UNDER THE SURFACE: WHY SOLEUS STRAINS ARE SIDELINING SO MANY FOOTBALLERS

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Introduction
Calf muscle strain injuries (CMSIs) are a significant cause of time-loss in professional football, consistently representing 6–12% of all muscle injuries in UEFA and FIFA surveillance studies.[1,2] However, these datasets rarely differentiate between gastrocnemius and soleus involvement, masking the true contribution of soleus pathology in elite football.

Clinical experience and smaller imaging-based studies suggest that soleus injuries are both common and under-recognised, in part due to their deep location and the subtle, often delayed onset of symptoms. When the intramuscular aponeurosis (IMA) is involved, recovery can be prolonged and reinjury risk elevated.[3-6]

This article explores why soleus strains are so disruptive in football, highlighting key anatomical, mechanical, and biological factors, and presents a practical rehabilitation model informed by tissue healing and performance demands.

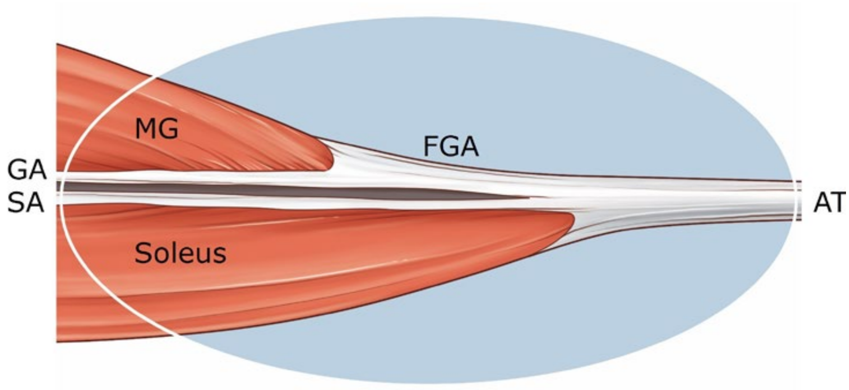

Figure 1: *Schematic of the triceps surae and FGA anatomy. Image designed by Massimiliano Crespi. Reproduced with permission from: Pedret C, Palermi S, Mechó S, Ahmad G, Lee J. Calf muscle injuries in the athlete. Aspetar Journal. 2025 Vol 14*[32]

Complex Anatomy of the Triceps Surae
The triceps surae—comprising the gastrocnemii and soleus—shares a mechanically sophisticated architecture. While functionally distinct, these muscles connect via the free gastrocnemius aponeurosis (FGA) at a V-shaped junction. This configuration enables efficient force sharing, but because the muscle heads can generate force independently, the interface is subject to significant internal shear.[1-2] Consequently, opposing vectors often converge across the distal medial gastrocnemius and FGA, creating focal stress zones that may help explain the high prevalence of injuries at this location. This mirrors the "T-junction" phenomenon described in hamstring injury patterns, where the interface between adjacent muscle-tendon units becomes a primary site of mechanical failure (Figure 1).

Soleus Architecture and Aponeurotic Complexity

The soleus has a particularly complex aponeurotic system and a multi-pennate fascicle arrangement. Five main aponeurotic structures are relevant clinically:

1. **Posterior aponeurosis** – covering the mid- and distal belly.
2. **Anterior aponeurosis** – covering the proximal region.
3. **Central tendon** – arising distally from the Achilles tendon via the posterior aponeurosis, joining the anterior aponeurosis proximally.
4. **Proximal tendinous arch.**
5. **Medial and lateral intramuscular aponeuroses (IMAs)** – descending from the proximal tendinous arch into the muscle.

Intramuscular aponeuroses act as anchorage sites for muscle fascicles and distribute force between compartments within the muscle, while peripheral aponeuroses transmit force to neighbouring structures and accommodate radial expansion. The central tendon, continuous with the Achilles, likely contributes to elastic energy storage and return (Figure 2).

