CHAPTER **24**

AVASCULAR NECROSIS OF THE TALUS

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Avascular necrosis (AVN) of the talus is a topic that should be understood by any practitioner treating pathology of the foot and ankle. Its pathogenesis is such that if it is not diagnosed early and treated properly, it may result in sequelae with a high degree of morbidity. The literature on AVN is extensive, and illustrates the fact that it is a complex, progressive disease that defies simple categorization. It is most easily defined as the death of bone cells secondary to complete interruption or a significant decrease in the vascular supply to bone.[1] Synonyms for the process include ischemic necrosis, osteonecrosis and aseptic necrosis.[1-3] The latter is actually a misnomer, and will subsequently be discussed in the section on etiology.

It is important to discern between the etiology and pathogenesis of AVN in that a working knowledge of both contribute to the diagnosis of the disease. The etiology of AVN encompasses the factors or conditions that predispose the patient to develop the pathology. Its significance often lies in the information rendered from the patient's history. The pathogenesis of AVN is the physiologic process resulting in the production of a bone infarct, and is diagnostically significant with respect to the changes seen on various imaging modalities.

ETIOLOGY

The determinant underlying all classifications or predisposing conditions is the interruption of blood supply to the bone. The etiology of AVN may be subdivided into mechanisms and predisposing factors. The mechanisms can involve the anatomic location of the circulatory compromise or vascular disruption. These are academic classification systems whose component parts overlap (Table 1).

Extraosseous arterial compromise includes such phenomena as trauma, atheromata, and microemboli. Intraosseous arterial obstruction is seen with fat emboli and in systemic disease processes, such as rheumatoid arthritis and systemic lupus erythematosus (SLE). Intraosseous extravascular compromise considers bone, in this instance the talus, as a closed compartment. An increase in marrow pressure causes a decrease in perfusion to the osteocytes and creates a "marrow compartment syndrome." This can occur in infectious processes where the infectious by-products increase the intraosseous pressure.

Vascular disruption is another etiologic mechanism. Traumatic vascular disruption is self-explanatory, and occurs most frequently with talar neck fractures. Disruption due to vascular compression refers to an increase in soft tissue volume and/or pressure which may occur either intraosseously or extraosseously, and again, is most commonly a sequela of infection. Intraluminal obstruction occurs with any embolic process.

The majority of literature dealing with AVN reflects the disease process as it effects the femoral head.[6,7] The factors predisposing a patient to developing AVN in general, and of the femoral head are numerous, and beyond the scope of this paper. Bone infarcts in the talus have been reported in patients with sickle cell anemia, Gaucher's disease (i.e. familial splenic anemia), hematogenous osteomyelitis, suppurative arthritis of the ankle joint, and in patients undergoing renal dialysis.[6,7] The factors most often responsible for predisposing a patient to developing AVN of the talus are: systemic lupus erythematosus (SLE), exogenous administration of corticosteroids, infection, and trauma.

Table 1

ETIOLOGY - MECHANISMS

Location of Circulatory Compromise[4]	Vascular Disruption[5]
Extraosseous Arterial	Traumatic
Intraosseous Arterial	Vascular Compression
Intraosseous Extravascular	Intraluminal Obstruction

Although SLE is associated with AVN, it is not thought to be a primary cause for its development. SLE is an autoimmune disease with a diverse array of clinical manifestations including: Raynaud's phenomenon, vasculitis/vasculopathy, hyperlipidemia, and antiphospholipid syndrome (a disease process associated with venous and arterial thrombosis). Patients with SLE are often treated with systemic steroid therapy.[8] It is believed that, as a result of these factors, patients with SLE develop AVN at lower and/or less frequent doses of steroids compared to patients taking steroids for other conditions such as rheumatoid arthritis, chronic obstructive pulmonary disease, and status-post organ transplantation.[5,9-11]

Infection is also not a primary cause of AVN. This explains why the term aseptic necrosis is a misnomer. Infection results in an inflammatory response which ultimately results in the direct destruction of bone. AVN is a bone infarction caused by compromising the blood supply to the bone. The increase in pressure from the local accumulation of fluids and exudates can result in either intraosseous or extraosseous vascular compression, which disrupts the blood supply to the osteocytes.[10] Therefore, infection by way of the inflammatory process and its sequelae, secondarily results in the development of AVN.

