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Advances in the Diagnosis and Treatment of Insertional Achilles Tendinopathy: A Comprehensive Review

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Abstract: ***Insertional Achilles tendinopathy is a chronic condition characterized primarily by degenerative changes at the insertion site of the Achilles tendon. It commonly affects athletes and middle-aged individuals, with typical clinical manifestations including pain at the posterosuperior aspect of the heel, morning stiffness, and start-up pain. Although various treatment methods are available, there is no unified standard of care, and the efficacy of each approach varies considerably. This review comprehensively examines the anatomy and pathological changes of the Achilles tendon, as well as the diagnosis and treatment of insertional Achilles tendinopathy, with a particular emphasis on therapeutic strategies, aiming to provide insights for clinical management.***

Keywords: Insertional Achilles tendinopathy, Achilles tendon insertion, Achilles tendinopathy, Conservative treatment, Surgical treatment.

1. Introduction

Insertional Achilles tendinopathy (IAT) is a common cause of chronic heel pain, accounting for approximately 24% of patients presenting with heel pain-related symptoms [1,2]. IAT involves tendinopathic changes at the insertion of the Achilles tendon onto the calcaneal tuberosity [1], and often manifests as pain, swelling, and limited mobility near the tendon insertion, with symptoms typically localized within 2 cm of the enthesis [3]. The etiology of IAT involves both intrinsic and extrinsic factors. Extrinsic factors, particularly overuse injuries, play a major role in the development and progression of IAT. Intrinsic factors may include Haglund's deformity, retrocalcaneal bursitis, aging, and chronic systemic conditions [4]. These factors often interact, accelerating disease progression [5]. IAT frequently affects both athletic populations and sedentary middle-aged and elderly individuals [6], with symptoms tending to worsen over time, significantly impacting work and daily life. This article aims to systematically review current advances in the understanding of IAT, with a focus on its pathological mechanisms, diagnostic approaches, and treatment strategies, thereby providing a reference for clinical practice.

2. Anatomical Basis and Pathological Changes

The Achilles tendon is the largest and strongest tendon in the human body [7]. It inserts onto a bony prominence on the posterosuperior aspect of the calcaneus, known as the calcaneal tuberosity. From a microstructural perspective, to achieve a gradual transition from soft to hard tissue and dissipate mechanical stress, the enthesis consists of four distinct tissue layers: tendon tissue, fibrocartilage, calcified fibrocartilage, and bone [8,9]. The retrocalcaneal bursa and the subcutaneous calcaneal bursa are located near the Achilles tendon insertion, helping to reduce excessive friction during tendon movement [10]. The pre-Achilles fat pad also facilitates smooth gliding of the tendon.

The blood supply to the Achilles tendon is relatively poor [11]. The midportion of the tendon has a zone of hypovascularity, approximately 2–6 cm above the calcaneal insertion, which is the most common site for midportion Achilles tendinopathy. Although the insertion site has a somewhat better blood supply, it remains relatively weak, resulting in inefficient clearance of metabolic waste and limited nutrient delivery, which impairs its healing capacity after injury. The tendon is richly innervated, particularly in the insertion area, enabling persistent pain signaling in response to local inflammation or injury [12].

Under microscopic examination, IAT is primarily characterized by tissue degeneration, structural disruption, and failed repair attempts, rather than significant inflammatory cell infiltration. It represents a degenerative condition of the tendon structure resulting from repetitive stress, featuring collagen disorganization, abnormal angiogenesis and neural proliferation, and alterations in extracellular matrix composition. This indicates that the condition is fundamentally a degenerative process rather than simple inflammation [13]. Furthermore, the pathology often extends to surrounding structures such as the bursae and the pre-Achilles fat pad, leading to bursitis and fat pad inflammation. Calcification and even enthesophyte formation may occur within the degenerative tissue or at the tendon insertion site, exacerbating pain and structural damage [14].

3. Clinical Manifestations and Diagnosis

The pain in IAT is precisely localized to the posterosuperior aspect of the calcaneus. In the early stages, it is often sharp, while in the chronic phase, it typically presents as a dull ache, soreness, or stiffness. The most characteristic symptom is severe pain taking the first few steps after waking up in the morning or following prolonged rest, which usually alleviates after walking a short distance. Pain can also be provoked or worsened by specific activities or movements and tends to improve with rest. Physical examination may reveal a thickened and hardened tendon at the insertion site, accompanied by significant tenderness [15].

