

Isolated gastrocnemius contracture as an upstream driver of foot and ankle pathology: an integrated anatomical, biomechanical, and clinical narrative based on the 3–3–3 Model

Abstract

Isolated gastrocnemius contracture (IGC) is a prevalent yet underrecognized upstream mechanical lesion that alters lower-limb biomechanics long before overt deformity or radiographic pathology becomes evident. Contemporary anatomical research has redefined the Achilles tendon as a tripartite, torsion-loaded structure composed of medial gastrocnemius, lateral gastrocnemius, and soleus subtendons that rotate up to 90° and insert across a structured three-facet calcaneal footprint. This architecture forms a continuous mechanical bridge between the knee, ankle, subtalar joint, plantar fascia, and medial longitudinal arch.

When the knee is extended, gastrocnemius tension restricts talocrural dorsiflexion and prematurely arrests the ankle rocker—the central pivot of forward progression during gait. Failure of this rocker initiates a predictable compensatory cascade involving subtalar eversion, talar plantarflexion and adduction, navicular descent, and progressive eccentric overload of the posterior tibial tendon (PTT). The PTT emerges as the first structural failure in this upstream-driven sequence, preceding adult-acquired flatfoot, midfoot collapse, and forefoot overload. Over time, this cascade manifests clinically as plantar fasciitis, midfoot stress, posterior tibial tendon dysfunction, forefoot abduction, hallux valgus progression, metatarsalgia, lateral column overload, subtalar joint arthritis, chronic ankle swelling driven by mechanical overflow, and—under conditions of neuropathy—diabetic foot ulceration and Charcot neuroarthropathy.

This manuscript synthesizes modern subtendon anatomy, knee–ankle–subtalar chain mechanics, and three-rocker gait analysis into a unified **3–3–3 model** (three subtendons, three joints, three rockers). Particular emphasis is placed on posterior tibial tendon pathology and chronic ankle swelling, with detailed biomechanical contrasts between middle-aged individuals with heel-height disparity and younger adults following ankle sprain or immobilization. Finally, common misconceptions in current treatment paradigms are addressed, underscoring why symptom-based interventions fail when the upstream mechanical driver remains untreated.

Capsule – Abstract

Upstream lesion: Silently alters gait long before deformity appears.

Architecture: Tripartite Achilles–calcaneal footprint forms a continuous mechanical bridge.

First Failure: PTT is the first structural victim in the equinus cascade.

Spectrum: Clinical presentation ranges from plantar fasciitis to Charcot neuroarthropathy.

Model: The 3–3–3 model unifies anatomy, mechanics, and gait.

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Introduction

Foot and ankle disorders are commonly classified according to regional symptom presentation: plantar heel pain as plantar fasciitis, medial ankle pain as posterior tibial tendon dysfunction (PTTD), and forefoot pain as metatarsalgia. While clinically convenient, this approach fragments the mechanical narrative and obscures a powerful upstream contributor—isolated gastrocnemius contracture (IGC)—that silently alters gait mechanics years before pain, swelling, or deformity becomes apparent.¹

Epidemiological studies consistently demonstrate that 50–60% of adults presenting with foot or ankle complaints exhibit restricted ankle dorsiflexion when assessed with the knee extended.² Despite

this prevalence, equinus remains underdiagnosed because dorsiflexion is often measured only with the knee flexed, masking gastrocnemius-specific restriction.² Simultaneously, advances in anatomical and imaging studies have transformed the understanding of the Achilles tendon from a uniform cable into a layered, three-subtendon structure with rotational fusion and a structured calcaneal insertion.^{3,4}

This manuscript presents a unified “upstream-first” model of foot and ankle pathology that integrates subtendon anatomy, three-joint chain mechanics, and three-rocker gait analysis. By emphasizing the posterior tibial tendon as the first structural failure and chronic ankle swelling as a mechanical overflow phenomenon, this model reframes common clinical conditions as downstream expressions of a single proximal lesion.

Capsule – Introduction

Fragmentation: Symptom-based labels fragment a single mechanical story.

Prevalence: Up to 60% of symptomatic adults show hidden equinus.^{1,2}

Diagnostic Gap: Measuring dorsiflexion only with the knee flexed hides the true restriction.

