An Unusual Presentation of Tennis Leg in the Setting of Anabolic Steroid Use
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Abstract
This case study illustrates an unusual dual injury of both the gastrocnemius muscle and the plantaris muscle that presented clinically as “tennis leg”. The initial depiction of “tennis leg” in 1883 connected the acute onset of mid-calf pain with rupture of the plantaris muscle. Recent literature has shown that most cases are caused by rupture of the gastrocnemius muscle rather than the plantaris muscle. Although a combination of gastrocnemius muscle and plantaris muscle injury has been described, such injuries are relatively uncommon. Isolated muscle injuries are seen at a much higher frequency. We present a case of concurrent injury in the setting of anabolic steroid use.

Keywords: Tennis leg, Plantaris Muscle, Tendon Injury, Gastrocnemius Muscle, Soleus Muscle

Abbreviations: PM: Plantaris Muscle; GM: Gastrocnemius Muscle; SM: Soleus Muscle; MMA: Mixed Martial Arts; AAS: Anabolic Androgen Steroid; ECM: Extracellular Matrix

Introduction
“Tennis leg” describes the acute onset of mid-calf pain during ankle dorsiflexion with simultaneous knee extension, putting strain on the calf musculature [1-3]. The original injury, described in 1883, was attributed to an isolated rupture of the plantaris muscle (PM) [4]. As new imaging emerged and with further investigation it was found that most clinical cases of “tennis leg” had radiologic findings of complete or partial tearing of the medial belly of the gastrocnemius muscle (GM) or a fluid collection between the GM and soleus muscle (SM) without visible muscle rupture. These findings were seen more frequently than the isolated rupture of the plantaris tendon [1, 2, 5]. Isolated calf muscle injury (predominantly the GM) was more commonly seen than concurrent injuries. Of the concurrent injuries, Koulouris et al. [6] reported that the combination of a GM injury with a SM injury was the most common, followed by injury to both heads of the GM, SM, and tibialis posterior [6]. The least common injury was that of the SM and flexor hallucis longus. There are few published cases documenting “tennis leg” with concurrent GM and PM rupture [7]. We believe that our patient’s chronic use of anabolic androgen steroids likely contributed to this injury.

Case Description
A 39-year-old male MMA (mixed martial arts) fighter with a history of chronic rhabdomyolysis and anabolic androgen steroid (AAS) use presented to the emergency room following ten days of worsening cramping pain and with ecchymosis along the left lower extremity, extending from the posteromedial calf to the medial malleolus and arch of the foot. The patient did not endorse any recent trauma to the area and reported to clinicians that he had been on a much less rigorous workout regimen since his last fight a month prior.

On physical exam, there was significant ecchymosis involving the left posterior calf, medial ankle, and arch of foot. Leg compartments were swollen but compressible and there were 2+ dorsal pedal and posterior tibial pulses. Strength was 4/5 with eversion, inversion, plantar and dorsiflexion, although it was difficult to determine if the patient was able to exert full effort during the exam. The patient had a negative Thompson test. Bedside ultrasound showed an intact left Achilles tendon and the patient had a negative bedside ultrasound for DVT.

Four-view radiographs of the left ankle, three-view radiographs of the left foot, and two-view radiographs of the left tibia-fibula, all showed no evidence of fracture or dislocation. A multiplanar multi-sequence MRI of the left tibia-fibula without contrast showed edema within the medial head of the gastrocnemius muscle with partial rupture of the medial head of the gastrocnemius (Figure 1 & 2). Fluid signal with areas of irregular low signal intensity was also noted between the soleus muscle and the medial head of the gastrocnemius. This was believed to represent the remnant of the ruptured plantaris tendon. These radiological findings together were consistent with “tennis leg”.

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Abstract

The most common presentation of "tennis leg" is acute pain and swelling involving the posterior calf in a middle-aged patient. Homans’ sign is often positive while Thompson’s test is negative. Some plantar flexion weakness may be noted when the patient performs a single-limb heel raise [3, 8].

