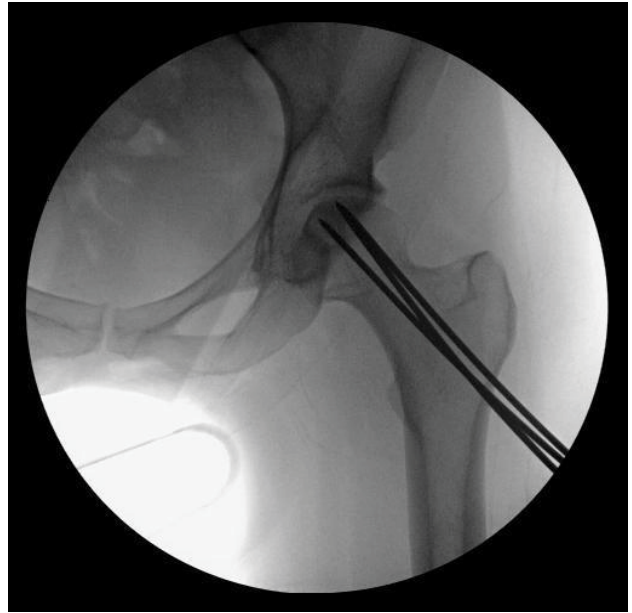


Figure 4: Intraoperative radiograph.

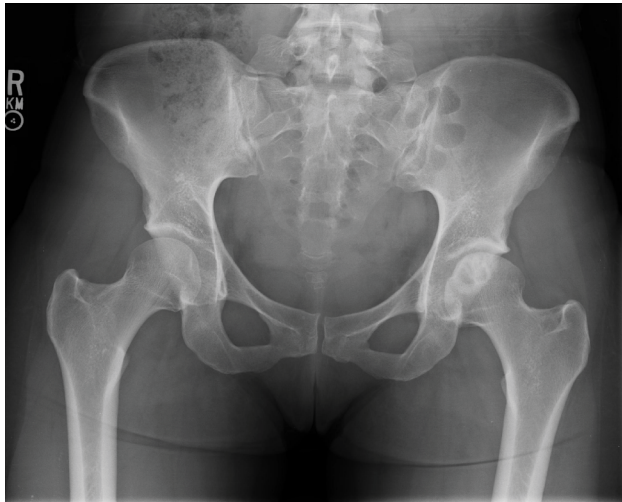


Figure 2: Preoperative AP pelvis.

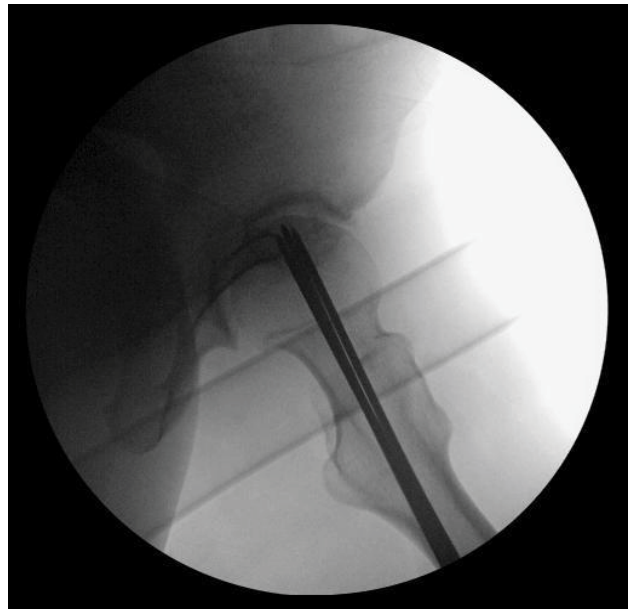


Figure 5: Intraoperative radiograph.

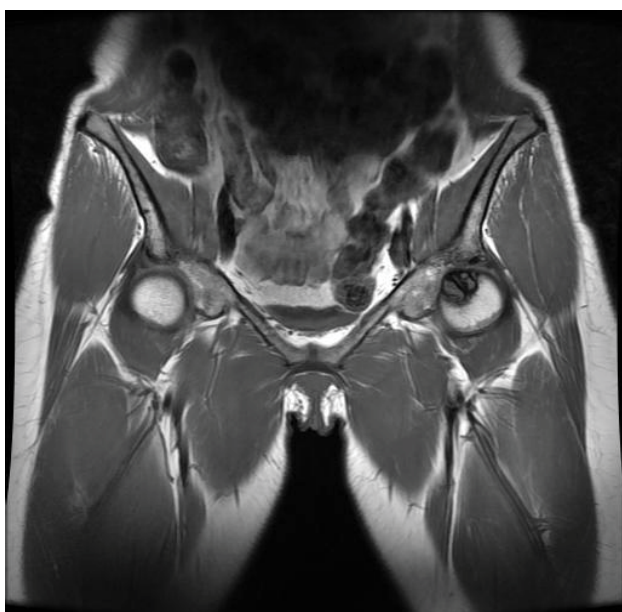


Figure 3: Preoperative coronal MRI.



Figure 6: Preoperative AP pelvis.

both hips. At presentation, she could no longer participate in recreational activity. She denied ever smoking and she does not use alcohol. Right hip range of motion was zero to ninety degrees of flexion, left hip range of motion was 0-100 degrees of flexion with impingement pain bilaterally. Initial Radiographs are seen in [Figure 6](#).

