Review Article

Posttraumatic Avascular Necrosis After Proximal Femur, Proximal Humerus, Talar Neck, and Scaphoid Fractures

Abstract

Posttraumatic avascular necrosis (AVN) is osteonecrosis from vascular disruption, commonly encountered after fractures of the femoral neck, proximal humerus, talar neck, and scaphoid. These locations have a tenuous vascular supply; the diagnosis, risk factors, natural history, and treatment are reviewed. Fracture nonunion only correlates with AVN in the scaphoid. In the femoral head, the risk is increased for displaced fractures, but the time to surgery and open versus closed treatment do not seem to influence the risk. Patients with collapse are frequently symptomatic, and total hip arthroplasty is the most reliable treatment. In the humeral head, certain fracture patterns correlate with avascularity at the time of injury, but most do not go on to develop AVN due to head revascularization. Additionally, newer surgical approaches and improved construct stability appear to lessen the risk of AVN. The likelihood of AVN of the talar body rises with increased severity of talar injury. The development of AVN corresponds with a worse prognosis and increases the likelihood of secondary procedures. In proximal pole scaphoid fractures, delays in diagnosis and treatment elevate the risk of AVN, which is often seen in cases of nonunion. The need for vascularized versus nonvascularized bone grafting when repairing scaphoid nonunions with AVN remains unclear.

Posttraumatic avascular necrosis (AVN) is unique to anatomic locations with a tenuous blood supply to the articular segment. It is commonly diagnosed after fractures of the femoral neck, proximal humerus, talar neck, and scaphoid waist or proximal pole. In areas of avascularity after a fracture, osteocyte necrosis occurs and a repair process ensues initiated by revascularization at the periphery of the lesion. Some of the dead bone is resorbed, and new bone formation occurs onto the dead trabeculae and osteocyte lacunae, creating areas of sclerosis and lucency, as seen on the radiographs. If the resorptive process exceeds new bone formation, subchondral fracture and articular collapse can occur.1 AVN may be diagnosed on routine postoperative radiographs, showing sclerosis, lucency, or subchondral fracture and articular collapse. On a T1-weighted MRI, the signals from the fat cells in viable marrow elements are quite apparent.2 AVN may or may not be symptomatic or accompanied by collapse and is only associated with nonunion in the scaphoid. As the
presentation and treatment varies between anatomic sites, each of these locations is explored further.

**Femoral Neck Fractures**

**Vascular Supply**

Posttraumatic AVN of the femoral head is due to disruption to the femoral head blood supply by fracture displacement or from increased intracapsular hematoma pressure in the case of minimally displaced fractures. The vascular supply to the femoral head is complex (Figure 1). The dominant supply is from the ascending branch of the medial femoral circumflex artery (MFCA), which becomes the deep branch after giving off the inferior retinacular branch which also supplies the femoral head. The deep branch enters the joint posteriorly at the superior gluteus, terminating as the superior retinacular arteries (posterior superior nutrient), which are the primary vascular supply to the femoral head. Consistent anastomoses occur from the piriformis branch of the inferior gluteal artery to the MFCA near the obturator externus. This anastomosis and the intraosseous anastomoses between the retinacular vessels and subfoveal plexus likely become important in the setting of trauma, especially via the inferior retinacular (posterior inferior nutrient) artery, an MFCA branch that runs at a greater intracapsular distance from the bone. Follow-up metal artifact reduction multiaquisition variable resonance image combination–MRI at 3 and 12 months after displaced femoral neck fractures confirmed small (13% of the femoral head) anterior superomedial osteonecrotic lesions in 87% of cases, but none of these went on to collapse. This correlates with the traumatic injury to the superior retinacular branches that provide perfusion to this region.

