Case Resolution

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DIAGNOSIS: Postsurgical reactive insertional tendinopathy.

DISCUSSION

Achilles tendinopathy is one of the most common injuries caused by overuse of the ankle and foot. It is a clinical syndrome characterized by the combination of pain, swelling, and impaired performance. The two main categories of Achilles tendinopathy are classified based on anatomical location and include insertional and non-insertional tendinopathy. The etiology of this tendinopathy is multifactorial and includes both intrinsic and extrinsic factors. There are variable conservative and surgical treatment options.

In patients with Achilles tendinopathy, the tendon is heterogeneous and thickened. The histological examination of the affected tissue does not reveal macrophages, neutrophils, or other inflammatory cells. Since histological studies show an increased number of tenocytes and glycosaminoglycan concentration in the ground substance, disorganization and fragmentation of collagen, and neovascularization, the term "tendinopathy" is preferred. The tenocytes present at the site of degeneration have an irregular shape and a higher rate of apoptosis. These tissue changes progress to chronic mucoid tendon degeneration with a variable amount of fibrocartilaginous metaplasia and calcium hydroxyapatite deposits. In chronic Achilles tendinopathy, important molecular changes occur. These changes are compatible with repair, but may also be an adaptive response to changes in mechanical loading, because repeated minor strain is believed to be the main trigger for tendinopathy.

Healthy tendons are relatively avascular. Neovascularization, a descriptive term for the appearance of abnormal vessels, is a feature of Achilles tendinopathy. Neovascularization and its accompanying nerves have been hypothesized to be the source of pain in chronic Achilles tendinopathy.

The sources of pain in Achilles tendinopathy are very complicated. Pain can be caused by multiple factors. Increased prostaglandin production in the matrix, neovascularization in the tendon body, changes in tenocyte structure and function, and metabolite changes in tendinopathy are believed to be the sources of pain. Certain chemical irritants, including cytokines and neurotransmitters, such as glutamate, have been found to be elevated in patients with tendinopathy and have been proposed as causing pain.

Tendon repair involves a sequence of three phases. The first inflammatory phase lasts a few days. Erythrocytes and inflammatory cells migrate to the injury site within the first 24 hours. Vasoactive and chemotactic factors are released with increased vascular permeability, initiation of angiogenesis, tenocyte proliferation, and collagen fiber production. After a few days, the proliferative phase begins. Type III collagen synthesis peaks during this stage, which lasts for a few weeks. Water content and glycosaminoglycan concentrations remain high during this stage. Tendon repair coincides with tenocyte proliferation in the epitenon and endotenon, as well as in the tendon sheath. Finally, after about six weeks, the modeling stage begins. The healing tissue is resized and reshaped. The syntheses of cellularity, collagen, and glycosaminoglycans decrease. This remodeling phase begins with a fibrous consolidation process.

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Failed healing responses and degenerative changes in the tendon have been found. The failed healing response includes three different and continuous stages known as the continuum theory (reactive tendinopathy, tendon deterioration, and degenerative tendinopathy). The quality of the tissue is weakened due to an abnormal healing process, with disordered proliferation of tenocytes, degenerative change of tendon cells, and rupture of collagen fibers. If the source of the tendon injury persists, the area of degeneration or the tear may persist or worsen over time.

The established imaging studies to evaluate the Achilles tendon are ultrasound and MRI. Both methods are excellent for assessing tendon structure, bone insertion, and peritendinous soft tissues.

Conventional radiology is relegated to confirming the presence or absence of intratendinous or enthesis calcifications.

On ultrasound, a normal tendon shows parallel bright linear echoes with a linear transducer with a frequency of 10 ± 22 MHz. Echogenicity is caused by reflection at the interfaces between collagen bundles (Figure 5). Elastography is a technique associated with ultrasound that makes it possible to assess the elasticity of tissues. Tissues that lose water become stiffer and elastography demonstrates this perfectly.