The multi-pennate architecture, regional variation in pennation angle and the pattern of intramuscular nerve branches create non-uniform strain within the muscle–aponeurosis complex.[3] There are also varying regional mechanical properties among aponeuroses within the soleus.[4] Combined with its large physiological cross sectional area and major force producing role during running, acceleration, cutting, and jumping,[9-13] the soleus aponeuroses and myotendinous structures are exposed to substantial internal stresses.

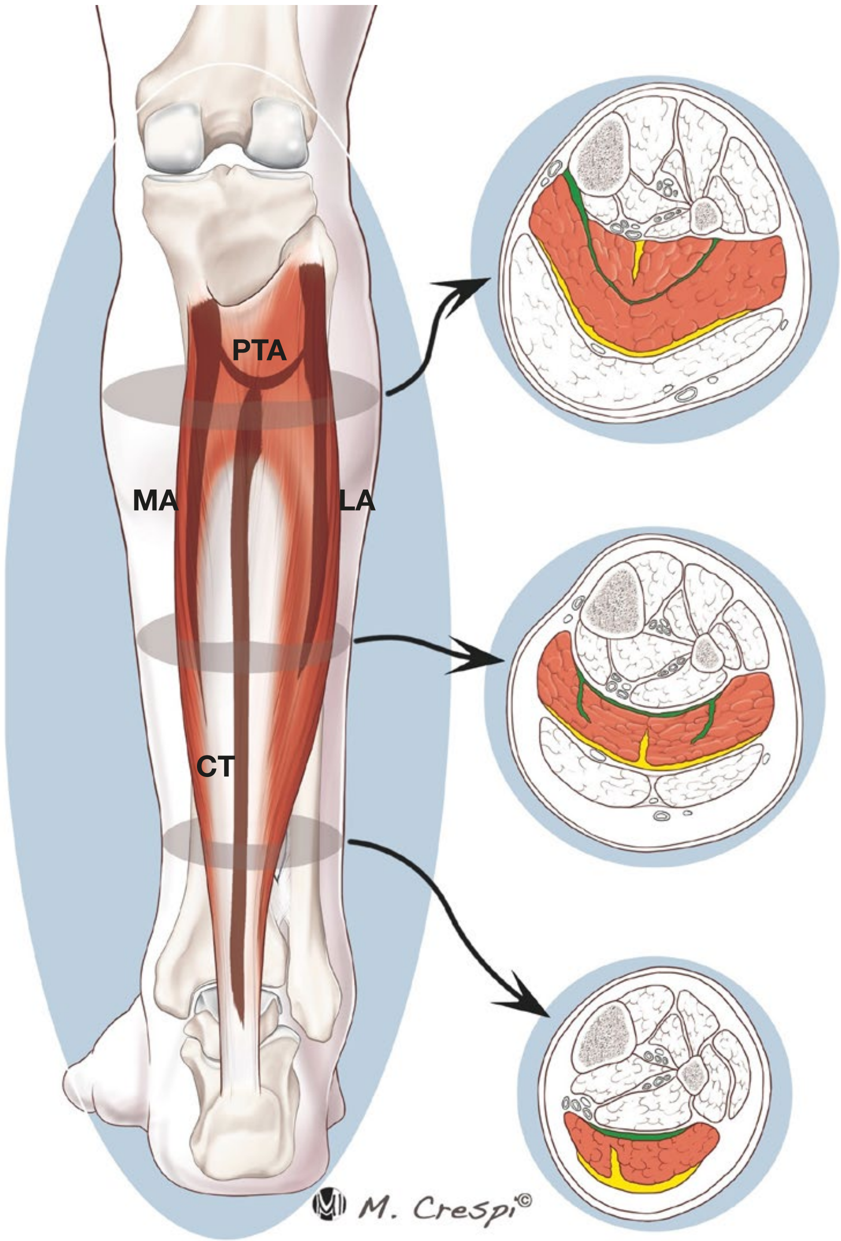

Figure 2: Schematic of the soleus intramuscular aponeuroses. Image designed by Massimiliano Crespi. Reproduced with permission from: Pedret C, Palermi S, Mechó S, Ahmad G, Lee J. Calf muscle injuries in the athlete. Aspetar Journal. 2025 Vol 14[32]