**Risk Factors and Diagnosis**

Historically, AVN developed in up to 45% of displaced femoral neck fractures and in 20% of nondisplaced fractures (Figure 2). A more recent meta-analysis of 21 studies found an overall incidence of 17.3%. The risk of AVN continues to be higher in displaced fractures. In an analysis of 39 studies, Slobogean et al. found an AVN incidence of 14.7% versus 6.4% in displaced versus nondisplaced fractures in patients younger than 60 years. Conflicting results for age as an AVN risk factor are reported in the literature; it is likely not a major factor and is a reflection of the higher rate of displaced fractures in young patients.

Technical issues possibly related to AVN development include surgical timing, open versus closed reduction, the need for capsulotomy, and reduction quality. Although some early literature supported early emergent treatment (less than 6 or 12 hours) resulting in lower AVN rates, more modern meta-analyses have failed to prove an association between timing and rates of AVN. Modern approaches have also not been shown to influence AVN rates. A percutaneous capsulotomy for fractures treated with closed reduction carries the theoretical benefit of decompressing the tamponade effect. Capsulotomy has not been shown to decrease AVN rates in these fractures but can be considered in fractures treated with closed reduction. Based on early reports, the anatomic reduction has long been considered critical to minimizing AVN rates. Assuming more frequent perfect anatomic reductions are achieved with an open approach, no evidence-based support exists for lower AVN rates in open approaches versus closed reductions with percutaneous fixation, perhaps due to the risk of additional injury to the blood supply during open approaches. Unfortunately to answer this question, a multicenter study with postoperative three-dimensional imaging to evaluate reduction quality and report resulting AVN rates is lacking. However, the authors support that every effort should be made to achieve an anatomic reduction of these injuries.

Patients typically develop symptoms and radiographic changes of AVN 6 months to 2 years after their injury, but it has presented as late as 6 years postinjury. Symptoms typically are groin, lateral hip, and/or buttock pain. The Ficat classification (Table 1, Supplemental Digital Content, http://links.lww.com/JAAOS/A343) is used,
with a worse prognosis for those with any collapse.13 Posttraumatic lesions typically are anterosuperior, and the size of the lesion correlates with the symptoms and risk of collapse.9,14 Surprisingly, some patients with AVN, including collapse, are minimally symptomatic and may not require further surgical treatment. Haidu-kewych et al15 reported an AVN rate of 23%, but only 65% of these required surgery and 29% were asymptomatic. Jain et al16 found that the 16% of patients with AVN had equivalent functional outcome scores to those femoral neck fracture patients without AVN. Nikolopoulos et al12 reported that only 5 of 24 patients with AVN went on to have total hip arthroplasty (THA) at a mean follow-up of 4.7 years.

Treatment

Hip preserving strategies for non-traumatic osteonecrosis (ON) are most successful in precollapse cases which are rarely identified in post-traumatic cases; thus, very limited data are available on their use in the posttraumatic osteonecrotic hip. Precollapse AVN in traumatic cases is difficult to diagnose due to metal artifact and normal changes in bone density which may occur during the healing process. Metal artifact reduction multiacquisition variable resonance image combination (MAVRIC)–MRI can be successfully used to diagnose AVN in the presence of metallic implants.3 Patients with precollapse disease and smaller lesions have better functional outcomes, lower rates of progression, and less need for further surgery.11 Core decompression has the best results in precollapse nontraumatic cases when combined with bone marrow concentrate injection.17 Because symptomatic, posttraumatic AVN lesions are larger and often postcollapse, core decompression has a limited role in traumatic cases.11 Although some posttraumatic cases are embedded within larger series, data are inadequate to draw conclusions on a variety of procedures used in atraumatic cases including vascularized fibula grafting, transtrochanteric/ intertrochanteric rotational osteotomies, tantalum rods, hemi-hip resurfacing, or metal-on-metal resurfacing.9,11,14 THA is generally considered the definitive treatment of symptomatic, posttraumatic AVN. Treatment is complicated in those with fracture-related deformity, retained implants, or other previous failed salvage procedures. Traditionally, complication rates including dislocation, infection, and revision rates were higher for posttraumatic AVN arthroplasty groups, but more recent studies show impressive survival rates more similar to those for osteoarthritis.9,11,18