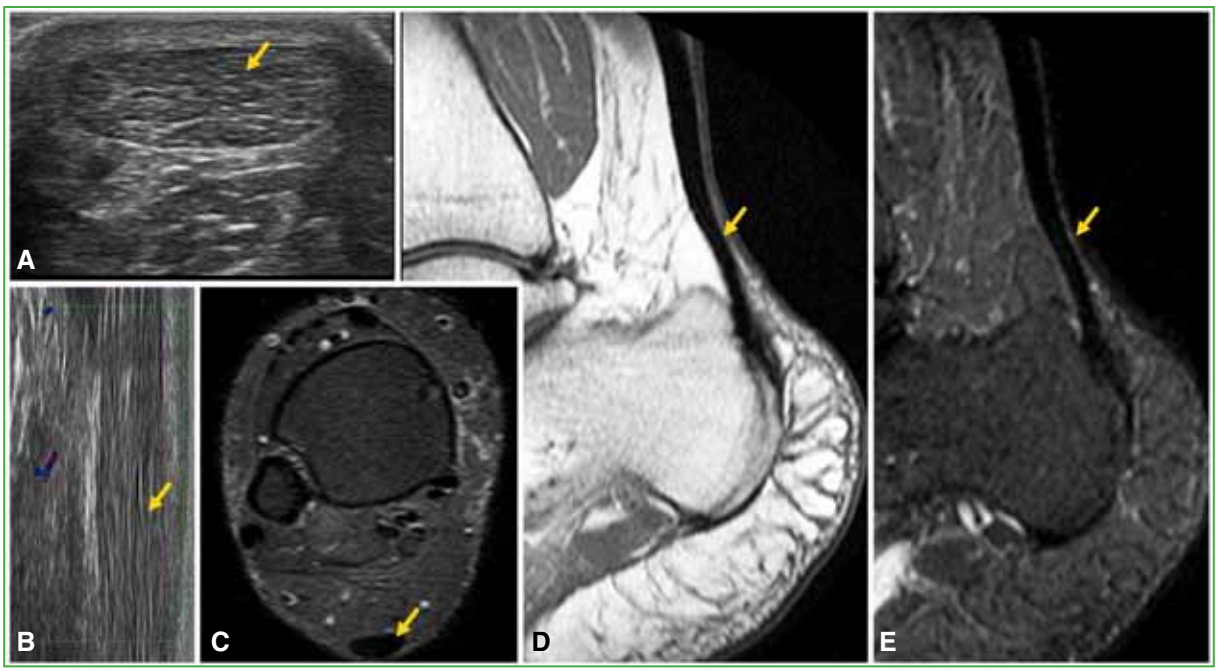

Figure 5. Normal Achilles tendon on both short (**A**) and long (**B**) axis ultrasound and magnetic resonance imaging in axial STIR (**C**), sagittal T1 (**D**), and STIR (**E**) sequences.

A very useful method in musculoskeletal cases is ultrasound with the "power Doppler" technique. Its usefulness in rheumatology to identify and classify this type of disease is better known. It allows assessing the degree of vascularization of the tissues both in inflammation and in the repair phase. Power Doppler ultrasound allows us to make accurate estimates of blood flow, even when vessels are too small. It has also been used to demonstrate soft tissue hyperemia and synovitis, and to study joint effusions and collections. Morphologically normal tendons do not have flow on power Doppler ultrasound, since they are hypovascularized structures.

Normal tendons show a low signal in T1 and T2 MRI sequences, this is due to the fact that they are structures with low water content. In patients with reactive tendinopathies, focal changes in the affected area are detected by ultrasound or MRI (Figure 6).

