Exhibit Selection

Everything Achilles: Knowledge Update and Current Concepts in Management

AAOS Exhibit Selection

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Abstract: Achilles tendon pathology is common and affects athletes and nonathletes alike. The cause is multifactorial and controversial, implicating biological, anatomical, and mechanical factors. A variety of conditions characterized by Achilles tendon inflammation and/or degeneration can be clinically and histologically differentiated. These include insertional Achilles tendinopathy, retrocalcaneal bursitis, Achilles paratenonitis, Achilles tendinosis, and Achilles paratenonitis with tendinosis. The mainstay of treatment for all of these diagnoses is nonoperative. There is a large body of evidence addressing treatment of acute and chronic Achilles tendon ruptures; however, controversy remains.

Anatomy and Function

The Achilles tendon, composed of fibers from the gastrocnemius and soleus muscles, is the body’s strongest and thickest tendon. Beginning at the midportion of the calf, the Achilles tendon internally rotates 90° as it courses distally in the posterior aspect of the leg, with the soleus contribution medial to that of the gastrocnemius1,2. It then inserts into the middle third of the flat surface on the posterior aspect of the calcaneal tuberosity.

The Achilles tendon does not have a true synovial sheath; instead, it has a paratenon composed of flexible connective tissue that allows for 1.5 cm of tendon gliding with activity3,4. The paratenon and tendon are innervated by nerves from attached muscles and small fascicles from local cutaneous nerves—in particular, the sural nerve5. The sural nerve crosses the tendon approximately 11 cm proximal to the calcaneal tuberosity and 3.5 cm distal to the musculotendinous junction6, making it vulnerable to iatrogenic injury7, particularly with minimally invasive repair techniques8. The Achilles tendon is relatively hypovascular, especially at its midportion9,10; in contrast, the paratenon is a highly vascular structure11 (Figs. 1 and 2).

The Achilles tendon is part of a musculotendinous unit that spans three joints, producing knee flexion, tibiotalar flexion, and subtalar inversion (exerting a medial pull on the calcaneus). Loads as great as 12.5 times body weight are placed on the tendon during running, contributing to its high rate of injury8,12. Male individuals have a higher maximum tendon rupture force and stiffness, in part due to a larger cross-sectional area, than female individuals. Younger individuals have a higher maximum tendon rupture force and lower stiffness than their older counterparts11,12.

Disclosure: None of the authors received payments or services, either directly or indirectly (i.e., via his or her institution), from a third party in support of any aspect of this work. None of the authors, or their institution(s), have had any financial relationship, in the thirty-six months prior to submission of this work, with any entity in the biomedical arena that could be perceived to influence or have the potential to influence what is written in this work. Also, no author has had any other relationships, or has engaged in any other activities, that could be perceived to influence or have the potential to influence what is written in this work. The complete Disclosures of Potential Conflicts of Interest submitted by authors are always provided with the online version of the article.
Etiology and Risk Factors for Injury

The Achilles tendon is the most commonly ruptured tendon, and the incidence of injuries is increasing. Injury causes are often multifactorial, related to overuse combined with mechanical overload and host susceptibility. Both internal (anatomical conditions or systemic diseases) and external (training conditions and equipment) influencing factors have been described. Internal risk factors include decreased blood supply with advancing age, corticosteroid or fluoroquinolone use, and male sex. External risk factors may include high-intensity plyometric exercises, training on unfamiliar surfaces, and the use of improper footwear.

Achilles Tendon Pathology (Table I)

Insertional Achilles tendinopathy, as its name suggests, occurs secondary to degeneration of tendinous fibers at their insertion on the calcaneus. The condition is associated with inflammatory arthropathies, corticosteroid use, diabetes, hypertension, obesity, gout, and use of fluoroquinolone antibiotics. Reported prevalence rates range from 6.5% to 18% in runners and 9% in dancers. Insertional Achilles tendinopathy is thought to be an inflammatory process, at least in its initial phase. It may be associated with a Haglund deformity, which is a prominent posterosuperior aspect of the calcaneal tuberosity that causes an overlying bursitis and tendinopathy. In its chronic form, insertional tendinopathy may no longer be an inflammatory process.