The posterior superficial calf muscles (GM, SM, and PM) act as the primary plantar-flexor of the ankle [8]. The Both the GM and the PM extend over both the knee joint and the ankle joint. The combination of ankle dorsi-flexion and knee extension, that is common with abrupt pivoting, puts increased strain on the musculotendinous junction. This elevates the risk of rupture due to excessive stress on the musculotendinous junction. The PM has a short muscle belly that attaches to the posteroslateral femur then quickly transitions into a long tendon that attaches to the medial calcaneus or directly onto the Achilles tendon. The PM runs between the GM and the SM. It is absent however in approximately 20% of the population without any functional sequela [3].

Both ultrasound and MR can be used for the diagnosis of “tennis leg”. Ultrasound is particularly helpful as it not only helps to visualize the location and severity of muscle injury but is also essential in ruling out life threatening conditions like deep vein thrombosis [5, 9]. Ultrasound most commonly appears as disruption of the medial gastrocnemius muscle pinnate pattern. This usually occurs at the junction of the triceps surae. Hypo echoic fluid is also commonly seen tracking between the medial gastrocnemius and soleus muscles without definite rupture [5].

Direct signs on MR that point to “tennis leg” are best seen on T2 weighted sequences in the axial and coronal orientations. Images showing increased signal within the medial GM head are consistent with “tennis leg”. A complete rupture is viewed as discontinuity of the medial GM head with interposition of high signal intensity. Injury to the PM on T2 weighted imaging appears as high signal intensity within and adjacent to the muscle. If the PM is completely ruptured a mass may be appreciated between the popliteus tendon and the lateral head of the gastrocnemius [7, 10].

Discussion

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The main treatment for isolated GM injury “tennis leg” focuses on RICE therapy (rest, ice, compression, and elevation) [3]. Providing patients with conservative treatment has been shown to be effective without major long-term disability [1, 5]. It can take anywhere from 4-12 weeks however for patients to return to normal activity without pain. The muscles usually heal fully and injury to the same leg is rarely seen [1, 11]. It has also been found that early compression and immobilization of the injured calf can lead to earlier recovery [10, 11].

“Tennis leg” presents most commonly in middle-aged adults. This is likely due to the muscle fatigue and degenerative changes that come with age [1, 3]. In addition, there are some reports that show a possible association between AAS use and tendon rupture [1, 12, 13]. AAS use is believed to alter the biomechanical properties of tendons resulting in reduced tendon elasticity, increasing the risk of tendon rupture [14]. Tendons are comprised of large amounts of highly organized extracellular matrix (ECM). The ECM of tendons are continuously remodeling and adapting to different mechanical loads. Exercise has been shown to increase the tendon thickness, cellularity, and blood vessel volume within the peritendinous sheath, while the concurrent use of AAS inhibit these beneficial changes. It is believed that collagen metabolism is also negatively impacted by AAS use which may cause detrimental changes to the plasticity of tendons [2].

It is estimated that around 2.9-4 million Americans have used AAS at least once in their lives with around a quarter of them forming a dependence [14]. AAS is seen in all age groups.
It is estimated that high school boys for example have a 1% lifetime prevalence of AAS use. This raises to 1.58% lifetime prevalence in adults 19-28 years old [15]. Although AAS is usually associated with athletes and body builders, AAS use also affects non-athletes [16-18]. Given the increasing use of AAS in all age groups with effects on athletes and non-athletes alike, we anticipate we will see these types of injuries more and more frequently.

This case is particularly important as it illustrates that injuries in patients who abuse AAS may not fit the typical, expected patterns. It is important for clinicians to be wary of unusual patterns of injury in this cohort of patients. Clinicians should assess these patients for concurrent injuries given their predisposition to tendon injury due to the structural changes caused by AAS use.

Reference