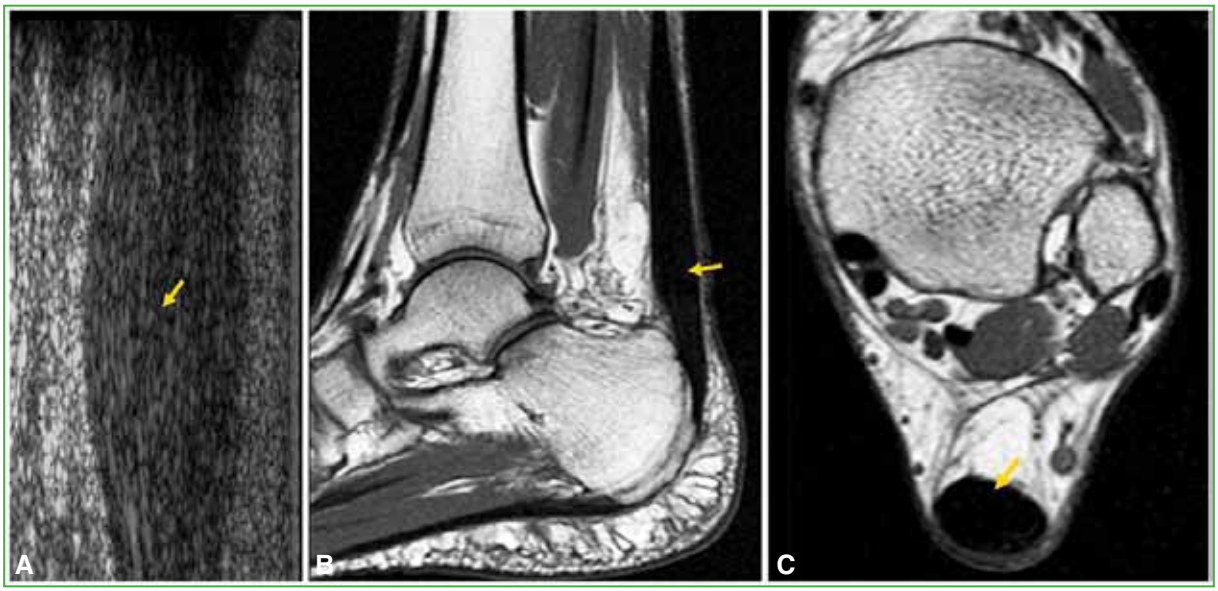

Figure 6. Achilles tendinosis. The tendon is thickened with changes in echogenicity (**A**) and magnetic resonance signal in sagittal T1 (**B**) and axial T2 (**C**) sequences.

The tears show a low signal in the T1 sequence and an increased signal in the T2 sequence. The same as the inflammatory changes in the insertional and peri-insertional soft tissues (Figures 7 and 8).

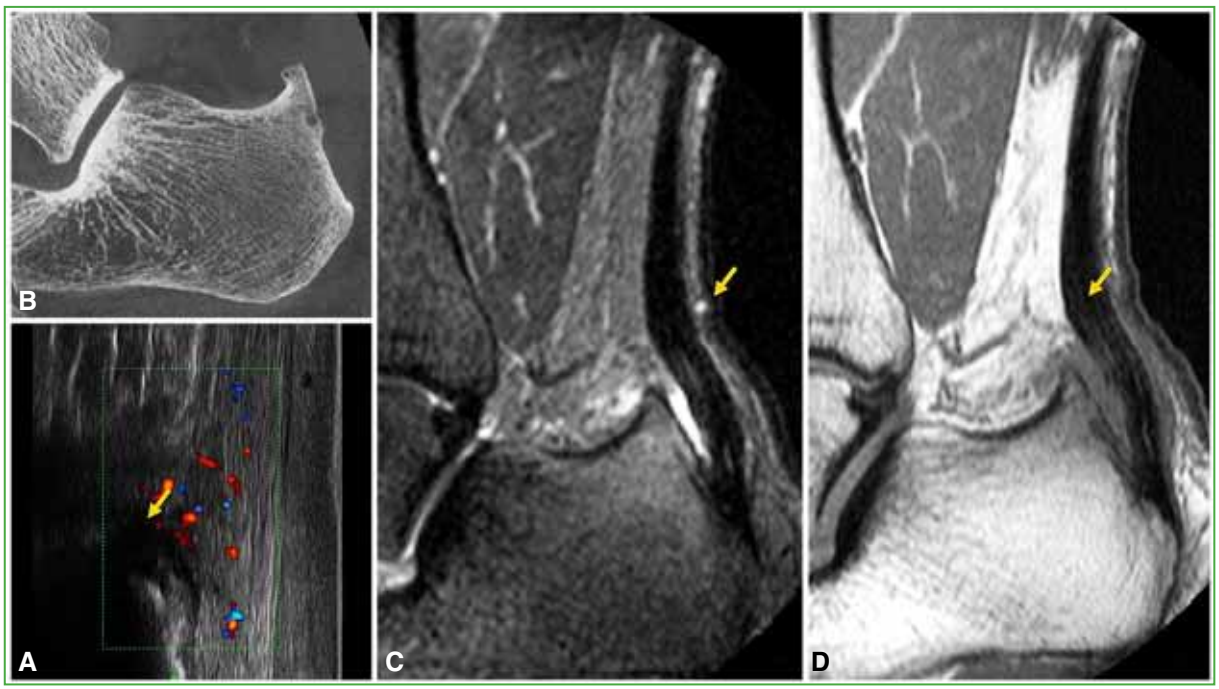

Figure 7. Insertional tendinopathy due to Haglund's deformity. The power Doppler technique reveals inflammatory changes with neovascularization in the insertional conflict zone of the deep tendon fibers (**A**). Sagittal computed tomography reconstruction where the bone deformity is recognized (**B**). In the magnetic resonance, sagittal STIR (**C**) and T1 (**D**) sequences, changes in the tendon signal are observed that suggest tendinosis and partial tear, bone edema, and fluid in the retrocalcaneal bursa.