Insertional Achilles tendinopathy is primarily a clinical diagnosis, and patients will often report a history of pain and stiffness at the back of the calcaneus in the morning that worsens with activity and severe pain the day after exercising. Commonly, the Achilles tendon insertion will be painful at the midportion of the posterior aspect of the calcaneus. In addition, an enlarged posterior border of the calcaneus may be palpable. MRI (magnetic resonance imaging) will reveal focal microtears and can be useful in confirming the diagnosis.

For most cases of insertional Achilles tendinopathy (85% to 90%), nonoperative treatment consisting of a short period of immobilization followed by gradual integration of reduced load-bearing activities and physical therapy provides pain relief. Prolonged periods of immobilization should be avoided. Platelet-rich plasma (PRP) therapy can be considered for refractory cases, although evidence supporting its use is scant. Surgical treatment to remove the diseased portions of the tendon, an osseous prominence irritating the tendon, and the inflamed bursa is rarely indicated, unless the condition becomes chronic.
and debilitating. In these cases, lengthening of the Achilles tendon may be necessary, depending on the extent of involvement of the tendon and its insertion site.

Retrocalcaneal bursitis occurs when the bursa anterior to the Achilles tendon becomes inflamed, hypertrophied, and adherent to the underlying tendon. This inflammation may be associated with degenerative changes within the tendon substance. The bursa becomes irritated as it is compressed in the recess between the anteroinferior aspect of the Achilles tendon and the posterosuperior aspect of the calcaneus during ankle dorsiflexion.

Patients will present similarly to those with insertional Achilles tendinopathy. The clinical diagnosis can be aided by the two-finger squeeze test, which is performed by applying medial and lateral pressure anterior to the Achilles tendon, often eliciting pain in patients with retrocalcaneal bursitis. MRI may demonstrate a partial Achilles tendon rupture, peritendinous thickening, tendinosis, or ossification.

As with insertional tendinopathy, treatment is largely nonoperative. NSAIDs (nonsteroidal anti-inflammatory drugs), modification of training regimens, and eccentric strengthening have been shown to be effective. A heel lift to relieve pressure on the bursa can be prescribed. Immobilization for a brief period may be helpful. When indicated, surgical intervention includes debridement of the degenerative tendon insertion, decompression of the bursa, resection of the osseous prominence, and repair of the tendinous insertion. Up to 50% of the tendon attachment can be debrided without high risk of rupture.

Noninsertional tendinopathy can be divided into three histopathologic entities: paratenonitis, paratenonitis with tendinosis, and tendinosis alone. The term tendinopathy encompasses the broad spectrum of inflammation levels that may be present histologically in a given injury. The terminology for these disorders can be confusing, and different descriptive paradigms have been advocated to provide clarification.

Paratenonitis involves inflammation of only the paratenon; grossly, the paratenon appears thickened and adherent to normal tendon. Histologic evaluation classically shows inflammatory cell infiltration and capillary proliferation in the paratenon or peritendinous tissue. Clinical signs include swelling, pain, crepitus, local tenderness, and warmth. Paratenonitis with tendinosis is defined as paratenon inflammation present in the setting of intratendinous degeneration, thought to be secondary to a failed healing response. Histology shows the same findings as paratenonitis with additional loss of tendon collagen, fiber disorganization, and scattered vascular ingrowth. However, there is typically no intratendinous inflammation at this juncture. The clinical signs and symptoms are similar to paratenonitis, often with the additional finding of a palpable asymptomatic tendon nodule. Finally, isolated tendinosis can occur without paratenonitis. It is thought that this condition results from an early inflammatory infiltrate followed by a failed healing response and ultimately tendon degeneration. Histologically, the tendon has a noninflamed appearance with collagen degeneration, hypocellularity, local necrosis, areas of calcification, and minimal vascular ingrowth. Clinically, tendinosis is often painless and without swelling.

Age is a primary culprit in the development of noninsertional tendinopathy; age-related degeneration of the Achilles tendon was reported in 34% of tendons at autopsy. Morphologic changes over time include a decrease in the maximum diameter and density of collagen fibrils and a reduction in vascularity. These changes result in fewer tensile strands and eventual tendon degeneration and rupture.