Future Directions

Augmented procedures are those in which a biologic procedure is added to the fixation construct. Luo et al loaded sliding hip screws with autologous bone graft, whereas Lazaro et al reported...
results of fixation constructs augmented with endosteal fibular allografts and Lin et al augmented cannulated screw fixation with percutaneous bone marrow injections. AVN rates were less than 6% in each study, but the largest was 30 patients, so further study is necessary to draw any conclusions. Additionally, measuring femoral head perfusion via drilling, intraoperative intracranial pressure monitoring, or dynamic contrast enhanced-MRI could help identify patients at high risk for AVN which may benefit from an augmented procedure.21

Proximal Humerus Fractures

Vascular Supply
The vascular anatomy of the proximal humerus and the typical fracture patterns that occur predispose the humeral head to AVN after trauma. The ascending branch of the anterior humeral circumflex artery provides a portion of the vascularity of the humeral head (Figure 3). While once thought to be the predominant blood supply, more recent studies have demonstrated that a significant portion of the humeral head derives perfusion from the posterior humeral circumflex artery.24 One or both these vessel systems may be injured traumatically or iatrogenically, leading to avascularity of the humeral head fracture fragment.

Diagnosis and Risk Factors
The diagnosis of humeral head AVN after a fracture is both clinical and radiographic (Figure 4). Increasing insidious pain, often with stiffness and crepitus, after a period of minimal symptoms, is a common clinical presentation. MRI can be useful in the diagnosis of early AVN after non-surgical treatment, but after fracture fixation with metallic implants, radiographic diagnosis and staging mainly relies on plain radiography. Typical findings include sclerosis, subchondral flattening, followed by osteolysis and collapse, with articular degeneration. When screws are present, slight collapse often leads to perforation of the screws through the articular surface. The Crues classification is often applied to the proximal humerus (Table 1) and can help guide treatment.

The orthopaedic surgeon is often faced with making early treatment decisions based on many factors, including the anticipated risk and severity of AVN. In general, AVN rates have been reported between 1% and 10%.25–27 Several specific fracture situations have been studied to assist with prognosis and decision making. For example, head-splitting fractures are a unique entity, and while once thought to be strongly predictive of AVN, recent studies with modern treatment methods have found relatively low rates of AVN.28 Similarly, fracture dislocations are not necessarily predisposed to developing AVN, and the risk appears to depend on residual capsular attachments to the humeral head, which may be intact in the dislocated head fragment.28–30 One factor that has been highlighted as a predictor for AVN is a short medial hinge on the head fragment.28 Interestingly, on long-term follow-up of these patients, it was found that intraoperative avascularity did not correlate with eventual AVN, implying that revascularization of the humeral head can occur and that eventual critical vascular disruption can develop later, potentially due to fracture instability.29
analysis found no significant difference in the rate of AVN between surgically and nonsurgically treated proximal humerus fractures.\textsuperscript{31}

The natural history of AVN is also critical to consider during early decision making. Several surgeon-controlled factors can substantially influence the risk of AVN. Gerber et al\textsuperscript{32} demonstrated that in patients with AVN but with anatomic fracture healing, shoulder elevation averaged 125° and Constant score was 65% of the contralateral shoulder, similar to hemiarthroplasty or complex fracture open reduction and internal fixation. Conversely, in the setting of a malunion, AVN led to significantly worse outcomes. This underscores the importance of initial reduction quality to minimize the negative impact of AVN in the setting of malunion.\textsuperscript{33} Regardless of the incidence and risk factors for AVN, patients with proximal humerus AVN often are minimally symptomatic but occasionally require additional surgery based on the presence and prominence of surgical implants. In a recent review of 166 surgically treated fractures, 4.8% were found to develop AVN at 22.9 months of follow-up, with 4 of 8 patients undergoing hardware removal and no arthroplasties.\textsuperscript{34}