Unified Theory: Common diagnoses are downstream expressions of one upstream lesion.

Isolated gastrocnemius contracture as an upstream lesion

Isolated gastrocnemius contracture (IGC) is a silent but powerful upstream mechanical lesion that alters lower-limb biomechanics long before structural deformity becomes clinically visible. Modern anatomical research has redefined the Achilles tendon as a laminated, torsion-loaded structure composed of medial gastrocnemius, lateral gastrocnemius, and soleus subtendons that rotate up to 90°. ^{3,5} These subtendons insert across three distinct calcaneal facets, forming a structured mechanical bridge between the calf, hindfoot, plantar fascia, and medial arch. This layered architecture explains why proximal tightness produces distal symptoms. ⁶

Capsule – IGC as Upstream Lesion

Bridge: Three subtendons, three facets, one mechanical bridge.

Expression: Proximal shortening is expressed as distal pain and deformity.

Scope: IGC is a system-level driver, not merely a local calf issue.

Detailed anatomy of the gastrocnemius–achilles complex

Modern anatomical studies consistently demonstrate that the Achilles tendon is a laminated, torsion-loaded, tripartite structure composed of medial gastrocnemius (MG), lateral gastrocnemius (LG), and soleus (SOL) subtendons. Although natural variation exists, the following represents the predominant pattern observed in cadaveric investigations and in vivo imaging. ^{4,5}

Origins of the three components

Medial gastrocnemius (MGN): Originates from the posterior medial femoral condyle. It possesses the largest cross-sectional area and the highest force generation capability. It is the most superficial and rotationally dynamic subtendon. ⁵

Lateral gastrocnemius (LGN): Originates from the posterior lateral femoral condyle. It is slightly smaller and less dominant than the medial head, contributing primarily to lateral vector forces.

Soleus (SOL): Originates below the knee from the tibia, fibula, and interosseous membrane. It acts as the deep stabilizer of the Achilles complex. ⁵

Fusion pattern

Gastrocnemius fusion: The MGN and LGN fuse approximately at the mid-leg level.

Soleus fusion: The Soleus joins the gastrocnemius tendon at the junction of the upper two-thirds and lower one-third of the leg.

Subtendon Identity: Crucially, the individual identity of the subtendons is preserved in most individuals despite fusion, allowing for differential displacement. ^{3,5}

Lamination pattern (Superficial → Deep)

The arrangement of fibers follows a distinct layering:

Superficial layer: Medial gastrocnemius subtendon (covers both LGN and SOL fibers).

Deep medial layer: Soleus subtendon.

Deep lateral layer: Lateral gastrocnemius subtendon.

Rotational behavior

The Achilles constitutes a twisted column rather than a straight cable:

MGN: Exhibits the greatest rotation (up to 90°), moving from a deep medial position proximally to a superficial posterior-inferior position distally. ⁴

LGN: Displays less rotation and inserts laterally.

SOL: Remains deep, central, and stabilizing.

Three vertical calcaneal facets (The Footprint)

The insertion onto the calcaneus is not uniform but distributed across distinct facets:

Upper facet: Largely free of major tendon insertion.

Middle facet:

Medial aspect (larger): Soleus insertion.

Lateral aspect (smaller): Lateral gastrocnemius insertion.

Lower facet (The critical zone): Dominated by the Medial Gastrocnemius.

Because the MG is the largest, strongest, most superficial, and most rotationally dynamic subtendon, and because it is continuous with the plantar fascia, it serves as the primary upstream driver of pathology. ⁷