Physiological Profile and Vascular Adaptations

The soleus is predominantly oxidative, with around 80% type I fibres,[5] and acts as an important peripheral "venous pump".[6] It tends to derive more energy from blood glucose and less from intramuscular glycogen than nearby, more glycolytic muscles.[7] This may also facilitate the sparing of muscle glycogen stores adding to its potential high work capacity.[7]

In older athletes, MRI often demonstrates adaptive deep veins in the soleus, frequently adjacent to or perforating the aponeuroses. Although not fully understood, these vascular changes may reflect chronic metabolic and haemodynamic adaptations and could theoretically reduce local structural resilience in regions already subject to high mechanical load.

Anatomical Variants

MRI and cadaveric studies have described several anatomical variants, including:

- Accessory central tendons[8]
- Absence of either the medial or lateral IMA[8,9]
- Asymmetry in medial vs lateral IMA coverage[10]

Pedret et al.[10] further classified variants such as a medialised or lateralised central tendon, altering compartment size. When injury occurs in a structurally dominant compartment, rehabilitation becomes more challenging due to higher force transmission, increasing reinjury risk if progression is too rapid.

Role of the Soleus in Football

During steady state running, the soleus can generate forces up to 10-times bodyweight, with even higher demands during accelerations and jumping.[11–13] Its main roles include:

- Providing vertical support during stance
- Accelerating the centre of mass vertically and horizontally
- Controlling anterior tibial translation[14]

As a mono-articular, type I-rich, multi-pennate muscle, the soleus functions as a workhorse, producing peak force in mid-stance when the centre of mass is lowest and the knee most flexed. In this phase it couples with the quadriceps with similar peak force timing and operates largely independently of the gastrocnemii.

The bi-articular gastrocnemii, being more fast-twitch and unipennate, contribute more to late-stance explosive propulsion and energy transfer between joints.[21] This division of labour highlights why soleus injuries often occur without a classic "push-off" mechanism and why they can be easily overlooked.

	Oedema or fluid adjacent to connective tissue	Myofibril detachment	Connective tissue increased signal, delamination or defect	Connective tissue retraction or failure
Grade 0	Yes	No	No	No
Grade 1	Yes	Yes	No	No
Grade 2	Yes	Yes	Yes	No
Grade 3	Yes	Yes	Yes	Yes

Figure 3: *Prakash grading system for soleus muscle strain injuries*[22]

Injury Patterns

Soleus Injuries

Soleus injuries are difficult to assess via ultrasound due to depth and architectural complexity. MRI is preferred for accurate localisation and grading. Although the British Athletics Muscle Injury Classification (BAMIC) is widely applied, oedema size alone is less predictive for soleus strains; the extent and location of aponeurotic disruption is more informative. The Prakash classification prioritises this and may offer better prognostic value (Figure 3).

Clinically, soleus injuries often occur during running with gradual or subtle onset. Players may feel fine during the session but describe tightness or stiffness the following day, which can be misinterpreted as benign post exercise soreness.

Healing Time Course and Re Injury Risk

One of the key challenges in practice is the mismatch between recovery of muscle capacity and the slower remodelling of aponeurotic tissue. Muscle fibre re-innervation, plyometric capacity and running performance can normalise relatively early, while aponeurosis remodelling and scar maturation continue over many weeks and months.[15] This creates a vulnerable period where players appear ready yet remain at elevated risk of reinjury.

Figure 4 illustrates:
- Early recovery of muscle capacity and running ability
- A slower curve for aponeurosis remodelling and scar progression (often extending beyond 12–24 weeks)
- A window of highest re injury risk, where functional performance appears adequate but the aponeurosis is not yet mature