Radiographic examination (X-ray) is primarily used to evaluate bone structures. It can detect abnormalities such as Haglund's deformity, a shadow indicating retrocalcaneal bursitis, tendon calcification, or enthesophyte formation. It is also valuable for excluding other conditions like fractures or tumors [16]. Ultrasonography allows for dynamic assessment and can evaluate tendon thickening and the presence of abnormal blood flow signals at the insertion. Magnetic resonance imaging (MRI) is considered the gold standard for diagnosing IAT [17]. It provides comprehensive information on soft tissues and anatomical structures. Findings suggestive of IAT on MRI include tendon thickening and increased signal intensity on T1-weighted images, while high signal intensity on T2-weighted or STIR (fat-suppressed) sequences is a highly sensitive indicator of edema, inflammation, or tearing. MRI can also clearly reveal the extent of damage to surrounding structures, such as retrocalcaneal bursitis, inflammation of the pre-Achilles fat pad, bone marrow edema, and Haglund's deformity.

4. Treatment Strategies

The management of IAT should follow a stepwise and individualized treatment principle. The vast majority of patients can achieve satisfactory outcomes through systematic conservative treatment. Surgical intervention is generally considered only after conservative measures have failed for more than six months [18]. Regardless of the approach, the ultimate goals are to promote normal tendon repair and functional recovery, alleviate pain and stiffness, restore the patient's level of sports activity and daily living capacity, and prevent recurrence.

4.1 Conservative Treatment

Conservative treatment forms the foundation of IAT management and is the first choice for most patients. It usually requires a period of 3 to 6 months or even longer, which significantly tests the patient's compliance and patience. Conservative management includes activity modification (reducing exercise volume), local ice application, non-steroidal anti-inflammatory drugs (NSAIDs), footwear and orthotic modifications, eccentric exercises, stretching and strength training, extracorporeal shock wave therapy (ESWT), as well as injections such as corticosteroids and platelet-rich plasma (PRP) [19].

4.1.1 Eccentric Exercises

Eccentric exercises represent the rehabilitation modality with the highest level of evidence and are considered a cornerstone of conservative management [20,21]. The standard protocol involves the patient standing on the edge of a step with the forefoot placed on the step. The non-affected leg is used to rise onto the toes, while the affected leg is slowly and controllably lowered until the heel descends below the level of the step, creating a deep stretch in the Achilles tendon. The non-affected leg then pushes up to return to the starting position. Patients are typically advised to perform this exercise twice daily, completing 3 sets of 15 repetitions each, for a duration of at least 12 weeks. It is emphasized that the exercises should be continued even during periods of pain, provided the pain remains tolerable [22]. The underlying mechanism is that mechanical loading through eccentric contractions stimulates tendon remodeling, promotes realignment of collagen fibers, and ultimately increases tendon strength [16]. A study by H. Alfredson et al. [23] involving 15 amateur athletes with IAT demonstrated that after 12 weeks of training, all patients showed restored calf muscle strength and reduced pain during activity to pre-injury levels, allowing a return to full-speed running.

4.1.2 Footwear and Orthotic Modifications

Footwear and orthotic modifications are applicable to all IAT patients, especially those unable to adhere to eccentric exercise regimens [24]. Wearing shoes with a slight heel lift or using heel inserts can reduce tensile stress on the Achilles tendon insertion, thereby decreasing tendon swelling and promoting circulation [25]. Furthermore, shoes with a soft posterior counter help minimize direct pressure and friction on the calcaneal tuberosity, preventing irritation of the retrocalcaneal bursa. A study by Dominic James Farris et al. [26] involving 10 female participants running barefoot on a force plate under three conditions (no heel lift, 12mm heel lift, and 18mm heel lift) found that the 18mm heel lift condition reduced ankle dorsiflexion, thereby decreasing Achilles tendon force and strain during running by effectively lengthening the tendon's moment arm. Thus, heel lifts can be a useful tool in IAT rehabilitation to reduce tendon load and mechanical strain.

4.1.3 Extracorporeal Shock Wave Therapy (ESWT)

Extracorporeal Shock Wave Therapy (ESWT) is a non-invasive and effective treatment modality, particularly suitable for patients who show inadequate response to 3 months of conservative management [27,28]. ESWT delivers high- or medium-energy acoustic waves to the affected area, creating microtrauma that induces neovascularization, activates stem cells, promotes tissue regeneration and repair, and inhibits pain signal transmission. It is one of the key non-invasive options for managing refractory IAT [29]. A study by Ziying Wu et al. [30] comparing ESWT outcomes in IAT patients with and without Haglund's deformity showed significant improvement in VISA-A scores for both groups post-treatment, with patients without Haglund's deformity achieving better results.