Corticosteroid induced AVN was first described by Pietrograde.[12] Currently, the use of systemic corticosteroids is the leading cause of subchondral AVN in adults.[11] Steroid use is rarely, however, the sole factor involved in the development of AVN, but is implicated in causing AVN with certain systemic diseases.[4] Several mechanisms have been proposed for steroid-induced AVN. These include a steroid-induced hypercoaguable state, fatty emboli, osteoporosis, and increased intramedullary lipocyte size.

The length of time of corticosteroid usage reportedly necessary to produce AVN varies markedly in the literature from one week to several months.[11] Most authors now agree that it is necessary to take large doses of steroids over several months, as it takes time for the pathologic changes to occur, specifically the increase in intramedullary lipocyte size resulting in an increase in intramedullary pressure.[5,9,10] The reason why certain patients have this apparent increased sensitivity to steroids remains unknown.[11]

Of the predisposing factors, the only one which directly results in the production of AVN is trauma, which damages the vessels supplying the talus. The primary blood supply to the body of the talus is the artery of the tarsal canal, a branch of the posterior tibial artery. Secondary supplies are from the deltoid artery and the artery of the sinus tarsi, branches of the artery of the tarsal canal and anterior tibial artery, respectively.[15,16] Although the current literature reports a case of an isolated AVN to the head of the talus,[17] AVN almost exclusively involves the body of the talus. In addition to the tenuous blood supply of the talus, the majority of the bone is covered with articular cartilage, a relatively avascular tissue.[5,15] The Hawkins' classification for talar neck fractures has classically been used to predict the incidence of onset of AVN, and is based upon the severity of the fracture and concomitant dislocations of the peritalar joint complexes.[18] Subsequently, various studies have reported incidences which can be summarized as follows: Type I: 0-13%; Type II: 20-50%; Type III: 84-100%.[18-21]

Type IV talar neck fractures as first described by Canale and Kelly are reported too infrequently in the literature to accurately quantitate the likelihood of the development of subsequent AVN.[22] Szyszkowitz devised a system of classifying fractures of the entire talus, not just the neck, and related them to the onset of various complications including AVN. In his classification scheme, only general statements concerning the onset of AVN are proposed. For example, in fractures of the proximal neck or body, necrosis seldom occurs, whereas in fractures of the proximal neck with ankle and/or subtalar dislocation, AVN nearly always occurs.[23] In general, AVN is reported to occur in 50% of talar neck fractures.[19,20,23,24] Daniels[25] reported a composite incidence of 37%. A number of authors report a significantly lower incidence of AVN following talar neck fractures.[26-29] A lower incidence of 15% to 16% has been attributed to early open reduction with internal fixation with protection of the blood supply from the deltoid artery. Talar neck fractures are not the only injury pattern where AVN of the talus occurs. Goldner et al.[30] report a 33% onset of talar body AVN with late segmental collapse in Gustillo Type 3 open subtalar joint dislocations.

Idiopathic AVN of the talus has been reported, but it is rare.[31] According to Kenzora, idiopathic AVN occurs due to an accumulated stress theory, where conditions associated with AVN produce a bone cell sickness. As the sickness progresses or other decompensating factors such as steroid use are added, a critical stress is reached, and AVN occurs.[4,32]

PATHOGENESIS

A bone infarct occurs when there is death of the marrow fat cells. Of the three cellular components of bone, hematopoietic, osteocytes, and marrow fat cells, the latter is least sensitive to the effects of anoxia, and takes up to five days to die after the onset of anoxia. Bone death from anoxia occurs in stages. First, there is an interruption of intracellular enzymes. This is followed by an alteration or cessation of intracellular metabolic activity. In the third stage there is irreversible disruption or dissolution of intracellular nuclear and cytoplasmic ultrastructure resulting in cell death.[6]

Understanding the pathogenesis of bone infarction, facilitates identification of an infarct via various imaging modalities. When considering the three dimensional infarct, there are four zones which need to be considered.[6] An outer rim of normal tissue surrounds an inflammatory zone of active hyperemia. The hyperemia produces a localized area of osteopenia and a relative increase in radiographic density of the innermost zone of infarcted bone. The central zone of cell death and its contiguous zone of ischemic injury are both contained within the zone of hyperemia. Repair of the infarcted tissue begins along the outer perimeter of the ischemic tissue, thereby creating the hyperemic zone of granulation tissue. It is this progressive margin between ischemic and viable tissue that is sensitive to detection with the use of magnetic resonance imaging.[4,33]