Given her history an MRI of the pelvis was done at that time showing bilateral femoral head osteonecrosis.

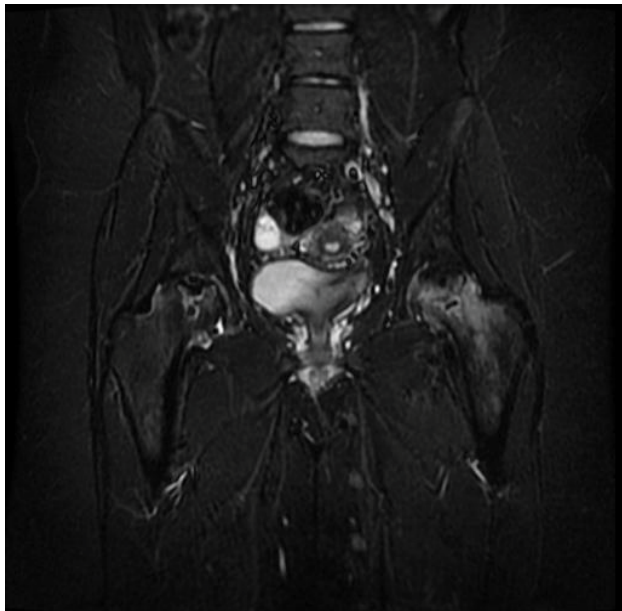


Figure 7: Preoperative coronal MRI.

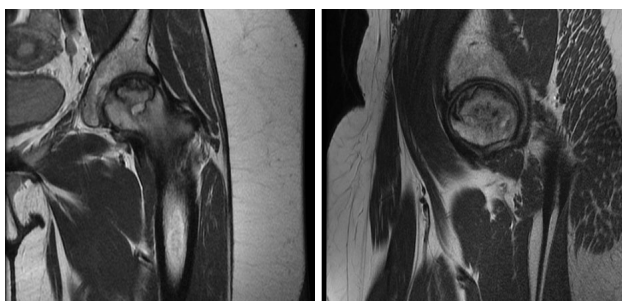


Figure 8: Preoperative coronal and sagittal MRI of left hip.



Figure 9: Repeat preoperative AP pelvis.

Both femora had extensive involvement with collapse [Figure 7](#) and [Figure 8](#).

Six months following initial presentation she had stopped attending college due to hip pain and was unable to ambulate without assistive device (crutches). Repeat radiographs showed advancement to collapse on bilateral femoral heads ([Figure 9](#)). She was scheduled for staged bilateral total hip arthroplasty and she has done well postoperatively. [Figure 10](#) is an intraoperative clinical photograph showing advanced femoral head collapse and postoperative radiograph showing bilateral THA after staged procedures ([Figure 11](#)).

While we attempted a trial of conservative treatment in this patient one can see the natural course of large osteonecrotic lesions that involve the weight-bearing surface of the femoral head. These lesions have a high rate of progression to collapse and eventually require arthroplasty to alleviate their pain and improve function.

Osteonecrosis of the Knee

Osteonecrosis (ON) of the knee can be largely divid-

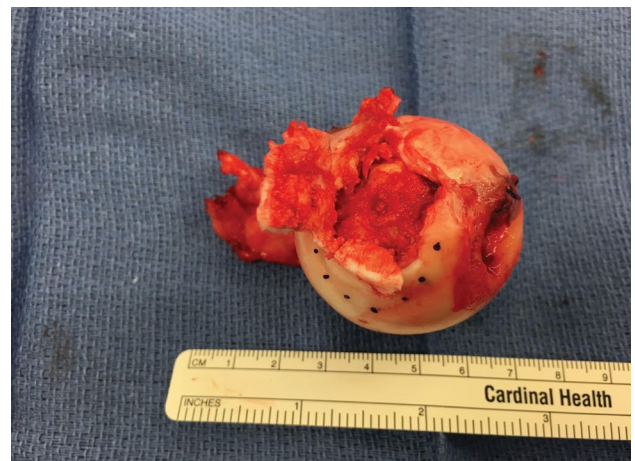


Figure 10: Intraoperative clinical photograph of femoral head.