The diagnosis of noninsertional tendinopathy is made largely through the history and physical examination. The examination should be performed with the patient prone. The physician should pay particular attention to muscle girth and the position of the foot and ankle at rest. Tenderness, swelling, warmth, nodularity, crepitation, and substance defects may be noted. Ultrasound and MRI may aid in diagnosis and detection of incomplete tendon ruptures.

Noninsertional tendinopathy usually responds to conservative treatment. Rest, ice, compression, modifications in training regimens (e.g., implementing eccentric exercise), NSAIDs, heel lifts or shock-absorbing orthotics, and night splints for heel cord tightness can all be utilized. Moderate evidence suggests that extracorporeal shock wave therapy may be effective; it should be considered for refractory tendinopathy cases. There is no evidence to support local injection of any pharmacologic substance intended to speed recovery. Corticosteroid injections in particular have a demonstrated lack of clinical efficacy and are associated with a potential compromise of tendon integrity; they should therefore be avoided.
TABLE I Inflammatory and Degenerative Disorders of the Achilles Tendon*

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Insertional Tendinopathy</th>
<th>Retrocalcaneal Bursitis</th>
<th>Paratenonitis</th>
<th>Tendinosis</th>
<th>Paratenonitis with Tendinosis</th>
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<tr>
<td>Definition</td>
<td>Inflammation caused by tendon degeneration at calcaneal insertion</td>
<td>Inflammation of retrocalcaneal bursa; may lead to Achilles tendon degeneration</td>
<td>Inflammation of only paratenon; may thicken and adhere to tendon</td>
<td>Infratendinous degeneration due to atrophy</td>
<td>Paratenonitis with associated degenerative changes in Achilles tendon</td>
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<tr>
<td>Histology</td>
<td>Inflammatory cell infiltrate, edema, mucoid degeneration, ruptured collagen bundles</td>
<td>If Achilles tendon is involved, degenerative changes may be seen</td>
<td>Inflammatory cell infiltrate and capillary proliferation in paratenon; normal Achilles tendon</td>
<td>Collagen degeneration, fiber disorientation, hypocellularity, scattered vascular ingrowth, occasional local necrosis</td>
<td>Paratenonitis findings plus collagen degeneration, fiber disorientation, and scattered vascular ingrowth in tendon</td>
</tr>
<tr>
<td>Clinical signs and symptoms</td>
<td>Pain and stiffness over posterior calcaneus, worse with activity</td>
<td>Pain anterior to Achilles tendon and superior to calcaneus; positive two-finger squeeze test</td>
<td>Swelling, pain, tenderness, crepitus, warmth over tendon</td>
<td>Palpable tendon nodule that is asymptomatic; swelling of tendon sheath is absent</td>
<td>Similar to paratenonitis plus palpable asymptomatic tendon nodule</td>
</tr>
<tr>
<td>Treatment</td>
<td>Mainly conservative: ice, NSAIDs, brief immobilization, gradual weight-bearing activities</td>
<td>Mainly conservative: avoidance of external pressure, NSAIDs, activity modification</td>
<td>Mainly conservative: rest, ice, NSAIDs, modification in training (eccentric exercises); brisement</td>
<td>Mainly conservative: rest, ice, NSAIDs, modification in training (eccentric exercises); brisement</td>
<td>Mainly conservative: rest, ice, NSAIDs, modification in training (eccentric exercises); brisement</td>
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*Although they differ at the cellular level, the various inflammatory and degenerative disorders of the Achilles tendon often present with similar historical and physical examination findings. In all conditions, initial treatment should be conservative and surgery should be reserved for refractory cases.

Brisement has proven to be helpful in treatment of paratenonitis and tendinosis. This procedure consists of taking a dilute anesthetic and injecting it into the tendon sheath to break up adhesions.