DIAGNOSIS

AVN of the talus is diagnosed using a combination of information gained from the history and physical exam, and from various imaging studies. As previously mentioned, the history gives clues as to the etiologic factors predisposing a patient to develop the disease. In addition to trauma, systemic illnesses, infection, and the use of glucocorticoids, a history positive for rearfoot or ankle surgery may also result in AVN of the talus. The information extracted from the history is more valuable than that from the physical examination because symptoms experienced by patients with AVN are nonspecific. Patients have pain with active or passive range of motion of the subtalar or ankle joint, tenderness, mild erythema, local edema, and may have joint clicking or locking, depending on the stage of the disease.[1] Of the three types of bone infarct: medullary, intracortical, and subchondral, only the latter occurs in the talus. This type of infarct develops in a predictable pattern. The insidious onset of symptoms parallels those seen in patients with osteoarthritis.[11]

AVN has been classically diagnosed by radiography. The radiographic changes correlate with bone resorption secondary to repair, and are not detectable until 6 to 12 weeks after the insult has occurred.[3,4,6,34-36] A talus with AVN presents with sclerotic densities involving all or part of the body. In cases where part of the body of the talus is spared, it usually occurs medially, in the area of blood supply from the deltoid artery.[37] Bobechko and Harris[38] reported that radiographic sclerosis seen in avascular bone is due to viable bone being deposited on necrotic bone, osteoporosis of surrounding bones from disuse and the hyperemia of repair, and calcification of necrotic bone marrow further increasing bone density. When attempting to diagnose AVN radiographically, it is important to understand that the talar body is normally more radiodense compared to the talar neck, and that the overlapping shadows of the malleoli also increase the density of the body compared to surrounding structures.[39]

Hawkins' sign is a subchondral radiolucency in the dome of the talus which appears 6 to 8 weeks after injury, suggesting bone resorption during revascularization.[18] The presence of Hawkins' sign is a good indicator that the vascularity of the talus is intact, and that AVN will not occur.[29] Hawkins' sign is often seen medially on the anteroposterior radiograph of the ankle, and again correlates with the blood supply of the deltoid artery.[40] It can also been seen on the lateral view but is made more difficult by the presence of the malleoli. It is important to note that the absence of Hawkins' sign is not a reliable indicator that AVN will occur.[19]

Although scintigraphic studies are more sensitive than radiography in the detection of AVN, they are not specific. There will be a decreased uptake immediately after the injury which reflects the disruption of the vascular supply to the bone. It must be remembered though, that if vascular disruption is secondary to trauma, the cold area in the talar body could be obscured by the increased uptake at the fracture site. Weeks to months after the injury, when the bone begins to revascularize, an increase in uptake may be expected.[4,11,34] This is

not particularly useful from a diagnostic standpoint. Despite this, it may have some prognostic value for predicting the onset of AVN in injury patterns consistent with its development. Canale and Kelly have used scintigraphy to determine the length of treatment following the onset of AVN. They found increased uptake in the entire talar body, representing revascularization, and continued treatment until the scan began to show a decrease in activity.[19] Should the practitioner have a high degree of suspicion for the development of AVN based on a positive history or injury pattern along with bone scan uptake patterns consistent with AVN, they may choose to use more aggressive therapy or perform a more specific diagnostic modality.

Computed tomography (CT) has also been investigated as a diagnostic modality. The vast majority of research has been done on AVN of the femoral head. CT can be used to assess the subtle changes in trabecular patterns and cortical integrity. Overall though, the effectiveness of computed tomography is currently questionable.