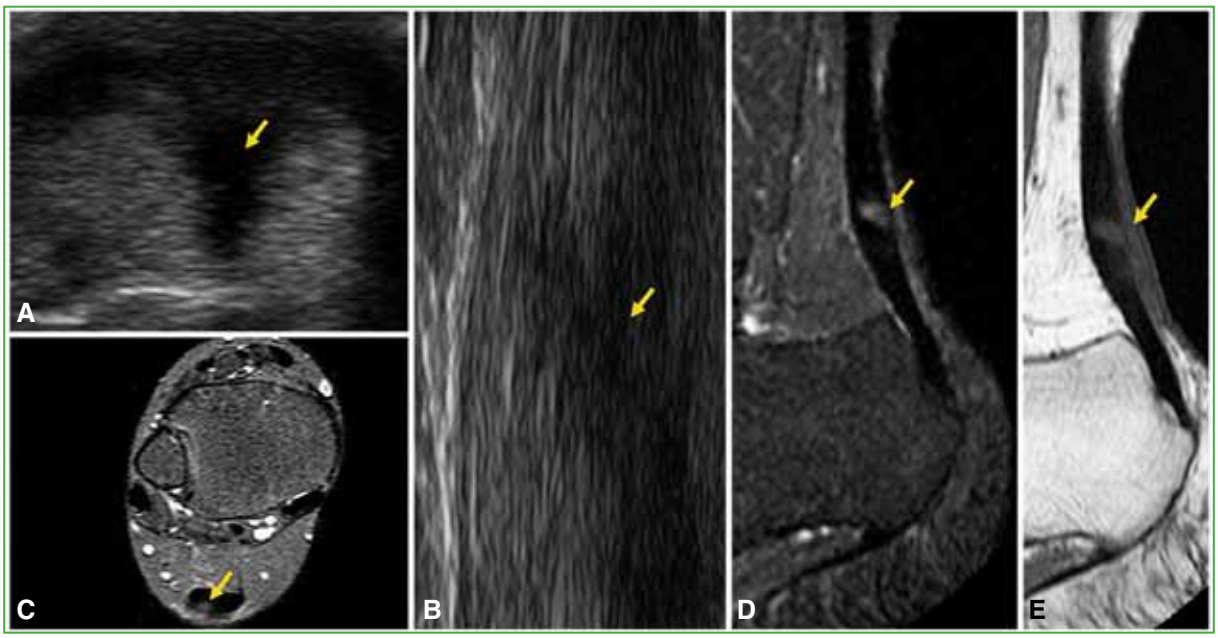

Figure 8. Partial tear of the Achilles tendon caused by a glass cut. The tendon shows the focal lesion on both ultrasound (**A and B**) and magnetic resonance imaging (**C-E**). The rest of the tendon is normal.

In chronic tendinopathy, the thickness of the tendon is altered, the structure is abnormal, ultrasound shows an irregular pattern with areas of low echogenicity, and MRI shows a modified signal (Figures 9 and 10).

