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Diagnostic Challenges in Plantaris Tear: A Case Study of Suspected TendoAchilles Injury

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Abstract: ***This case study examines the complexities in diagnosing a plantaris tear initially suspected to be an incomplete TendoAchilles tear. The patient, a 54 - year - old male, presented with significant pain and functional impairment in the left ankle following a minor trauma. Initial examinations indicated a probable Achilles tendon injury; however, further imaging revealed a plantaris tear with associated hematoma. The case highlights diagnostic challenges, potential misdiagnoses, and the conservative treatment path taken to achieve successful recovery.***

Keywords: plantaris tear, TendoAchilles injury, diagnostic challenges, proprioception, conservative therapy

1. Introduction

The plantaris muscle is a vestigial muscle component of the triceps surae complex. It is a proprioceptive organ for ankle plantarflexors having a high density of muscle spindles. Plantaris tears are uncommon and difficult to diagnose as it masquerades as painful swollen calf / deep vein thrombosis. Tears usually occur at the myotendinous junction – along with injury of ACL, arcuate ligament, medial head gastrocnemius, Soleus or combinations (Tennis Leg). Injury occurs with eccentric ankle loading with the extended. Mechanism is indirect, but patient may have direct trauma. The pain increases after resting or the next day, the swelling spreads distally.

This case study contributes valuable insights into the diagnostic nuances of plantaris tears, underscoring the importance of accurate imaging and comprehensive clinical assessment to prevent misdiagnoses and facilitate effective treatment

2. Methodology

Case Report

A 54 year old male patient was wheeled into our clinic on a wheelchair. He complained of inability to stand up or walk or even keep his left foot on the foot rest of the wheelchair. He complained that the left ankle was painful, swollen and bruised since the last 24 hours. He was an autorickshaw driver who was repairing his vehicle in a squatting position, as he was getting up he felt that someone has thrown a hard rubber ball at his left calf. He was gripped by pain and was unable to get up on his own. It was not possible for him to remain standing or to walk, or to push the pedals of his vehicle. He was then taken to a general physician who diagnosed him as Achilles' tendon injury since he had a painful swollen ankle with an inability to perform plantarflexion. Inspite of being treated with an ice pack and intra muscular injection of Diclofenac his calf pain had increased over the night. On presentation in our clinic – he had a painful swollen ankle, with plantar flexion being almost absent (although a slight jog was present), Symmond's test was not positive, and he had a vague ache in his calf – he was clinically diagnosed as having an incomplete Tendo Achilles tear. He carried an USG report with him dated the previous day where the sonologist had diagnosed him as having Deep Vein Thrombosis. We requested a repeat USG from our institutional consultant (Professor) – who ruled out DVT, and diagnosed him as having a possible plantaris tear with hematoma tracking between the soleus and gastrocnemius upto the ankle. The tendo Achilles was seen to be intact in USG.

We were now looking at 3 clinical problems.1) Ankle Bruise – we initiated treatment with a short course of NSAIDs (Aceclofenac) and followed the RICE protocol (Rest, Immobilisation, Ice Compression, Limb Elevation).2) Soft tissue injury with impending compartment syndrome – for which he was put under close clinical observation 3) DVT prophylaxis - he had a BMI over 35 and was confined to his bed. Further, we decided to get additional imaging of his leg by obtaining an MRI once his clinical condition improved.

5 days post injury, his ankle swelling subsided with improvement in plantar flexion. Compartment syndrome did not develop in the leg and there was no neurovascular deficit. Color Doppler ruled out Deep Vein Thrombosis. Clinical Examination of knee was unremarkable. With this set of findings we proceeded to image the posterior leg compartment by means of an MRI.

Our institutional radiology consultant reported that there was a plantaris tear at the Myotendinous junction, along with a medial head of gastrocnemius intramuscular tear and tendon strain of that head. Hematoma was tracking down the leg in the intermediate posterior compartment. There was no other significant injury to the soft tissues and the popliteal vessels were unremarkable.

The patient was managed conservatively and rehabilitative physiotherapy was started 2 weeks after the injury.