Conversely, magnetic resonance imaging (MRI) is currently the gold standard for the early and most specific non-invasive diagnosis of AVN. Coleman et al.[41] reported MRI to be sensitive to detecting AVN of the hip in asymptomatic patients when radiographic studies were negative. Henderson[42] reported a case however, where serial MRI scans failed to show AVN of the talus in a patient with a negative Hawkins' sign. Although rare, false negatives of AVN have been reported.[43-45] The hallmark MRI finding is the appearance of a reactive interface representing a layer of fibromesenchymal tissue at the margin between viable and infarcted medullary bone.[46,47] This presentation relates to the previously described three dimensional layers of a bone infarct. The images correlate with the stages of the disease process.[7] Early in the process, a line or arc of decreased signal intensity can be seen on T1-weighted images. This represents granulation tissue replacing fat and is the transition between normal and ischemic bone.[4,33,46,48] On T2-weighted images, this line or arc demonstrates a bilaminar appearance characterized by an outer border of decreased signal intensity, and an inner layer of increased signal intensity, the latter of which represents the zone of hyperemia.[4,33,47] Mitchell et al. referred to this as the "Double Line Sign," which is diagnostic for AVN.[41,47,49]

Brody et al.[50] found that on T1-weighted images changes could be seen within the first week of devascularization. By days 16 and 23, areas of patchy and more homogeneous decreased signal intensity could be seen, respectively. The detection of changes to the fat cells themselves is variable. However, the changes cause an increase in vascularity, inflammation and the production of a granulation tissue interface. As discussed in the section on pathogenesis, MRI is very sensitive in detecting the presence of this granulation tissue interface.

COMPLICATIONS

The complications of AVN of the talus are summarized in Table 2 The production and revascularization of osteonecrotic bone can occur without symptoms or morbid changes, and are usually diagnosed incidentally. Bone is unique in that an infarction of osseous tissue can completely revascularize and repair itself.[11] Therein lies the mechanism responsible for producing the morbid complications associated with AVN of the talus. The hyperemia is an inherent part of the repair process. As the bone repairs itself there is a transient structural weakness involving the trabeculae supporting the subchondral bone in the dome of the talus. If the biomechanical stresses of weight bearing are superimposed upon the talus during this stage of hyperemia and compromised structural integrity, the trabeculae may fracture, and the collapse of the subchondral bone and the overlying articular cartilage may ensue.

Table 2

COMPLICATIONS OCCURRING SECONDARY TO AVN OF THE TALUS

None
Collapse of articular surfaces
Ankle or subtalar DJD
Intra-articular loose bodies
Infection

Catto described a phenomenon known as "late segmental collapse," in which there is a fracture along the sclerotic bone rim with collapse of the entire remaining infarct into the zone of revascularization.[11] DeLee found that one in three patients with total body avascular necrosis experienced collapse of the ankle or subtalar joint.[22] Degenerative joint disease of the ankle and/or subtalar joint, and the production of intra-articular loose bodies are a direct result of the collapse of the articular surfaces. Infection is rare, but is mentioned since any necrotic tissue can provide an environment which can serve as a nidus for infection.[11]

TREATMENT

There are two primary goals of treatment. The first is to prevent the onset of the previously mentioned complications. Early diagnosis translates into a better prognosis.[4] The key is to have a high level of suspicion for AVN by fully understanding the etiologic factors and the pathogenic process. This knowledge will guide the practitioner to order the appropriate studies in a timely manner. Once AVN has been diagnosed, the practitioner must choose a treatment plan appropriate to the patient which allows the second goal of treatment to occur, namely, revascularization of the talus. Full revascularization of the talus may take two or more years, and correlates with the previously described dense sclerotic talar body.[20,35] If the revascularization occurs in a slow homogenous way, collapse of the talar dome does not occur. When revascularization occurs rapidly in a patchy distribution there is collapse along the interface of avascular trabeculae and the invading vascular granulation tissue. Penny and Davis felt this is due to the devitalized bone being reabsorbed faster than the new bone is being deposited, resulting in structural weakness.[20]

Most authors currently recommend protected weight-bearing. Controversy arises with respect to the degree of protection as well as the time for therapy. Borner,[51] Zilch,[52] and Kazar[53] believe non-weight bearing should be instituted for 6 to 18 months when treating AVN. Canale and Kelly found that in their patients, those who remained non-weight bearing for at least 8 months had the most favorable results. Those patients treated by means of protected weight-bearing with a patellar tendon-bearing brace also had good outcomes, but less so. Adelaar,[54] DeLee,[22] and Pennal[21] also agree that protected weight-bearing should be instituted. This is significant because these authors feel that it is both unreasonable and impractical to prescribe such a long treatment of non-weight bearing. O'Brien et al.[55] and Hawkins[18] believe that weight bearing does not necessarily result in collapse. Hawkins[18] and Gillquist et al.[56] state that even if collapse occurs, it is well tolerated by most patients, and rarely requires reconstructive surgery. This conclusion is not supported by many authors, including Monkman et al.,[57] who indicate that late segmental collapse following AVN is a poor prognostic sign. Additional conservative therapy involves a modification of activity level. In symptomatic patients, a decrease in activity will usually be self-imposed because of pain. Dictating specific changes in activity levels or exercise routines, such as a change over to swimming from running, will decrease the potential risk of articular collapse.