**Treatment**

Initial treatment of proximal humerus AVN involves nonnarcotic analgesia, activity and work modifications with restricted overhead activity, and physical therapy. These modalities are continued based on patients’ symptoms and lifestyle and labor needs. In a series of 200 patients with humeral head AVN, the need for surgical treatment increased with later stage disease, and 78% of 37 patients with a traumatic etiology required arthroplasty within 3 years.\textsuperscript{35} For early-stage AVN, core decompression and resurfacing have been described, but typically after trauma, with implants present and altered anatomy, removal of implants or conversion to an arthroplasty is the next surgical procedure.\textsuperscript{34–37}

**Future Directions**

One factor related to AVN risk is the surgical approach. Historically, the deltopectoral approach has been used exclusively for surgical fracture fixation and no specific studies have attributed increased AVN risk to the deltopectoral approach. However, a critical blood supply to the humeral head from the ascending branch of the anterior humeral circumflex artery is at risk for iatrogenic injury given its location adjacent to the bicipital groove.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>Stage I</td>
<td>No changes visible.</td>
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<tr>
<td>Stage II</td>
<td>Sclerosis and areas of osteopenia.</td>
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<tr>
<td>Stage III</td>
<td>Subchondral fracture (crescent sign) with minimal depression of the articular surface.</td>
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<tr>
<td>Stage IV</td>
<td>Collapse of the joint surface and subchondral bone (fragmentation, loose bodies, secondary arthritis).</td>
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<tr>
<td>Stage V</td>
<td>Degeneration involves the glenoid.</td>
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Plate fixation through an anterolateral acromial (deltoid-splitting) approach may minimize iatrogenic vascular injury to this anterior blood supply and also may decrease iatrogenic injury to the perichondrium and periosteum of fracture fragments during reduction maneuvers. Several recent studies using this approach for locked plating of complex fractures have shown very low AVN rates, but clearly larger and randomized studies are necessary to determine the effect of surgical approach on AVN rates.\textsuperscript{27,38}

Initial construct stability may decrease AVN risks. Several modern fixation concepts appear to minimize AVN risk. The use of inferomedial “calcar” screws improves stability and decreases overall complication rates.\textsuperscript{39} Additionally, the use of fibular strut intraosseous allografts has increased in popularity and provides immediate improved stability. This technique has led to decreased mechanical failure rates and near zero AVN rates.\textsuperscript{27,40}

Fractures and dislocations of the talus can disrupt these extraosseous and intraosseous anastomotic networks, leading to AVN. To avoid iatrogenic contributions to AVN, the surgical dissection should avoid the deltoid ligament, the inferior talus by the tarsal canal, and the dorsalis pedis anteriorly.

**Diagnosis and Classification**

Although AVN of the talus can occur after talar body fractures, talar neck fractures, and pantalar dislocations, 90\% of talar AVN cases are after a talar neck fracture.\textsuperscript{42} Accordingly, the Hawkins classification is the most well-established talus fracture classification and is specific to talar neck fractures (Figure 6). It predicts...
the occurrence of AVN, with fractures and dislocations of increasing severity corresponding with increased AVN incidence.42,43

A 2015 meta-analysis noted a recent decrease in the rate of AVN after a talar neck fracture, with studies after 2000 having a lower incidence of AVN than those done before 2000. This decrease was thought to be secondary to an improvement in treatment methods. For all studies, this meta-analysis identified the ON rate as 10% for Hawkins type I, 27% for type II, 53% for type III, and 48% for type IV.43

However, a 2014 study of 80 patients proposed a modification to the Hawkins classification, differentiating the type II group into those with a subluxated subtalar joint (IIA) and those with a dislocated subtalar joint (IIB). No AVN occurred in type I and IIA injuries; AVN did occur in 25% of type IIB, 41% of type III, and 33% of type IV.44