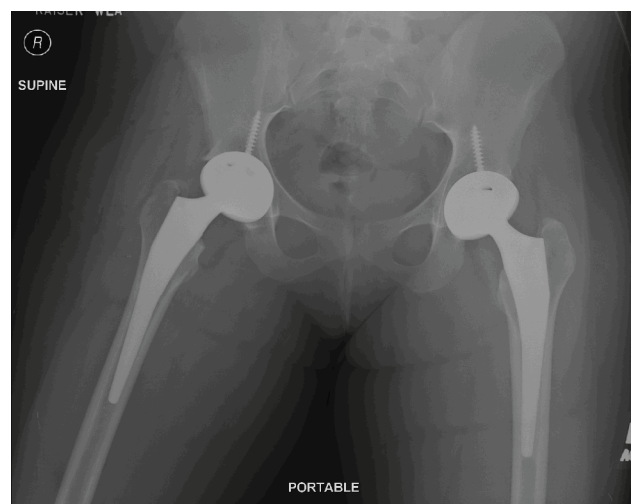
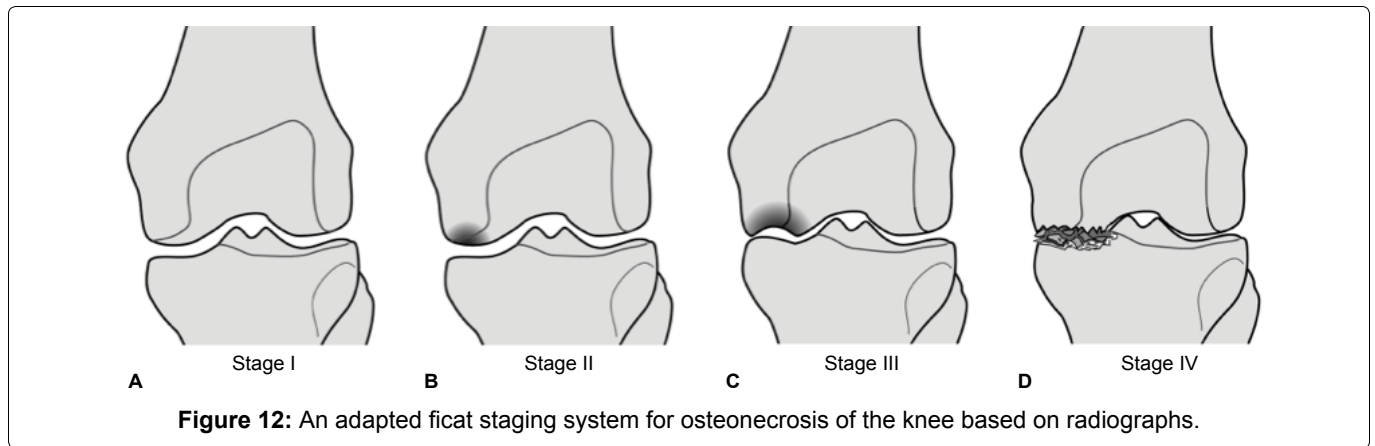


Figure 11: Postoperative AP pelvis.



ed into two categories; secondary ON and Spontaneous Osteonecrosis of the Knee (SONK). Secondary ON can be thought of similar to our understanding of osteonecrosis of the hip with etiologies that impart ischemic insult, direct cellular damage, or altered differentiation of stem cells to the affected area resulting in increased intraosseous pressure [33]. We use the term spontaneous osteonecrosis of the knee when there exists no history to place the patient at an otherwise increased risk. Spontaneous osteonecrosis of the knee has also been described as a subchondral insufficiency fracture. While postarthroscopic osteonecrosis of the knee has been described as a third distinct category, some authors believe it is the trauma from the surgical procedure itself results in secondary osteonecrosis of the knee while others have hypothesized that it is lasting effects of the arthroscopic procedure resulting in a subchondral insufficiency fracture [34,35].

Risk factors for secondary osteonecrosis include trauma, chemotherapy, myeloproliferative disease, radiation, alcohol, coagulation abnormalities, corticosteroid use, and smoking [36]. Approximately 90% of cases of secondary osteonecrosis have either alcohol abuse or corticosteroid use as an associated risk factor, however, secondary osteonecrosis is likely due to multiple risk factors working in concert as only a small percentage of patients with these risk factors go on to develop clinically significant osteonecrosis [37].

Spontaneous Osteonecrosis of the Knee (SONK) most often affects the medial femoral condyle in elderly patients [38]. They often present with an episode of acute pain to the affected region of the knee. MRI analysis has found a 9.4% prevalence of SONK in persons aged > 65 years [39]. Reddy, et al. discuss a “watershed area” that exists in the blood supply to the medial femoral condyle which could predispose at risk individuals to osteonecrosis [40]. Another theory for SONK is that these areas may represent insufficiency fractures as they often occur in elderly women with osteopenic bone [41].

Radiographic studies should begin with a standard X-ray series of the involved joint. However, standard radiographs can dramatically underestimate the involvement of the affected joint. Therefore, if osteonecrosis is

suspected and radiographs are normal there should be a low threshold to obtain an MRI. MRI will show early evidence of osteonecrosis with typical features such as serpentine lesions and bone marrow edema.

Staging

Mont, et al. describe an adapted Ficat staging system for Osteonecrosis of the Knee based on radiographs (Figure 12) [36].

In stage I there is no radiographic evidence of osteonecrosis. In stage II there is evidence of sclerosis surrounding the involved lesion. Stage III shows the progression to subchondral collapse and Stage IV shows collapse of the involved condyle. While precise staging is important in research our clinical decision is largely based on patient symptoms, extent of the lesions, and whether or the not patient is pre-collapse or post-collapse.

Management of knee osteonecrosis broadly consists of conservative management versus operative intervention. Operative treatment consists of arthroplasty or joint preserving procedures. Medical therapy is largely derived from hip literature and lacks compelling evidence for its use in the setting of knee osteonecrosis. More recently and specific to the knee, six patients predisposed to clot formation with early stage osteonecrosis treated with enoxaparin had no collapse in short term follow-up [42]. In another study, iloprost (PGI₂), a vasoactive prostaglandin analogue, proved to be an effective medical therapy for pre-collapse lesions in a study that included analysis of multiple joints with osteonecrosis (hip, knee, and ankle). The authors of this study strongly advocate for surgical intervention in post-collapse lesions [43]. While evidence for medical therapy in the setting of osteonecrosis of the knee is promising, more is needed for its application in clinical practice.