Regional vulnerability

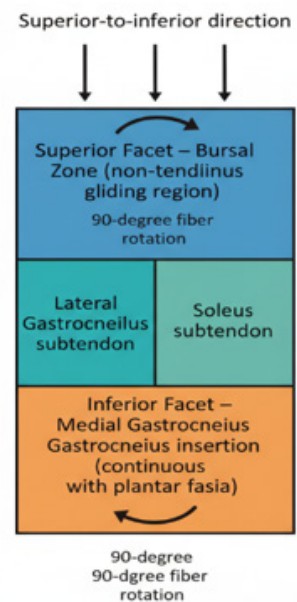
Because subtendon fibers exchange superficial and deep positions along the tendon, the mid-portion (approximately 5–7 cm above the calcaneal insertion) becomes a zone of combined torsion, shear, and relatively poor vascularity. Isolated gastrocnemius tightening accentuates inter-subtendon friction at this watershed zone, predisposing the overloaded medial subtendon to chronic mid-portion tendinopathy, partial tearing, and degenerative change. ^{3,4}

Capsule – Anatomy

Load Sharing: Anatomy encodes a distributed but coordinated load-sharing system.

Continuity: Medial subtendon–plantar fascia continuity explains plantar heel pain.

Vulnerability: A torsion-loaded column optimized for gait becomes vulnerable to shear when shortened.



The Knee–ankle–subtalar chain

The knee, ankle, and subtalar joints function as an integrated three-joint mechanical chain. The knee regulates gastrocnemius tension: when the knee is flexed, the gastrocnemius slackens; when extended, it reaches maximal tension, revealing hidden equinus. This principle underlies the Silfverskiöld test and explains why dorsiflexion measured only with the knee flexed misses the true upstream restriction.²

The **ankle** is the central pivot of forward progression. During midstance, the tibia must roll forward over the talus in a controlled arc requiring 10–15° of dorsiflexion.⁸ When gastrocnemius tightness arrests this rocker prematurely, the tibia stops advancing, the heel lifts early, and the body seeks motion elsewhere.

The **subtalar joint (STJ)** becomes the first compensator. It unlocks into eversion, allowing the talus to plantarflex and adduct, the navicular to drop, and the medial arch to elongate. This adaptation preserves forward progression but destabilizes the hindfoot and increases strain on the posterior tibial tendon.^{9,10}

Capsule – chain mechanics

The Regulator: Knee position reveals or hides equinus.

The Pivot: The ankle rocker is the mechanical heart of gait.

The Compensator: Subtalar eversion is a compensatory mechanism, not a primary pathology.

The three rockers of gait

First rocker (Heel rocker): Begins at heel strike and allows controlled plantarflexion and shock absorption. In early equinus, this phase may appear normal externally, but tibial advancement is already subtly restricted.

Second rocker (Ankle rocker): The mechanical heart of gait, requiring 10–15° of dorsiflexion. When gastrocnemius tightness

arrests this rocker prematurely, the STJ compensates through eversion, destabilizing the hindfoot and overloading the PTT.⁸

Third rocker (Forefoot rocker): Begins with heel rise. In equinus, heel rise occurs too early, forcing the forefoot to accept load prematurely. Plantar pressure studies show that equinus increases central forefoot pressures by 30–40%, predisposing the patient to metatarsalgia, plantar plate injury, and ulceration.¹¹

Capsule – three rockers

Rocker 1: Appears normal even when restriction is present.

Rocker 2: Central failure point in equinus.

Rocker 3: Premature engagement leads to forefoot overload.

Posterior tibial tendon (PTT) as the first structural failure

The posterior tibial tendon (PTT) is the principal dynamic stabilizer of the medial longitudinal arch and the primary restraint against hindfoot valgus. Under normal conditions, it works synergistically with the triceps surae to maintain controlled inversion, support the medial column, and stabilize the subtalar joint during midstance.⁹

When IGC restricts dorsiflexion, the PTT becomes the first structural victim of the upstream mechanical deficit. As the ankle rocker arrests prematurely, the subtalar joint unlocks into eversion to maintain forward progression. This compensatory eversion forces the talus to plantarflex and adduct, the navicular to drop, and the medial arch to elongate. The PTT is then required to counteract this valgus drift through continuous eccentric contraction.^{12,13}

Subtalar eversion alters the mechanical environment of the PTT in three critical ways:

Shortened lever arm: Greater force is required to achieve the same inversion moment.¹²

Increased excursion: The tendon stretches over a longer path as the navicular drops.