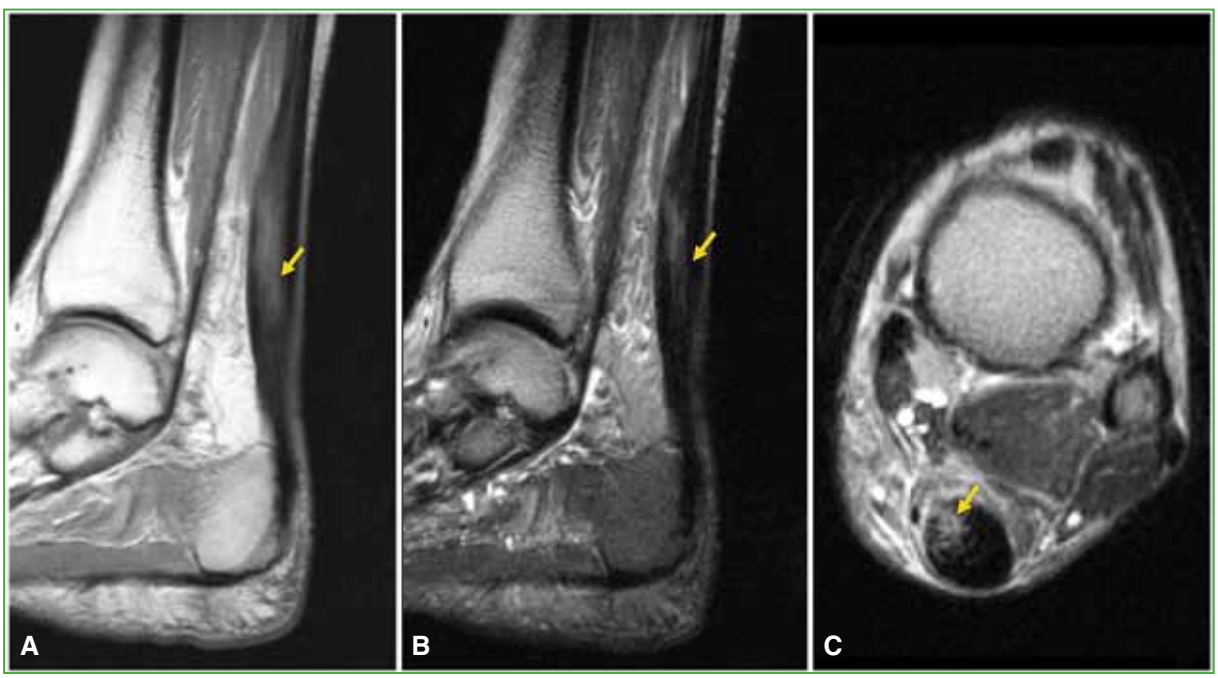

Figure 9. Magnetic resonance imaging showing a partial tear in chronic tendinopathy. The tendon is thickened and shows signal changes suggestive of tendinosis (**A and B**). A partial tear is recognized in the medial fibers in the axial T2 sequence (**C**).

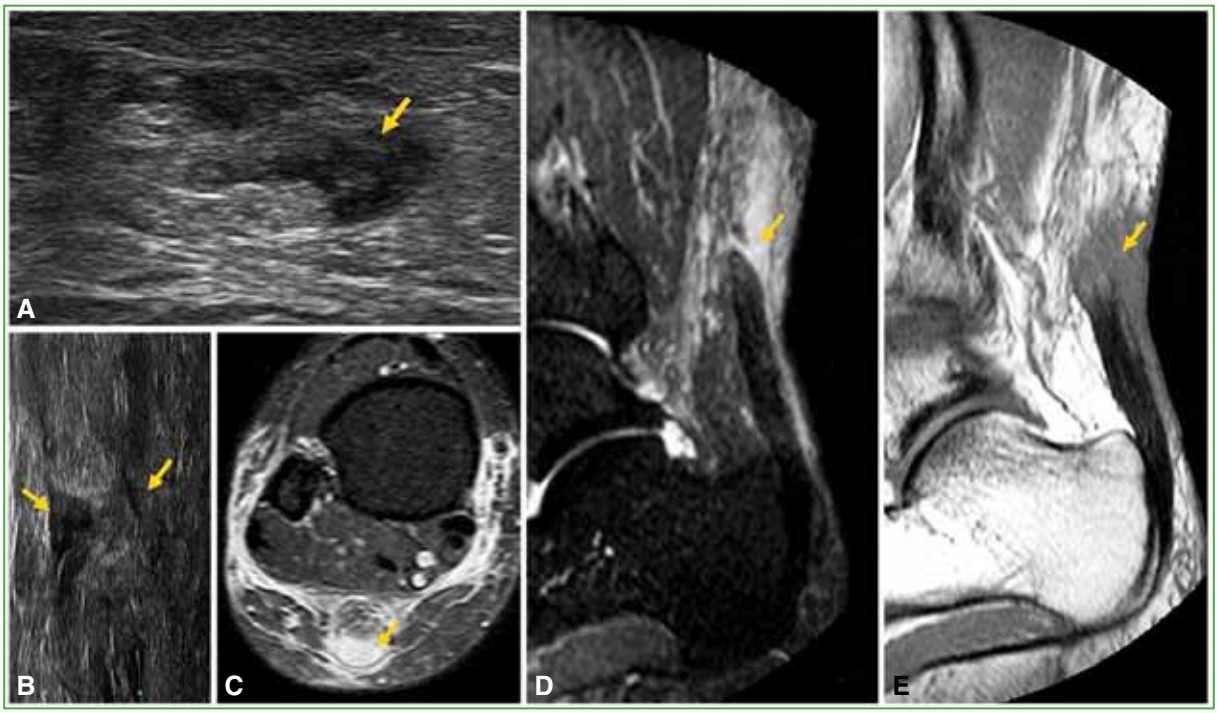

Figure 10. Short (**A**) and long (**B**) axis ultrasound, axial STIR (**C**), sagittal STIR (**D**), and T1 (**E**) sequences showing a complete tear of the Achilles tendon in the proximal third.