4.1.4 Corticosteroid and Platelet-Rich Plasma (PRP) Injections

Corticosteroid injections provide significant short-term anti-inflammatory and analgesic effects [31]. However, they carry substantial risks, including inhibition of collagen synthesis, increased tendon tissue brittleness, and a significantly elevated risk of tendon rupture [32]. Therefore, their use in the enthesis area should be extremely cautious or avoided altogether, especially repeated injections. A case report by V. So and H. Pollard described a patient who suffered a partial Achilles tendon rupture, followed by calf muscle atrophy and functional impairment, after repeated corticosteroid injections, underscoring the need for extreme caution with this treatment.

Platelet-Rich Plasma (PRP) injection is a relatively novel

approach for repairing tendon injuries. It utilizes concentrated growth factors and bioactive substances from the patient's own blood. When injected locally, it delivers a potent biological signal to the chronically degenerated tendon tissue, initiating and accelerating the repair process. PRP promotes angiogenesis, improves blood supply, stimulates tenocyte proliferation and the synthesis of healthy collagen, and modulates the local inflammatory environment. This leads to structural repair and tissue regeneration, rather than merely symptomatic relief [33-35]. A study by Davide Erroi et al. [36] on 45 IAT patients treated with either ESWT or PRP confirmed that both methods are effective and safe for managing IAT.

4.1.5 Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs) are primarily used for short-term relief of pain and discomfort. They do not alter the underlying pathological process of tendon degeneration and carry a risk of gastrointestinal complications. Therefore, their long-term use is not recommended [37].

4.2 Surgical Treatment

Surgical intervention for IAT is generally considered when a formal and systematic course of conservative treatment fails after more than six months, when pain severely impacts daily life and work, or in the presence of significant structural abnormalities such as a prominent Haglund's deformity. The goals of surgery are to debride pathological tissue, relieve mechanical impingement, and reconstruct the Achilles tendon insertion. Postoperatively, immobilization with a cast or brace is typically required for 4–6 weeks, followed by 6 months to 1 year of supervised rehabilitation therapy before a full return to sports activities can be achieved [38].

4.2.1 Achilles Tendon Debridement and Posterior Superior Calcaneal Excision

Achilles tendon debridement combined with resection of the posterosuperior calcaneal prominence is the most commonly performed surgical procedure for IAT. Through a medial or lateral approach, degenerated and mucoid tendon tissue, along with proliferative neurovascular bundles, are excised. The hypertrophic posterosuperior aspect of the calcaneus is then remodeled. If present, the retrocalcaneal bursa is also removed [39]. A study by Kenneth J. Hunt et al. [40] compared outcomes of isolated tendon debridement versus those with additional flexor hallucis longus (FHL) tendon transfer and found no significant differences in pain relief, functional scores, or patient satisfaction between the two groups.

4.2.2 Achilles Tendon Insertional Reconstruction

When debridement requires resection of more than 50% of the tendon insertion, the remaining healthy tendon tissue may be insufficient, necessitating reconstruction to maintain tendon continuity. This is often achieved using suture anchors to reattach the residual tendon to the calcaneus. Nicola Maffulli et al. [41] reported on 21 patients treated with this technique. At a mean follow-up of 48.4 months, all patients showed significant improvement in VISA-A scores, with the average increasing from 62.4 to 88.1.

4.2.3 Tendon Transfer

In cases with severe tendon tissue loss or poor function, tendon transfer may be employed to augment strength and endurance. The transfer of the flexor hallucis longus (FHL) tendon is the most common choice, wherein a portion of the FHL is rerouted and attached to the calcaneus to reinforce plantar flexion. Ahmad El-Tantawy et al. [42] conducted a prospective study over 4 years involving 13 IAT patients with a mean age of 58.2 years who underwent FHL transfer. Outcomes were excellent in 11 patients and good in the remaining 2.

5. Summary and Outlook

In summary, the contemporary management of IAT constitutes an integrated system ranging from patient education and load management to biological therapies and surgical intervention. Its successful implementation relies on a profound understanding of the disease pathology and the delivery of personalized, precise treatment strategies. Although significant progress has been made in diagnosis and treatment, clinical practice and research still face numerous challenges, including uncertainties regarding treatment efficacy, difficulties in personalizing therapy, optimization of surgical indications and techniques, as well as standardization of rehabilitation protocols and patient compliance. Looking forward, advancements in basic research and continuous innovation in clinical technologies are expected to lead to more precise, minimally invasive, and effective management strategies for insertional Achilles tendinopathy. The ultimate goal remains to help patients achieve maximal functional recovery, enabling a return to sports and healthy daily living.

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