Surgical Treatment

Nonoperative treatment has a high clinical failure rate [44]. It is generally agreed upon that the management of late stage osteonecrosis that has progressed to debilitating arthritis should be definitively treated

with joint replacement. Non-arthroplasty procedures such as osteochondral allograft, autologous chondrocyte implantation, core decompression, and offloading procedures such as high-tibial osteotomy have all been proposed as reasonable treatment options but clinical evidence is lacking [38]. Of these options, small core decompression has shown the most promising results. Marulanda, et al. showed a 92% success rate and avoiding TKR in 97% of patients at 3 years for SONK [45]. However, to our knowledge no randomized trials are currently available for joint preserving procedures.

While prior studies had demonstrated less than optimal results with total knee arthroplasty relative to their non-osteonecrotic counterparts, more recent studies have shown success rates as high as 97% for total knee arthroplasty in the setting of osteonecrosis. Similarly, results of unicompartmental knee arthroplasty for SONK has improved, with excellent results being achieved in properly selected patients. The use of cement for fixation and femoral and tibial stems to bypass the osteonecrotic lesions is likely responsible for the improved results [46,47].

Our Algorithm for Osteonecrosis of the Knee

Once again, all patients presenting with osteonecrosis of the knee undergo a routine hypercoagulation screen (see Our Algorithm for Osteonecrosis of the Hip). Our first distinction in clinical decision making is the size and involvement of the lesion. For small lesions, such as those isolated to the medial femoral condyle often seen in SONK, we offer a trial period of protected weight-bearing (approximately three months) with symptomatic pain control with NSAIDs and corticosteroid injections.

After three months, if the patient fails to improve we consider small core decompression for patients less than 50 years of age and we offer either UKA or TKA to older patients. We offer UKA to patients with symptomatic unicompartmental osteonecrosis with minimal bone involvement (less than 8 mm depth of lesion and good rim bone for implant stability) without evidence of multicompartamental arthritis. The indications and contraindications for UKA are similar to those for osteoarthritis. Preoperative advanced imaging is required to ensure that all implants have adequate non-osteonecrotic bone stock to ensure fixation. Patients who have small focal lesions of osteonecrosis in the setting of diffuse osteoarthritis should bypass UKA and proceed to TKA.

For patients who present with diffuse osteonecrosis, we have found little efficacy in treatments short of TKA. In patients who present pre-collapse one can consider protected weightbearing and pain control. There is little role for core decompression in diffuse osteonecrosis. It is more likely that a patient seeking treatment for painful, diffuse osteonecrosis has already progressed to a collapsed stage and requires arthroplasty. We advocate the use of cement and stemmed prosthesis in order to achieve adequate fixation as well as transfer the forces away from the osteonecrotic bone and towards more healthy bone (Figure 13).

Case 1

SS is a 60-year-old Female, with no significant past medical history, who presented to our facility with a long-standing history of bilateral knee pain (R > L), however six months prior to presentation she had