3. Discussion

Powell in the late 18th century reported "Tennis Leg" – a complex of injuries presenting as acute mid calf pain in the middle aged adult male. Forced ankle dorsiflexion with the knee extended was reported as the mechanism, with a snap both felt and heard. Classically plantaris and gastrocnemius were injured together (as seen on cadaver dissections). The

medial head of gastrocnemius was most commonly injured, and an intramuscular tear was most common.

Delgado, however, reported in his extensive survey of 141 patients only 2 had plantaris tears. Anterior Cruciate Ligament and Posterolateral Corner were reportedly injured concomitantly. Deep vein thrombosis was associated with tennis leg injuries. In some patients, anatomical variations of the plantaris tear would make the torn muscle entrap the popliteal vessels leading to a vascular catastrophe.

The plantaris muscle is small but unique component of the triceps surae complex (made up of gastrocsoleus). According to Kurtis & Vloit, the plantaris muscle has the highest concentration of muscle spindles per unit cross sectional area. These muscle spindles are the end organs of proprioceptive feedback – cutting off that feedback would lead to a breakdown of the sensorimotor reflex arc and thus explain the loss of plantarflexion in our patient.

Proprioceptive end organs are usually of the following types 1) Muscle Spindle Ensemble/ Golgi End Organ – measuring speed, rate, rhythm, force of contraction. Furthermore, it recognises pattern of contractions, and retains memory of previous contractions. The spindles can further assess the strength of contraction, defined as Force Strength (FS). As an effect of aging this sensation may decrease, resulting in problems in postural balance and standing (Kim & Chung - hwi), known as deafferentation. Joint position sense (JPS) – comes from antagonists, therefore dorsiflexors would be the the JPS sensors in case of ankle plantar flexors.

The next end organ of proprioception is the joint capsule (JC) – it contains Ruffini like mechanoreceptors and Pacinian corpuscle equivalents (Proske & Gandieva) which are stretched with movement. These receptors are either slow adaptive receptors (70% of receptors) or fast adaptive receptors (30% of receptors, active at extremes of joint movement).

The 3rd end organ of proprioception is skin overlying the joint. The overlying skin creates movement illusions by means of Meissner, Merkel, Pacinian & Ruffini corpuscles. Stretched skin due to mechanical or organic causes (for eg oedema) can occlude other proprioceptive input similar to Gate Control theory of pain.

The loss of proprioception in our patient was therefore due to 1) Deafferentation due to plantaris tear thus severing the muscle spindle input.2) Swelling of joint capsule and skin overlying ankle thus occluding all proprioceptive input from the ankle. An ankle devoid of proprioceptive input would lose it's movement and recover it slowly with resorption of edema and healing of the torn muscle.

Our patient's ankle movement recovered with the tissue edema settling down (in the ankle and plantaris) and recovery of proprioception. There was no objective signs of neurological or vascular injury, and Deep Vein Thrombosis or Compartment Syndrome did not develop. Gradual mobilisation was started at 2 weeks with full weight bearing achieved at 6 weeks, the entire rehabilitation being closely monitored by our institutional physiotherapist.

Ankle movement recovered fully with edema subsiding and healing of plantaris muscle (as evidenced by MRI findings subsequently) with no clinical loss of proprioception.

4. Conclusion

TendoAchilles tears make us ignore the presence or consequences of a proximal tear of the plantar - flexor complex. Plantaris tear disrupts the proprioception of ankle plantar flexor complex resulting in motor deficit & deceptive clinical presentation. Simple conservative therapy usually recovers the plantar flexion lost due to Deafferentation, though a strict vigil should be maintained for complications like Deep Vein Thrombosis or Compartment Syndrome.

In summary, this case emphasizes the importance of considering plantaris tear in differential diagnosis when dealing with lower limb injuries mimicking TendoAchilles tears. Proper diagnosis, coupled with conservative management, allowed for a full recovery without further complications. Continued awareness of this uncommon injury can aid clinicians in improving patient outcomes in similar cases

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