There are currently no reports in the literature that address the use of electric bone stimulation as a modality for the treatment of AVN of the talus. There are however, studies that address AVN of the femoral head. The rationale for using electric bone stimulation is based on the loss of structural integrity during the repair process of AVN, and the increase in bone formation that is induced with electric stimulation.[58] Electric bone stimulation has also been shown to decrease osteoclastic bone resorption in vitro.[59] Since structural integrity is compromised because of an increase in the amount of bone resorption compared to new bone formation, this could potentially be a viable treatment for subchondral AVN of the talus. Aaron et al. showed a decreased incidence of clinical and radiographic progression of femoral head AVN in patients treated with pulsed electric magnetic fields (PEMF) bone stimulation.[60] PEMF and implanted direct current bone stimulation have been shown to be successful in decreasing the progression of AVN, while electrical bone stimulation via capacitive coupling has apparently not added any therapeutic value.[58] Steinberg et al. found no indication that the addition of capacitive coupling gave better results than decompression and grafting alone.[61] Finally, Mont and Hungerford accurately state that electric bone stimulation for the treatment of AVN of the femoral head is currently still experimental, and that it has not yet been

approved by the Food and Drug Administration for the treatment of AVN.[48] However, as a result of the positive data concerning the use of PEMF and implantable direct current bone stimulation, studies evaluating the use of such modalities for the treatment of AVN of the talus are justified.

Theories justifying surgical intervention as a primary treatment for AVN of the talus are based upon the premise of creeping substitution revascularization from surrounding well-vascularized bone into avascular areas, in this case within the talus. Bone grafting with either subtalar or triple arthrodesis has been attempted and advocated by many authors.[62-67] Boyd[62] theorized, however, that the revascularization process occurring from an arthrodesis procedure would extend only 3 to 4 millimeters into the body of the talus. Supporting Boyd's hypothesis, various authors have proven that primary arthrodesis is unsuccessful at causing more rapid revascularization of an osteonecrotic talus.[18-20] Penny and Davis[20] recommended a Blair fusion consisting of advancement of a corticocancellous strut from the distal tibia into the neck of the talus, once the talar body has been removed. As a result of the occasional development of a painful pseudarthrosis, modifications to stabilize and increase compression were developed.[67,68] Tibiocalcaneal arthrodesis has also been recommended, and found to be more effective than ankle fusions or talectomies.[19,21,31] Unfortunately, this procedure results in an average leg length discrepancy of 1.25 inches.[69] Arthrodesis procedures are currently recommended for patients with symptomatic, secondary sequelae from late segmental collapse.[3,5] It is important to reiterate that not all patients with collapse of the talar dome are symptomatic. Numerous authors have found that late segmental collapse does not always guarantee a painful, afunctional result.[19,24,56,57,70] Canale and Kelly[19] found that two-thirds of their patients with AVN needed no secondary surgical procedures.

CONCLUSION

AVN of the talus is a disease process that any practitioner treating pathology of the foot and ankle must consider in their differential diagnosis of rearfoot and ankle pain. The key is to have a high index of suspicion based upon details gained from a comprehensive history. Then by understanding the natural progression of the disease, the clinician can recognize the characteristic changes as they present on various imaging studies. Though isolated incidents of false-negative studies have been reported, magnetic resonance imaging is currently the gold standard for early and accurate diagnosis. Once the diagnosis is made, prompt aggressive treatment consisting of protected weight-bearing or non-weight bearing is necessary in order to prevent the onset of morbid complications, namely the collapse of the talar dome.

Surgical treatment should be reserved for two conditions associated with AVN of the talus. The first addresses decreasing the chances of developing AVN with early open reduction and internal fixation of fracture dislocations patterns predisposing to AVN. Secondly, should late stage collapse occur rendering the patient symptomatic, arthrodesis of the affected joints is indicated.

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