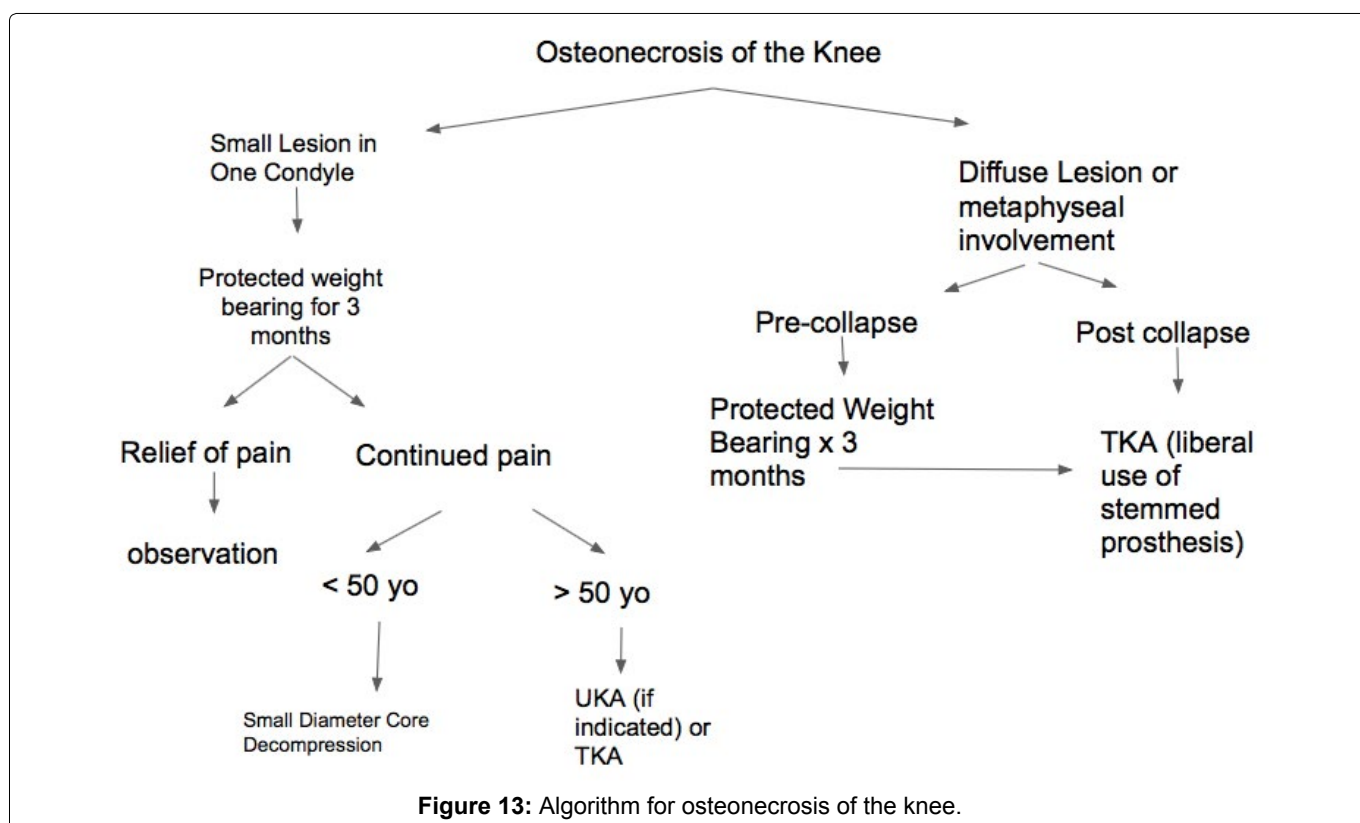




Figure 14: Preoperative radiographs of a 60 F with acute onset of bilateral knee pain.

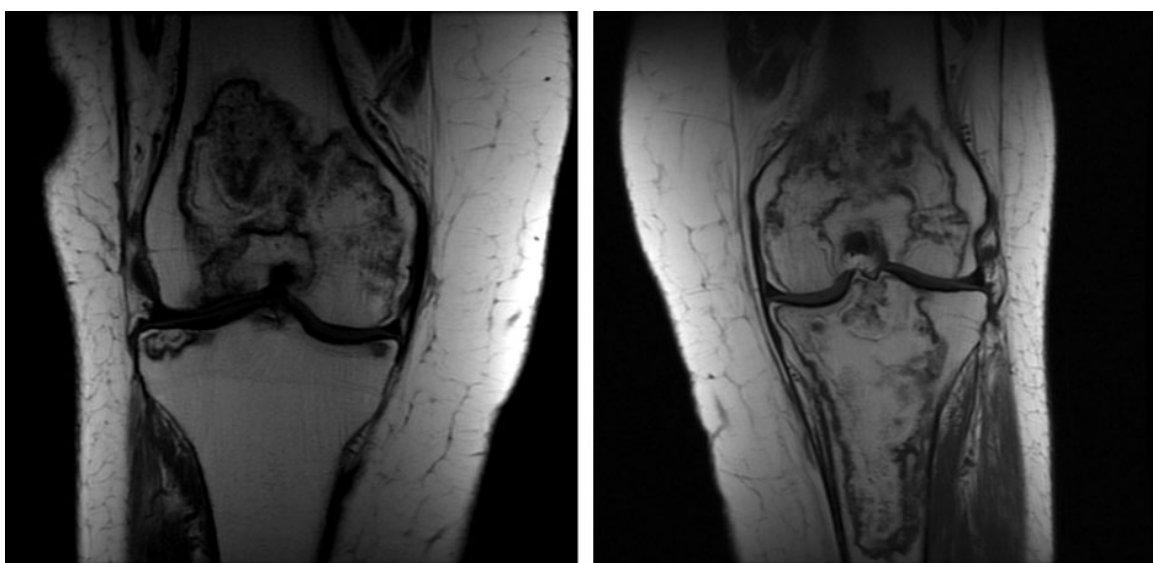


Figure 15: Right knee, left knee showing large areas of serpentine lesions in bilateral medial and lateral femoral condyles as well as the proximal tibia.

an acute episode of overnight bilateral knee pain which was so severe that she sought treatment in the emergency room. She denied trauma to either of her knees. She has history of corticosteroid injections into her knee and low back as well a short course of corticosteroid uses for her low back pain. Her physical exam, including her bilateral knee exam was unremarkable. She described the pain as primarily on the anterior aspect of both her knees. Initial radiographs were unremarkable (Figure 14). An MRI was ordered of her bilateral knees which revealed extensive osteonecrosis of bilateral distal femurs and proximal tibias (Figure 15). She received a diagnosis of spontaneous osteonecrosis of the knee and desired staged bilateral Total Knee Replacement (TKR) surgery. She underwent Right TKR initially, followed by Left TKR. Intraoperative photographs can be seen in Figure 16. Postoperatively her pain has markedly improved with unlimited ambulation. Her right knee ROM is 0-135 degrees and her left knee ROM is 0-125 (Figure 17).

One of the remarkable aspects of this case was the diffuse extent of our patient's osteonecrotic lesion given her relatively benign appearing x-rays. Moreover, the collapse that can be seen of her right femoral trochlear region cannot be appreciated on either the plain radiographic films or the preoperative MRI. One can assume that the severe pain that led to ER eval could have been her lesion collapsing. As one can see we used a stemmed femoral prosthesis to bypass the osteonecrotic lesion on the right knee and stemmed femoral AND tibial prosthesis to bypass the larger osteonecrotic lesion on the left side.