No classification of talar body AVN exists to guide treatment. After a fracture, patients typically are evaluated with serial radiographs. On early radiographs (6 weeks), the presence of a talar dome subchondral lucency on an AP or mortise view, known as the Hawkins sign (Figure 7), reliably indicates that the talar dome is perfused and that the patient will not develop AVN. In a 2007 study, it was found to be 100% sensitive, with 57% specificity, meaning that many patients without a Hawkins sign still did not develop AVN.45 Radiographic features such as sclerosis, irregularity of the talar dome, and talar dome collapse are consistent with AVN (Figure 8). Detailing the location of the AVN and the presence of collapse are particularly important for the clinician to track over time, because lesions without collapse have the potential for revascularization.42,44,46

MRI and bone scans can also diagnose AVN when radiographs are inconclusive. The use of titanium implants and/or a metal artifact reduction MRI may improve MRI diagnostic accuracy.47 A bone scan is another option for diagnosis. As the osteoblasts in the bone necrose, their uptake of the radiopharmaceutical technetium-99 decreases.42 Although both modalities are accurate in their diagnostic capability, the precise timing for a bone scan or MRI is not well established. An MRI before 2 months after injury is considered too early, because there is extensive edema throughout the talus.2

**Treatment**

A variety of nonsurgical and surgical treatment options exist for patients with AVN; however, critical outcome studies are lacking. Historically, in patients in whom avascular changes
were noted early, prolonged non-weight bearing (up to 1 to 2 years after the injury) was advocated to help patients avoid collapse of the dome and facilitate revascularization. However, due to the impracticality of this restriction and its failure to be validated, this prolonged restriction is no longer commonplace. Once AVN is clearly established, the clinician has to discern whether the patient is symptomatic. Patients can have avascular changes in the talus and be asymptomatic, thus obviating the need for further treatment. For symptomatic patients, several surgical treatment options are available, but the literature does not clearly support any one procedure. Fortunately, conservative treatment is often successful with orthotics, bracing, treatment, stretching, physical therapy, and pharmacologic agents. In patients who have failed conservative treatment, surgical procedures are considered, with the presence or absence of collapse being particularly important. Patients who have AVN of the talus without collapse are candidates for core decompression. This encourages revascularization of the affected area after drilling. Long-term data are available on this procedure in atraumatic cases with a high percentage of good to excellent results. Several reconstructive procedures are available for patients with collapse (Figure 8). They are divided into two main categories: joint sacrificing and joint sparing. Joint sacrificing procedures include partial or total takedown and arthrodeses. The takedown has fallen out of favor due to its poor results. A successful arthrodesis requires sufficient healthy bone and should create a plantigrade foot. Depending on the size and location of the necrosis within the talus, the surgeon can choose from the following arthrodeses: talonavicular, tibiotalar, subtalar, Blair tibiotalar, tibioatlecty, tibiocalcaneal, and pantalar (triple). The incidence of secondary procedures for AVN and/or degenerative changes after talar neck fracture is 18%, with subtalar arthrodesis being the most common, followed by tibiotalar and pantalar. Joint sparing procedures are an appealing alternative to avoid the disability of arthrodesis. Case series documenting their outcomes show promise but are extremely limited in scope and number. The options include allograft replacement, vascularized autograft, and arthroplasty. Although distraction arthroplasty is a described option for ankle arthritis and has been suggested as a potential treatment of talar AVN, no reports of its use for talar AVN exist. Results for posttraumatic AVN cases are limited, and case series for ankle arthroplasty have noted a high complication rate, with subsidence of the prosthesis and complex regional pain syndrome being noted frequently, 55% and 22%, respectively.

Future Directions

There is considerable room for improvement in the care of the patient with talar AVN with many important unanswered questions. Currently, the Hawkins classification helps us predict which patients will develop AVN, but we are unable to predict which patients will be symptomatic from AVN. In terms of treatment, as core decompression shows promise as a technique for patients without collapse, an established way of identifying these patients early may be of potential benefit. Furthermore, outcome data on numerous AVN surgical procedures are lacking to drive evidence-based treatment decisions.