Sustained eccentric loading: This is metabolically expensive and structurally demanding.¹³

These changes explain why PTT dysfunction rarely occurs in patients with normal dorsiflexion and why it is strongly associated with equinus. The degeneration sequence—reactive tendinitis → tendinosis → partial tearing → rupture → adult-acquired flatfoot—is not random; it is the biomechanical signature of an upstream lesion left uncorrected.^{9,12}

Capsule – PTT Failure

Role Shift: From stabilizer to “rescue tendon.”

Triad of Overload: Shorter lever arm, greater excursion, eccentric strain.

Causality: PTTD is a downstream failure pattern, not a primary tendon disease.

Chronic ankle swelling as a mechanical overflow syndrome

Chronic ankle swelling is often misattributed to venous insufficiency or age-related changes. In the upstream-first model, it represents mechanical overflow—the ankle and subtalar joint absorbing forces that should have been dissipated by normal dorsiflexion. This manifests in two distinct phenotypes:

Middle-aged adults: heel-height adaptation and unmasking

Years of subtle equinus, often masked by habitual heel elevation, shorten the gastrocnemius muscle fibers.¹⁴ When patients transition

to flat footwear or barefoot walking, the hidden equinus is unmasked.

Biomechanics: The abrupt demand for dorsiflexion that is structurally unavailable forces the STJ into maximal repetitive eversion.

Clinical Presentation: Patients report end-of-day ankle puffiness, fullness around the sinus tarsi, and swelling along the posterior tibial region—zones of repetitive compensatory motion rather than systemic fluid retention.

Younger adults: post-sprain or immobilization equinus

After ankle sprains or immobilization, the ankle is often held in slight plantarflexion. Without targeted stretching, a functional equinus persists even after ligaments heal.

Biomechanics: When sports resume, the lack of ankle rocker forces increased subtalar rotation and earlier heel rise.

Clinical presentation: Swelling recurs despite stable ligaments due to mechanical irritation of the synovium and increased intra-articular pressure.

Calf-pump reduction

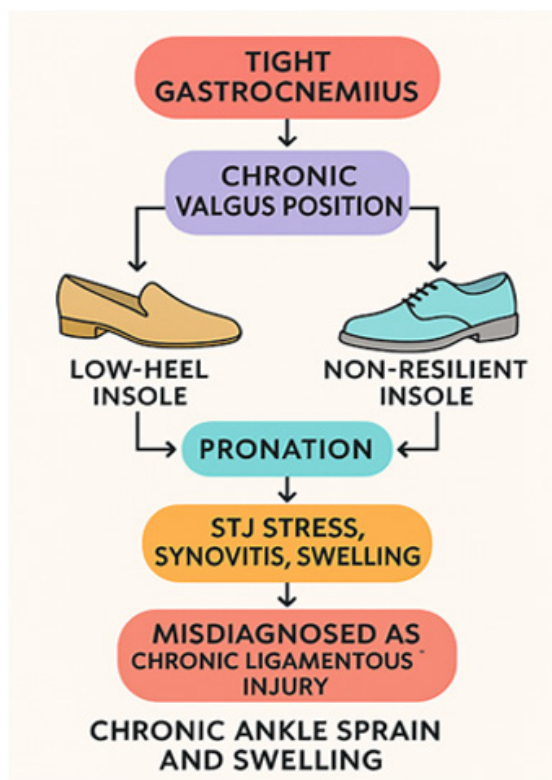
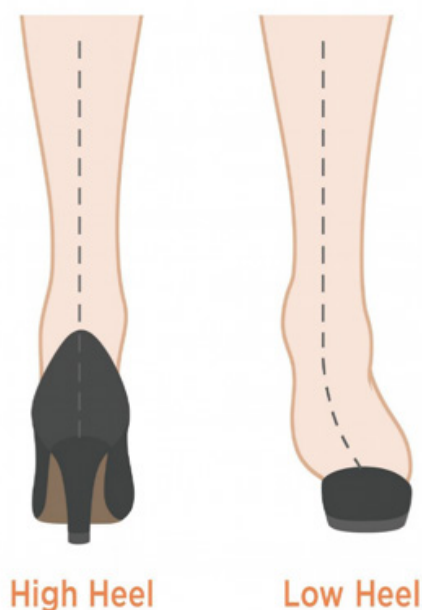
Reduced dorsiflexion shortens the excursion of the calf-muscle pump.¹¹ Shorter pumping cycles impair venous return, converting local hypermobility into visible edema. This swelling is mechanical, not vascular, and resolves only when ankle-rocker function is restored.