Conclusion

Osteonecrosis of the hip and knee present challenging cases for the orthopaedic surgeon. While many non-arthroplasty interventions have been described in the literature we have found little role for them in clinical practice. Core decompression may be used in small, pre-collapse areas of osteonecrosis especially

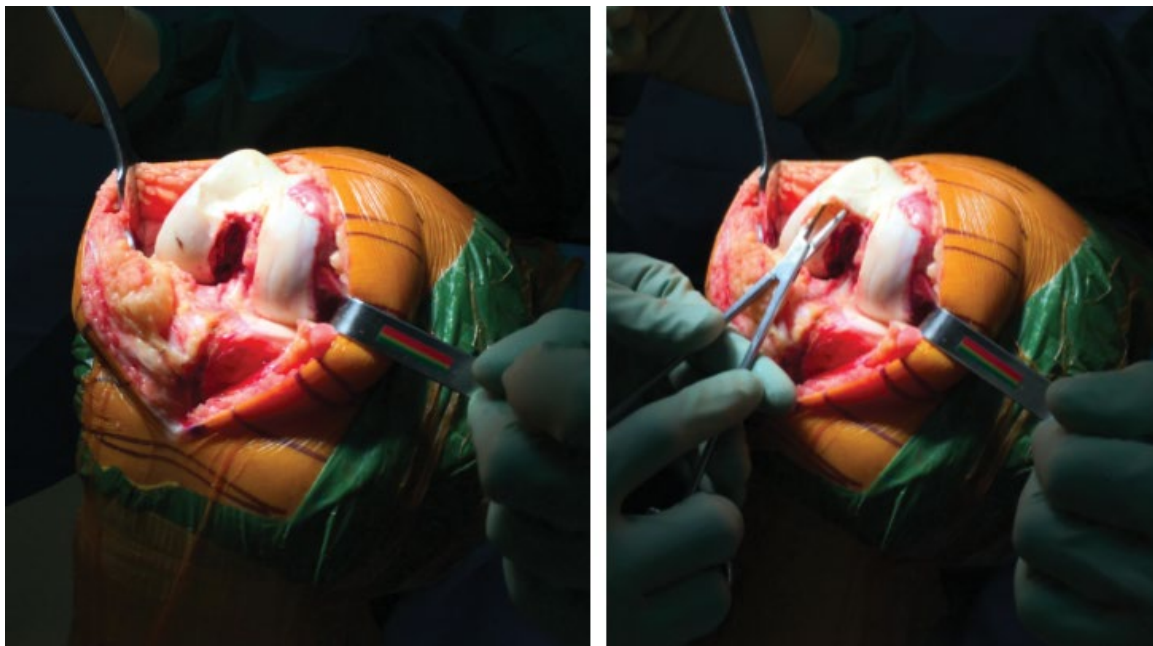


Figure 16: Intraoperative photographs of the right knee showing complete articular collapse of the femoral trochlea.

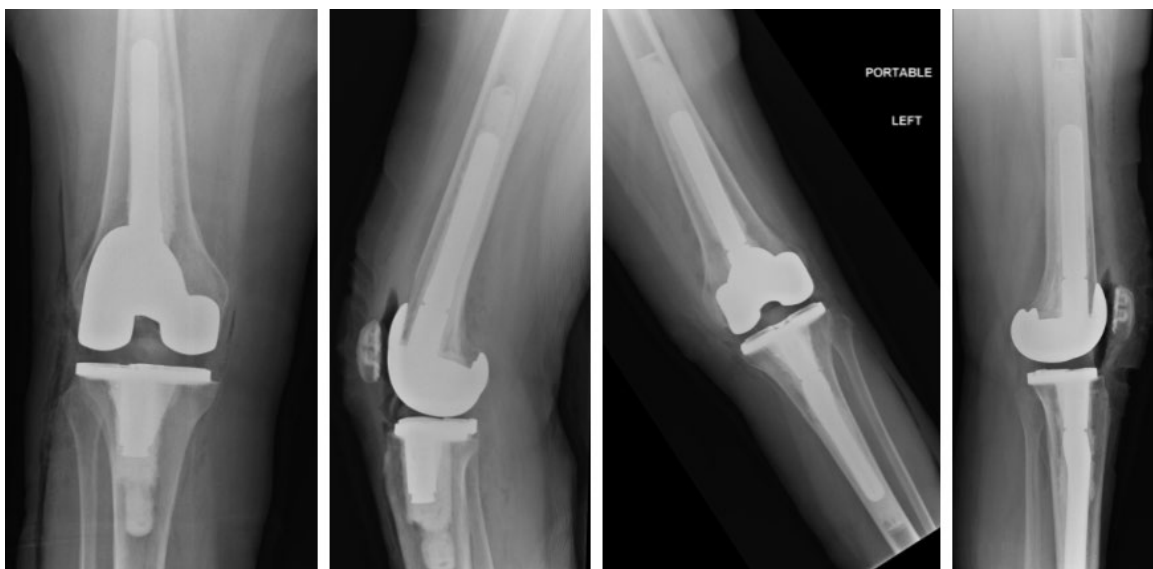


Figure 17: AP and lateral postoperative radiographs. Note that stemmed prosthesis we used in the R femur and Left Femur/Tibia in order to bypass all osteonecrotic lesions. Cement was also used for fixation in both knees.

if the involved bone has minimal involvement of the weight bearing surface. Arthroplasty has proven to be an excellent option for patients with osteonecrosis and debilitating pain and function. While special attention must be paid to these patients as they generally present younger than patients with degenerative arthritis and the native bone stock may be severely compromised, we have found that one can achieve excellent results with arthroplasty in the setting of osteonecrosis.