Scaphoid

Vascular Supply

The primary blood supply to the scaphoid flows in a retrograde pattern from the dorsal carpal branch of the radial artery (Figure 9). Scaphoid waist fractures and proximal pole fractures can disrupt this blood supply, resulting in impaired flow to the proximal pole. This reduced flow occurs across a spectrum, and it is important to differentiate between an ischemic proximal pole that maintains normal morphology and a necrotic, collapsed proximal pole.

Diagnosis

The benchmark for diagnosing scaphoid AVN is histologic analysis for signs of necrosis and bone remodeling, but the need for preoperative and real-time intraoperative assessments for AVN has traditionally been emphasized to help create surgical treatment plans. Preoperative and intraoperative attempts to make a definitive diagnosis of proximal pole AVN have limitations but are best evaluated with a combination of preoperative MRI with...
MRI with gadolinium appears superior to MRI without contrast in diagnosing AVN with greater sensitivity, specificity, and accuracy, compared with nonenhanced MRI. Intraoperative evaluation to assess for punctate bleeding of the proximal pole is commonly performed but may not correlate with preoperative MRI findings because a recent study found no correlation between preoperative MRI and intraoperative bleeding or histologic findings of AVN. In the study by Rancy et al, histologic AVN confirmation on intraoperative biopsy did not correlate with preoperative MRI or intraoperative bleeding.

**Avascularity and Fracture Healing**

The presence of proximal pole avascularity does not appear to definitively correlate with scaphoid healing potential. Dawson et al reported on 32 acute scaphoid fractures treated with cast immobilization. MRI with gadolinium was obtained on all, and no relationship was observed between nonunion (12%) and MRI evidence of avascularity. Interestingly, an inverse relationship exists between MRI enhancement and fracture healing. Günal et al achieved a 92% union rate in 13 cases of scaphoid nonunion, with evidence of avascularity on either preoperative gadolinium-enhanced MRI or intraoperative assessment of punctate bleeding from the proximal pole. The aim of treatment is primarily to prevent the development of posttraumatic arthrosis, or SNAC wrist, which invariably occurs with untreated proximal pole AVN and scaphoid nonunion. Nonunion with AVN and collapse, particularly in the setting of previous surgical treatment, are negative prognostic factors. When minimal or no arthrosis is present with the maintained architecture of the proximal pole, surgical technique includes aggressive curettage of the nonunion site proximally and distally with robust autogenous bone grafting. In another study, preoperative gadolinium-enhanced MRI assessment of proximal pole vascularity did not correlate with subsequent scaphoid union. A more recent study also demonstrated that scaphoid proximal pole vascularity did not correlate with healing rates or the time to union. These studies suggest that pre- and intraoperative evidence of proximal pole ischemia does not translate to compromised scaphoid healing potential. Although nonunion can occur without AVN, AVN occurs almost exclusively in the setting of nonunion. Anatomic reduction with bone grafting and stable fixation can reestablish sufficient blood flow to allow for nonunion healing independent of preoperative proximal pole perfusion in most cases.

**Treatment**

Surgical fixation of displaced scaphoid fractures is indicated to prevent nonunion, humpback deformity, carpal collapse, AVN, and subsequent development of scaphoid nonunion advanced collapse (SNAC) wrist. Surgery is performed to revascularize the proximal pole and maintain carpal kinematics by restoring scaphoid length and carpal alignment. In the commonly encountered setting of AVN with nonunion, the goals of treatment are to obtain fracture union and reestablish perfusion to the proximal pole. The aim of treatment is to prevent the development of posttraumatic arthrosis, or SNAC wrist, which invariably occurs with untreated proximal pole AVN and scaphoid nonunion. Nonunion with AVN and collapse, particularly in the setting of previous surgical treatment, are negative prognostic factors. When minimal or no arthrosis is present with the maintained architecture of the proximal pole, surgical technique includes aggressive curettage of the nonunion site proximally and distally with robust autogenous bone grafting and rigid internal fixation. Bone grafting is performed to augment union rates, and selection of the ideal graft (nonvascularized, vascularized, and free vascularized) to achieve this goal is controversial. Although previous literature supported the role of vascularized bone graft (VBG) for scaphoid nonunions with AVN, more recent literature suggests that non-VBG may...
be equally efficacious in achieving union.\textsuperscript{61,62}