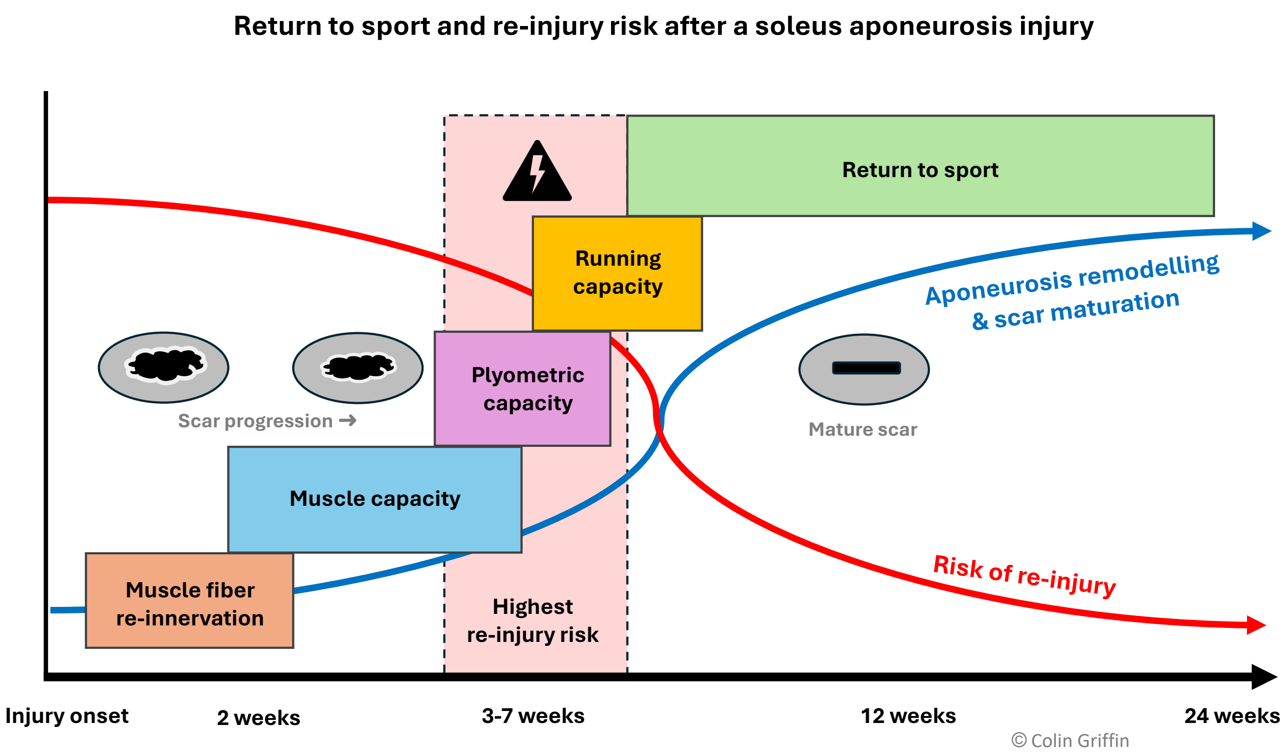

Figure 4: *Calf tissue healing framework*

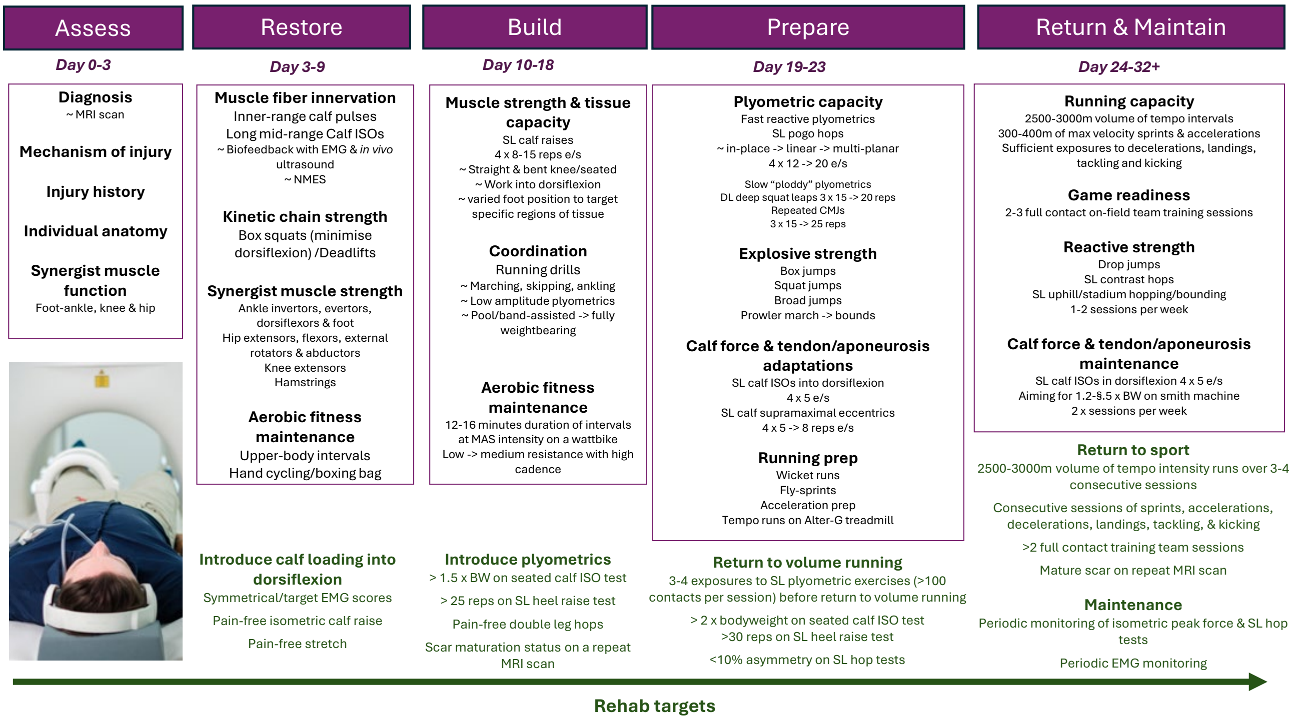

Figure 5: *Calf tissue healing framework*

This underscores the need to go beyond strength thresholds or distance-based running progressions. Monitoring aponeurotic loading tolerance and maintaining structured progression beyond RTP are essential.

Risk Factors in Football Context

Age

Players >30 years have a higher CMSI risk.[16-17] Potential mechanisms include altered metabolism, low-grade inflammation, impaired collagen turnover and age-related changes in neuromuscular control. In older players with soleus injury, MRI and indeed ultrasound, frequently reveal deep adaptive veins around the aponeuroses, which may reduce local structural robustness. These factors argue for more individualised loading in older athletes—especially around congested fixture periods—and a conservative approach to high-load calf work.

Previous Injury

Previous calf strain is associated with a higher risk of recurrence. Architectural changes, aponeurotic thickening and partial denervation can persist following medial gastrocnemius injury.[18] Anecdotally similar architectural and activation alterations are observed in the soleus following injury. Other conditions also influence triceps surae function:

- **Achilles tendinopathy:** reduced lateral gastrocnemius drive with compensatory soleus over-activity.[19-20]
- **Ankle sprain:** often associated with soleus inhibition.[21]
- **ACL reconstruction:** calf strength and unilateral reactive strength deficits often persist.[22]

These adaptations can shift load towards specific parts of the soleus–gastrocnemius–Achilles complex, increasing loading demands on already vulnerable regions.

Training Patterns and Match Demands

Over a decade ago many field sport programmes moved toward low-volume, high-intensity interval formats as a time- and mechanically-efficient way of developing aerobic fitness.[23] Meanwhile, match data (e.g. BJSM report) indicate increased total running distance and higher game congestion compared with previous seasons.[24]

If training no longer matches the sustained running demands of competition, some players may lack calf endurance capacity relative to match loads. Historically, calf-specific conditioning has also been under-emphasised, and even where it has improved, the focus is often on peak force rather than time-under-tension and long muscle–tendon unit (MTU) lengths.

Neural recovery from fatiguing calf work can be relatively rapid, whereas collagen remodelling is slow.[25] This can leave partially remodelled aponeuroses exposed to high load—particularly in players returning from injury or facing congested schedules.

A Practical Rehabilitation Framework for Soleus Muscle Strain Injuries

A structured framework that aligns clinical decision-making with tissue biology and performance demands is particularly useful in the professional football environment.