Surgical treatment for paratenonitis should be considered for chronic cases resistant to a conservative program. Tendoscopy involves removal of locally thickened, inflamed paratenon and release of the plantaris tendon. For open surgery, a medial longitudinal incision is made and full-thickness flaps are developed. The thickened paratenon is excised and the crural fascia is closed to decrease scarring. For resistant symptomatic tendinosis, the paratenon is evaluated and excised if needed, degenerative tendon material is debrided, and defects are repaired. A full repair may be required, depending on the amount of tendon excised and on the size of the defect, as described in subsequent sections on rupture repair.

Achilles Tendon Rupture

Achilles tendon ruptures generally occur in healthy, vigorous, "young" adults with a previous history of prodromal calf or heel pain. However, the injury can also occur in older, sedentary patients. Ninety to 100% of these injuries occur during active, forceful, and sometimes unexpected foot plantar flexion. Proposed mechanisms for acute rupture include (1) pushing off with the weight-bearing foot while extending the knee, as would occur in a sprinter at the start of a race (53% of injuries); (2) sudden, unexpected dorsiflexion of the ankle, for example during an unexpected step into a hole (17%), and (3) violent ankle dorsiflexion of a plantar flexed foot, as can occur in a fall from a height (10%).

The Achilles tendon most commonly ruptures 3 to 4 cm proximal to its insertion on the calcaneus. This typical location is likely related to the presence of an avascular zone 2 to 6 cm above the tendinous insertion. Kannus and Józsa found that 864 (97%) of the 891 spontaneously ruptured tendons that they examined histologically exhibited degenerative changes. Factors potentially contributing to Achilles tendon rupture include age-related degeneration, poor tendon vascularity, gastrocnemius-soleus dysfunction, sex, changes in training pattern, previous injury, and choice of footwear. Some authors argue that ruptures occur in a poorly conditioned individual secondary to a sudden overloading of the musculotendinous unit rather than to an underlying pathologic process.
Ruptures have become more common over the past two decades, with a reported annual incidence of eighteen per 100,000 people. The reported mean age of patients with Achilles tendon rupture ranges from thirty-seven to 43.5 years. When a patient ruptures the tendon on one side, there is a 6% to 26% chance that they will also rupture the contralateral tendon. Within the U.S., basketball and racquetball sports account for more than half of all injuries. Males have a higher incidence of rupture than females.

Patients presenting with acute Achilles tendon rupture often report a sudden snap in the heel region at the time of injury followed by pain with ankle plantar flexion and difficulty with weight-bearing activity. Thirty-three percent of patients report having had prodromal symptoms prior to sustaining a rupture. Acute ruptures typically present with the presence of a palpable gap and a positive Thompson test on physical examination. The Thompson test has been shown to be positive in 96% to 100% of acute ruptures. This can occur because of a large hematoma disguising the tendon defect, retained plantar flexion power secondary to extrinsic foot flexors, or a false-negative Thompson test if accessory ankle flexors are inadvertently squeezed. The Thompson test is positive in only 80% of chronic cases.

Radiographs are obtained in cases of suspected Achilles tendon pathology to rule out concomitant fractures or calcific tendon changes. The physician should look for disruption of the normal triangular pre-Achilles fat pad (Kager triangle) on a lateral radiograph. Additionally, disruption of the Achilles tendon complex can uncommonly result in avulsion of a large osseous fragment from the calcaneus. Other imaging modalities such as ultrasonography and MRI are more sensitive and specific with respect to confirming the clinical diagnosis. Ultrasonography is inexpensive, is quickly obtained, and can be used for dynamic assessment (Fig. 4), for example, during evaluation in the emergency room. MRI is more expensive and cannot be used for dynamic testing. However, benefits of MRI evaluation include its ability to assess the condition and orientation of the torn fibers and the extent of retraction and gapping. MRI is also useful for detecting partial ruptures.

**Treatment of an Acute Rupture**

The optimal treatment remains controversial, and both operative and nonoperative treatments have distinct advantages and disadvantages (Table II). Figure 5 presents a treatment algorithm for management of all forms of pathology discussed in this article.