References

- Lieberman JR, Scaduto AA, Wellmeyer E (2000) Symptomatic osteonecrosis of the hip after orthotopic liver transplantation. *J Arthroplasty* 15: 767-771.
- Lieberman JR, Roth KM, Elsisy P, Dorey FJ, Kobashigawa JA (2008) Symptomatic osteonecrosis of the hip and knee after cardiac transplantation. *J Arthroplasty* 23: 90-96.
- Ficat RP (1981) Aseptic necrosis of the femur head. Preliminary remarks concerning staging: stage. *Acta Orthop Belg* 47: 239-241.
- Mont MA, Marulanda GA, Jones LC, Saleh KJ, Gordon N, et al. (2006) Systematic analysis of classification systems for osteonecrosis of the femoral head. *J Bone Joint Surg Am* 88: 16-26.
- Kerboul M, Thomine J, Postel M, Merle d'Aubigné R (1974) The conservative surgical treatment of idiopathic aseptic necrosis of the femoral head. *J Bone Joint Surg Br* 56: 291-296.
- Gillespy T 3rd, Genant HK, Helms CA (1986) Magnetic resonance imaging of osteonecrosis. *Radiol Clin North Am* 24: 193-208.
- Glickstein MF, Burk DL Jr, Schiebler ML, Cohen EK, Dalinka MK, et al. (1988) Avascular necrosis versus other diseases of the hip: sensitivity of MR imaging. *Radiology* 169: 213-215.

8. Ha YC, Jung WH, Kim JR, Seong NH, Kim SY, et al. (2006) Prediction of collapse in femoral head osteonecrosis: a modified Kerboul method with use of magnetic resonance images. *J Bone Joint Surg Am* 88: S35-S40.
9. Lieberman JR, Berry DJ, Mont MA, Aaron RK, Callaghan JJ, et al. (2003) Osteonecrosis of the hip: Management in the 21st century. *Instr Course Lect* 52: 337-355.
10. Sen RK (2009) Management of avascular necrosis of femoral head at pre-collapse stage. *Indian J Orthop* 43: 6-16.
11. Mont MA, Hungerford DS (1995) Non-traumatic avascular necrosis of the femoral head. *J Bone Joint Surg Am* 77: 459-474.
12. Wang CJ, Wang FS, Huang CC, Yang KD, Weng LH, et al. (2005) Treatment for osteonecrosis of the femoral head: Comparison of extracorporeal shock waves with core decompression and bone-grafting. *J Bone Joint Surg Am* 87: 2380-2387.
13. Glueck CJ, Freiberg RA, Sieve L, Wang P (2005) Enoxaparin prevents progression of stages I and II osteonecrosis of the hip. *Clin Orthop Relat Res* 435: 164-170.
14. Lai KA, Shen WJ, Yang CY, Shao CJ, Hsu JT, et al. (2005) The use of alendronate to prevent early collapse of the femoral head in patients with nontraumatic osteonecrosis. A randomized clinical study. *J Bone Joint Surg Am* 87: 2155-2159.
15. Chen CH, Chang JK, Lai KA, Hou SM, Chang CH, et al. (2012) Alendronate in the prevention of collapse of the femoral head in nontraumatic osteonecrosis: A two-year multicenter, prospective, randomized, double-blind, placebo-controlled study. *Arthritis Rheum* 64: 1572-1578.
16. Rajagopal M, Balch Samora J, Ellis TJ (2012) Efficacy of core decompression as treatment for osteonecrosis of the hip: a systematic review. *Hip Int* 22: 489-493.
17. Wei BF, Ge XH (2011) Treatment of osteonecrosis of the femoral head with core decompression and bone grafting. *Hip Int* 21: 206-210.
18. Keizer SB, Kock NB, Dijkstra PD, Taminiau AH, Nelissen RG (2006) Nelissen. Treatment of avascular necrosis of the hip by a non-vascularised cortical graft. *J Bone Joint Surg Br* 88: 460-466.
19. Berend KR, Gunneson EE, Urbaniak JR (2003) Free vascularized fibular grafting for the treatment of postcollapse osteonecrosis of the femoral head. *Bone Joint Surg Am* 85: 987-993.
20. Biswal S, Hazra S, Yun HH, Hur CY, Shon WY (2009) Transtrochanteric Rotational Osteotomy for Nontraumatic Osteonecrosis of the Femoral Head in Young Adults. *Clin Orthop Relat Res* 467: 1529-1537.
21. Abbas AA, Yoon TR, Lee JH, Hur CI (2008) Posttraumatic Avascular Necrosis of the Femoral Head in Teenagers Treated by a Modified Transtrochanteric Rotational Osteotomy: A Report of Three Cases. *J Orthop Trauma* 22: 63-69.
22. Hasegawa Y, Sakano S, Iwase T, Iwasada S, Torii S, et al. (2003) Pedicle bone grafting versus transtrochanteric rotational osteotomy for avascular necrosis of the femoral head. *J Bone Joint Surg Br* 85: 191-198.
23. Ortiguera CJ, Pulliam IT, Cabanela ME (1999) Total hip arthroplasty for osteonecrosis: Matched-pair analysis of 188 hips with long-term follow-up. *J Arthroplasty* 14: 21-28.
24. Kim SM, Lim SJ, Moon YW, Kim YT, Ko KR, et al. (2013) Cementless modular total hip arthroplasty in patients younger than fifty with femoral head osteonecrosis: Minimum fifteen-year follow-up. *J Arthroplasty* 28: 504-509.
25. Bedard NA, Callaghan JJ, Liu SS, Greiner JJ, Klaassen AL, et al. (2013) Cementless THA for the treatment of osteonecrosis at 10-year follow-up: Have we improved compared to cemented THA? *J Arthroplasty* 28: 1192-1199.
26. Issa K, Naziri Q, Maheshwari AV, Rasquinha VJ, Delanois RE, et al. (2013) Excellent results and minimal complications of total hip arthroplasty in sickle cell hemoglobinopathy at midterm follow-up using cementless prosthetic components. *J Arthroplasty* 28: 1693-1698.
27. Kim YH, Park JW, Kim JS (2012) Cementless metaphyseal fitting anatomic total hip arthroplasty with a ceramic-on-ceramic bearing in patients thirty years of age or younger. *J Bone Joint Surg Am* 94: 1570-1575.
28. Kim YH, Kim JS, Park JW, Joo JH (2011) Contemporary total hip arthroplasty with and without cement in patients with osteonecrosis of the femoral head: A concise follow-up, at an average of seventeen years, of a previous report. *J Bone Joint Surg Am* 93: 1806-1810.
29. Solarino G, Piazzolla A, Notarnicola A, Moretti L, Tafuri S, et al. (2012) Long-term results of 32-mm alumina-on-alumina THA for avascular necrosis of the femoral head. *J Orthop Traumatol* 13: 21-27.
30. JF Camp, CW Colwell Jr (1986) Core decompression of the femoral head for osteonecrosis. *J Bone Joint Surg Am* 68: 1313-1319.
31. Mont MA, Ragland PS, Etienne G (2004) Core decompression of the femoral head for osteonecrosis using percutaneous multiple small-diameter drilling. *Clin Orthop Relat Res* 429: 131-138.
32. Tabatabaee RM, Saberi S, Parvizi J, Mortazavi SM, Farzan M (2015) Combining Concentrated Autologous Bone Marrow Stem Cells Injection With Core Decompression Improves Outcome for Patients with Early-Stage Osteonecrosis of the Femoral Head: A Comparative Study. *J Arthroplasty* 30: 11-15.
33. Zalavras CG, Lieberman JR (2014) Osteonecrosis of the femoral head: evaluation and treatment. *J Am Acad Orthop Surg* 22: 455-464.
34. MacDessi SJ, Brophy RH, Bullough PG, Windsor RE, Sculco TP (2008) Subchondral fracture following arthroscopic knee surgery: A series of eight cases. *J Bone Joint Surg Am* 90: 1007-1012.
35. Chambers C, Craig JG, Zvirbulis R, Nelson F (2015) Spontaneous Osteonecrosis of Knee After Arthroscopy Is Not Necessarily Related to the Procedure. *Am J Orthop (Belle Mead NJ)* 44: E184-E189.
36. Mont MA, Marker DR, Zywiell MG, Carrino JA (2011) Osteonecrosis of the knee and related conditions. *J Am Acad Orthop Surg* 19: 482-494.
37. Mont MA, Baumgarten KM, Rifai A, Bluemke DA, Jones LC, et al. (2000) Atraumatic osteonecrosis of the knee. *J Bone Joint Surg Am* 82: 1279-1290.
38. Von Keudell A, Gomoll AH, Bryant T, Minas T (2011) Spontaneous osteonecrosis of the knee treated with autologous chondrocyte implantation, autologous bone-grafting, and osteotomy: a report of two cases with follow-up of seven and nine years. *J Bone Joint Surg Am* 93: e149.
39. Pape D, Seil R, Fritsch E, Rupp S, Kohn D (2002) Prevalence of spontaneous osteonecrosis of the medial femoral condyle in elderly patients. *Knee Surg Sports Traumatol Arthrosc* 10: 233-240.
40. Reddy AS, Frederick RW (1998) Evaluation of the intraos-