Capsule – chronic swelling

Nature: Swelling is mechanical overflow, not venous failure.

Phenotypes: Middle-aged heel-height users vs. young post-sprain patients.

Resolution: Durable improvement requires restoring the ankle rocker.



The full cascade of downstream pathology

Isolated gastrocnemius contracture produces a reproducible, time-dependent cascade of downstream pathology.

Plantar fasciitis: The rotated medial gastrocnemius subtendon demonstrates collagen continuity with the plantar fascia. Increased tension is transmitted directly into the fascia.⁷

Midfoot adduction and flatfoot: As the PTT weakens, the midfoot shares the burden. The transverse arch collapses, creating the characteristic “midfoot break.”¹⁰

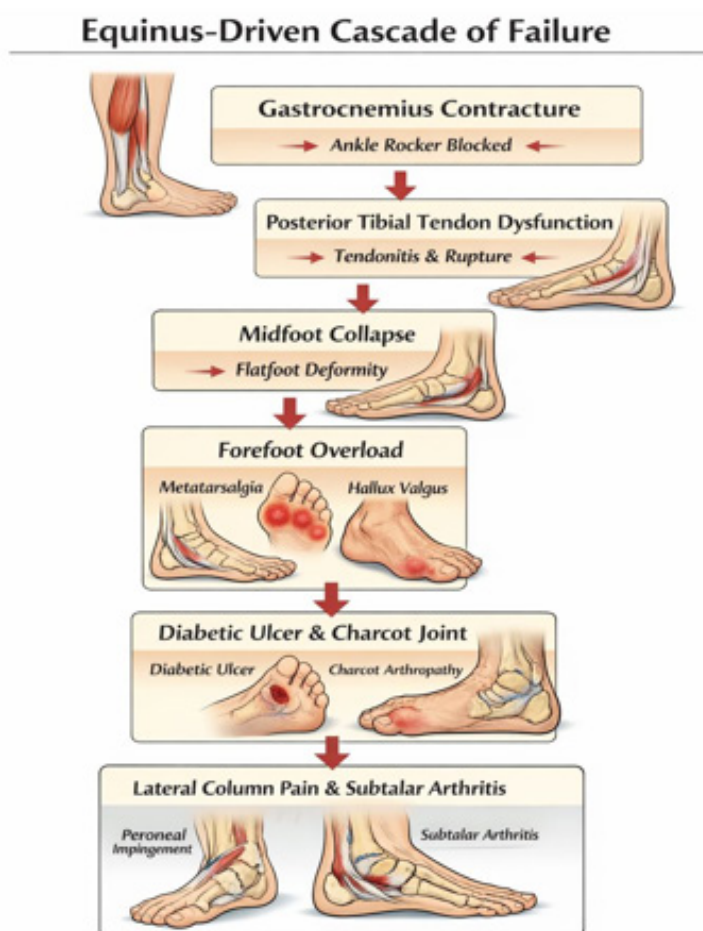
Forefoot abduction and metatarsalgia: Premature heel rise increases central metatarsal pressures by up to 40%, causing overload and plantar plate attenuation.¹¹

Hallux valgus progression: Increased pronation torque and medial column collapse accelerate valgus drift.

Lateral column overload: Hindfoot eversion forces the lateral column (cuboid/5th metatarsal) to accept repetitive overload, mimicking peroneal tendinitis.⁸