Nonoperative treatment begins with an initial two-week period of non-weight-bearing and immobilization in a cast or boot with an elevated heel wedge; this is to permit hematoma consolidation. At this point, continued immobilization in a cast may be prescribed, or a functional bracing protocol may be commenced. Compared with strict immobilization in a cast, functional bracing has been associated with an increased range of motion, an earlier return to the pre-injury level of activity, and greater comfort. Several protocols for functional bracing exist. These generally consist of fitting a patient with an orthosis that can be fixed in various degrees of plantar flexion, with progressive decreases in plantar flexion on a week-by-week basis. Additionally, the patient is allowed to perform plantar flexion exercises with the use of variable-resistance bands with unrestricted plantar flexion; however, the dorsiflexion stop is maintained.

**TABLE II Comparison of Operative and Nonoperative Treatment of Acute Achilles Rupture**

<table>
<thead>
<tr>
<th></th>
<th>Operative Treatment</th>
<th>Nonoperative Treatment</th>
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<tbody>
<tr>
<td><strong>Advantages</strong></td>
<td>Improved functional outcome (e.g., return to sport).</td>
<td>No complications related to surgery.</td>
</tr>
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<td></td>
<td>Faster return to work.</td>
<td>Lack of scar</td>
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<td></td>
<td>Decreased rerupture rate (depending on protocol)</td>
<td></td>
</tr>
<tr>
<td><strong>Disadvantages</strong></td>
<td>Invasive (major and minor complications of surgery, especially wound-related).</td>
<td>Greater likelihood of patient dissatisfaction.</td>
</tr>
<tr>
<td></td>
<td>Greater cost</td>
<td>Adherence to functional rehabilitation protocol necessary for good outcome</td>
</tr>
</tbody>
</table>

*The choice between operative and nonoperative management of acute Achilles tendon rupture remains controversial, with certain advantages and disadvantages ascribed to each modality.
Strict immobilization is also an option. As initially described by Lea and Smith, strict immobilization for eight weeks followed by the use of a heel lift (with concurrent institution of strengthening exercises) was associated with an 11% rerupture rate at twenty-six months of follow-up.\(^\text{49}\) Strict immobilization was often reserved for older, less active individuals as it resulted in substantial loss of power, strength, and endurance.\(^\text{37,40,48}\)

Functional bracing has gained popularity recently, with several reviews favorably comparing functional bracing with surgical repair.\(^\text{57,58}\) Soroceanu et al. completed a stratified analysis and determined that there was no significant difference in the rerupture rate between operative and nonoperative treatment when similar functional postoperative mobilization protocols were employed.\(^\text{59}\) A recent randomized controlled trial had similar conclusions; no significant difference in rerupture rate was observed when rehabilitation following acute Achilles tendon rupture consisted of early accelerated tendon loading used as either postoperative or nonoperative treatment.\(^\text{60}\)

Despite increasing evidence of successful nonoperative management, operative treatment remains popular. This is likely due to reported differences in functional outcomes between nonoperative and operative treatments. Following nonoperative treatment, 78% of patients with functional bracing reported no pain, 56% had no weakness, 55% reported no stiffness, and 98% returned to full employment. However, only 37% returned to the same level of sport.\(^\text{56,61,62}\)

Early functional mobilization following repair has been shown to yield improved tendon healing.\(^\text{63}\) Numerous surgical techniques have been described, including open repair (with or without augmentation), percutaneous repair, and limited open repair. It is crucial to optimize the condition of the skin by delaying operative intervention until acute swelling has subsided; no negative impact on functional outcomes has been found with a surgical delay of up to thirty days following rupture.\(^\text{39,40}\) Some advocate draping the contralateral leg free to assess the resting position of the uninjured ankle, allowing for an intraoperative assessment of repair site tension.

When performing an open repair, the surgical approach can be midline, medial, or lateral. A longitudinal incision along the medial aspect of the tendon is preferred to avoid injury to the sural nerve and lesser saphenous nerve plexus (Fig. 6). A midline incision may increase pressure from the repaired tendon; thus, a medially based incision may be less likely to slough. Sharp incision and dissection of the crural fascia and...
remaining paratenon is performed, as these layers can be closed after repair completion. A multilayer closure helps to reduce skin tension and helps to prevent the formation of postoperative tendon adhesions to skin. Stitch patterns described include the modified Bunnel, Kessler, Krackow, and triple-bundle techniques. A biomechanical study found that a triple-bundle repair technique was the strongest suture repair\(^8\). Key elements of open repair are bringing tendon edges together under appropriate tension (as compared with the contratralateral side), using nonabsorbable suture, and performing meticulous soft-tissue management with minimal retraction of the soft tissues (to prevent wound complications, which are commonplace). Augmentation of a primary repair is sometimes utilized, with evidence for and against its use. Augmentation can be performed by weaving the plantaris tendon into the repair, using a flexor tendon graft, or performing a turnudown graft of the gastrocnemius fascia when necessary.