Figure 5 summarises a phase-based approach with example timelines and criteria. In brief:

Assess

- Confirm diagnosis and location (MRI for soleus).
- Consider mechanism, age, previous injury, individual anatomy and anatomical variants.
- Assess synergist muscle strength (foot/ankle, knee, hip) and, where available, use historical strength and running data to set return-to-play (RTP) targets.

Restore

- Early protection of the aponeurosis while restoring activation: inner-range calf

pulses, mid-range isometrics, NMES and EMG/ultrasound-guided biofeedback where available.
- Begin kinetic-chain strength (e.g. box squats, deadlifts with limited dorsiflexion) and maintain aerobic fitness via upper-body intervals.

Build
- Progress to SL calf raises (straight and bent knee/seated), working into dorsiflexion with varied foot positions to target specific regions.
- Introduce low-amplitude plyometrics and running drills (marching, skipping, ankling), progressing from pool/band-assisted to full weightbearing.
- Maintain aerobic fitness with MAS-based intervals on a bike or equivalent.

Prepare
- Develop plyometric capacity: fast SSC (e.g. SL pogo hops) and slower, deep-flexed rebounds at higher volumes.
- Use high-intensity SL isometrics in dorsiflexion and supramaximal eccentrics to target tendon/aponeurosis adaptation.
- Progress running from wicket runs and accelerations to tempo runs and higher-speed exposures, initially on bodyweight-support treadmills if needed.
- Aim for >3 exposures to SL plyometrics (>100 contacts/session) before returning to volume running.

Return & Maintain
- Progress to 2,500–3,000m tempo intensity runs over 3–4 sessions and 300–400m of high speed running and accelerations, with adequate exposure to decelerations, landings, tackling and kicking.
- Ensure completion of 2–3 full contact team sessions without adverse response.
- Use objective criteria (e.g. seated calf isometric >1.5–2.0 x bodyweight, >25–30 SL heel raises, <10% SL hop asymmetry) plus imaging evidence of advanced scar maturation where available.
- Maintain calf and aponeurosis capacity with 1–2 weekly high-intensity loading sessions working in dorsiflexed positions, and periodic monitoring of isometric strength and hop performance.

When combined with the healing-time framework (Figure 4), this rehabilitation model (Figure 5) provides a practical roadmap for aligning loading decisions with both tissue healing and football-specific performance demands.

Conclusion
The soleus is no longer the "forgotten" muscle of the lower limb. Its intricate aponeurotic architecture and unique physiological profile allow for extraordinary force production, yet these same features create a "hidden" vulnerability. The central challenge for the sports medicine team lies in the biological mismatch between the rapid restoration of muscle function and the protracted timeline required for aponeurotic remodelling. To mitigate the high risk of recurrence, practitioners must look beneath the surface of clinical symptoms and early running milestones. By adopting a framework that respects tissue healing timelines and prioritises long-term aponeurotic robustness, we can move beyond reactive management toward a more resilient model of calf health in the elite game.

Key Messages for Football Practitioners
- Soleus-dominant CMSIs are increasingly common in professional football and carry significant time-loss and recurrence risk.
- The complexity of soleus aponeurotic anatomy and variable anatomy between players have important implications for injury location, risk and rehabilitation planning.
- A central challenge is the delay between early recovery of muscle/running capacity and the slower maturation of aponeurotic scar tissue.
- The two frameworks presented–**healing and re-injury risk over time** (Figure 4) and a **phase-based rehabilitation model** (Figure 5)–offer practical tools to align loading, monitoring and RTP decisions with underlying tissue biology in the elite football environment.

Abbreviations:

ACL: Anterior cruciate ligament

CMSI: Calf muscle strain injury

EMG: Electromyography

FGA: Free gastrocnemius aponeurosis

IMA: Intramuscular aponeurosis

MAS: Maximal aerobic speed

MRI: Magnetic resonance imaging

MTU: Muscle-tendon unit

NMES: Neuromuscular electro-stimulation

RTP: Return to play

SSC: Stretch-shortening cycle

SL: Single-leg

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