- seous and extraosseous blood supply to the distal femoral condyles. *Am J Sports Med* 26: 415-419.
41. Yamamoto T, Bullough PG (2000) Spontaneous osteonecrosis of the knee: The result of subchondral insufficiency fracture. *J Bone Joint Surg Am* 82: 858-866.
 42. Glueck CJ, Freiberg RA, Wang P (2014) Medical treatment of osteonecrosis of the knee associated with thrombophilia-hypofibrinolysis. *Orthopedics* 37: e911-e916.
 43. Claßen T, Becker A, Landgraeber S, Haversath M, Li X, et al. (2016) Long-term Clinical Results after Iloprost Treatment for Bone Marrow Edema and Avascular Necrosis. *Orthop Rev (Pavia)* 8: 6150.
 44. Mont MA, Tomek IM, Hungerford DS (1997) Core decompression for avascular necrosis of the distal femur: long term followup. *Clin Orthop Relat Res* 334: 124-130.
 45. Marulanda G, Seyler TM, Sheikh NH, Mont MA (2006) Percutaneous drilling for the treatment of secondary osteonecrosis of the knee. *J Bone Joint Surg Br* 88: 740-746.
 46. Mont MA, Rifai A, Baumgarten KM, Sheldon M, Hungerford DS (2002) Total knee arthroplasty for osteonecrosis. *J Bone Joint Surg Am* 84: 599-603.
 47. Myers TG, Cui Q, Kuskowski M, Mihalko WM, Saleh KJ (2006) Outcomes of total and unicompartmental knee arthroplasty for secondary and spontaneous osteonecrosis of the knee. *J Bone Joint Surg Am* 88: 76-82.