There are proponents of less-invasive surgical repair techniques, including percutaneous and mini-open repairs. A percutaneous repair is defined as a procedure without direct exposure of the tendon rupture site. Two Level-II studies have compared percutaneous repair with open repair\(^65,66\). Those studies revealed no significant difference in the number of patients who returned to functional activities, in the ability to perform activities of daily living, or in patient satisfaction. There was also no difference in rerupture rate compared with open repair\(^65,66\). However, patients treated with the percutaneous technique had a lower incidence of wound breakdown or healing complications\(^65\). The percutaneous technique requires posterior stab incisions, both medially and laterally, increasing concern for sural nerve injury\(^67,68\). Mini-open repair is also commonly used. This approach involves smaller incisions that allow direct visualization of the ruptured tendon ends. Studies have found that patients who underwent mini-open repair returned to walking, stair climbing, and sports in significantly less time compared with those undergoing standard open repair\(^69,70\). There was no difference in the number of reruptures compared with open repair\(^70\). Mini-open repair also had significantly fewer severe wound infections and superficial infections than open repair, as well as fewer minor surgical site infections\(^70\).

Postoperative rehabilitation protocols vary widely. Components include a short (less than two-week) period of immobilization to minimize wound complications. Early protected weight-bearing with early mobilization is recommended, as it has demonstrated initial benefits in the time to return to activities, including work, sports, and normal walking\(^71,72\). The use of a modified splint or orthotic with early mobilization at two to four weeks is recommended, as a higher rate of return to sport activities at twelve months has been demonstrated\(^72\). Patients can usually return to light jogging at three to six months\(^76\).

Treatment of a Chronic Rupture

Chronic Achilles tendon rupture is a fundamentally different entity than acute rupture. There is no single rule for determining what is “chronic,” with time points from four weeks to 2.5 months described in the literature\(^77\). However, chronic ruptures demonstrate poor healing potential and often require surgical treatment, usually with augmentation and/or tendon transfer.

Options for surgical treatment of chronic tears include direct repair, local tissue transfer, soft-tissue augmentation, and augmentation with synthetics or allografts. Direct repair may be achieved for gaps that are ≤3 cm in length after debridement\(^78\). However, because of tissue retraction over a period of weeks or months, defects of such small size are rare in the setting of chronic rupture\(^76,77\). The flexor hallucis longus (FHL) tendon is commonly used to augment the strength of a direct repair and supplies vascularity to the tendon ends. The FHL tendon is harvested through a medial arch incision and then introduced into the posteromedial incision. Once the Achilles tendon and muscle belly are reflected, the deep posterior compartment fascia is incised and released, exposing the FHL muscle belly and tendon.

Augmentation with soft tissue can be done using medial and lateral aponeurotic fascial turnudown flaps, plantaris tendon, sliding V-Y advancement of the gastrocnemius-soleus complex aponeurosis, and fascia lata\(^79,80\). For gaps of 3 to 5 cm, V-Y advancement flaps are particularly useful\(^80,81\). Acellular human dermal tissue matrix has also been used for augmentation with good results\(^82\). Achilles tendon allografts have been used to treat large defects (approximately 10 cm); however, results are limited to case reports.

Conclusion

Achilles tendon pathology is common in athletes and nonathletes alike. The cause is often multifactorial, involving biomechanics and tendon degeneration. Nonoperative treatment is the mainstay for Achilles tendon inflammation and degeneration. There is a large body of evidence addressing treatment of acute and chronic Achilles tendon ruptures; however, controversy remains. Orthopaedic surgeons must understand the various options available for augmentation or reconstruction.

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