There is limited evidence to support the routine use of VBGs in the setting of scaphoid nonunion with AVN. A meta-analysis of seven studies of 64 patients all treated before 1999 found 88% union for VBG versus 47% for non-VBG treatment of proximal pole AVN, but no direct comparisons were made and only one study used CT scans to assess fracture healing.\textsuperscript{61} However, a recent systematic review of 48 studies including 1,602 patients found no significant difference in union rates between VBG (92%) and non-VBG (88%). Overall, union rate of 91% was noted for proximal pole nonunions with AVN. In this systematic review, the authors found no evidence of superior union rates with any particular technique.\textsuperscript{62} In cases where SNAC wrist has developed, salvage procedures typically are performed. These include proximal row carpectomy or scaphoid excision and mid-carpal fusion for radioscaphoid arthritis (stage I SNAC). When scaphocapitate arthrosis develops (stage II), scaphoid excision and mid-carpal fusion are performed. In cases that have progressed to more pervasive periscaphoid arthrosis (stage III), a total wrist arthrodesis is recommended.

**Future Directions**

Free vascularized medial femoral condyle (MFC) bone grafting is an emerging treatment of scaphoid nonunion with promising results. A recent study reported higher union rates and faster union with MFC compared with structural iliac crest bone graft and 1,2-intercompartmental supraretinacular artery vascularized grafts. MFC may have a particularly valid role in the treatment of scaphoid nonunions and AVN with poor prognostic factors such as carpal collapse and previous surgery.\textsuperscript{63}

**Summary**

Posttraumatic AVN may be diagnosed on routine postoperative radiographs or with MRI, often using metal artifact reduction sequences. The diagnosis on plain radiographs can be potentially subjective in cases with only increased density but no irregularities or collapse. Some of these patients are asymptomatic as are some with collapse, though collapse is associated with a worse prognosis. In displaced femoral neck fractures, evidence is inadequate to support that emergent surgery within 12 hours lowers AVN rates. Achieving an anatomic reduction closed or open decreases the rates of AVN. Small osteonecrotic lesions are very common, whereas larger lesions are more likely to progress to symptomatic collapse, but not all patients with collapse are symptomatic. Precollapse lesions are rarely identified but can be considered for a variety of procedures used on atraumatic ON hips. Symptomatic postcollapse lesions are most reliably treated with total hip arthroplasty. AVN after proximal humeral fractures may be from traumatic injury to the humeral head blood supply or iatrogenic from the
surgical approach and fracture mobilization, though no clear increase in AVN rates is present after surgical treatment. To decrease AVN rates, a quality reduction with a stable construct is paramount with minimal iatrogenic vascular insult including consideration of an anterolateral deltoïd-splitting approach. Talar body AVN risk increases with injury severity via the Hawkins fracture classification. Many patients are successfully managed with nonsurgical measures, but others with refractory symptoms may require arthrodesis. Proximal pole AVN after scaphoid fracture can be difficult to assess, especially in the presence of fixation hardware. The initial presence of proximal pole avascularity does not appear to negatively affect scaphoid healing potential, but when AVN develops, it is nearly always accompanied by nonunion and the literature is inconclusive regarding the role of various surgical procedures to treat it and achieve union. Recent studies have demonstrated higher union rates with screw fixation combined with nonvascular, vascular, or free VBG. Much work remains to be done in determining the optimal treatment of patients with symptomatic, posttraumatic AVN after these fractures.

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