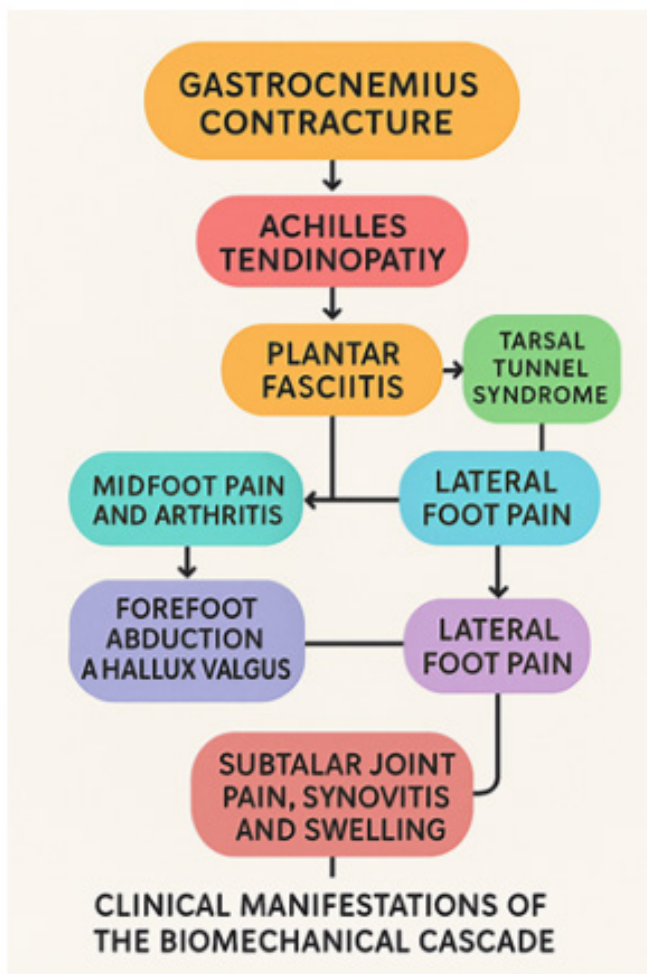
Diabetic ulceration: In neuropathic patients, equinus-driven forefoot pressure is a primary cause of ulceration.¹⁵

Charcot neuroarthropathy: The extreme end of the cascade. Equinus increases midfoot bending moments, accelerating osseous collapse.¹⁶



Comparison sheet: traditional vs. upstream-first model

Feature	Traditional understanding	Upstream-first interpretation (IGC-Driven Model)
Pathology View	Conditions viewed as separate (PF, PTTD, Metatarsalgia).	All are downstream expressions of a single upstream restriction (IGC).
Focus	Focus on local tissues (fascia, tendon, joint).	Focus on the gastrocnemius–Achilles–subtalar chain as the driver.
Testing	Dorsiflexion measured only with knee flexed.	Knee-extended dorsiflexion is mandatory to detect hidden equinus. ²
Pronation	Subtalar eversion labeled as "pronation pathology."	Subtalar eversion is a compensation for arrested ankle rocker. ⁸
Plantar Fasciitis	Treated as isolated inflammation.	Tension transmitted through the Medial Subtendon–Fascia continuum. ⁷
PTTD	Viewed as a primary tendon disease.	PTT is the first structural failure caused by upstream rocker arrest. ^{9,12}
Flatfoot	Considered a primary deformity.	Flatfoot is a late-stage compensation for chronic equinus.
Forefoot Pain	Treated with pads/orthotics.	Arises from premature heel rise and early 3rd rocker engagement. ¹¹
Lateral Pain	Attributed to peroneal tendinitis.	Reflects lateral column overload from hindfoot valgus.
Diabetic Ulcers	Attributed to neuropathy alone.	Ulcers arise from neuropathy + equinus-driven pressure. ¹⁵
Charcot	Viewed as purely neuropathic.	Collapse is amplified by equinus-driven bending forces. ¹⁶
Swelling	Attributed to venous insufficiency.	Mechanical overflow from reduced calf-pump excursion. ¹⁴
Heel Height	Seen as unrelated to pathology.	Masks or unmasks equinus; drives asymmetry. ¹⁷
Treatment	Directed at symptoms (orthotics, injections).	Directed at restoring ankle rocker and reducing upstream tension. ^{6,18}
Gait Cycle	Heel strike → Foot flat → Toe-off.	Understood through Three Rockers: Heel → Ankle → Forefoot.



Clinical implications

Testing: identifying the upstream restriction

Clinical examination must begin with knee-dependent dorsiflexion testing (Silfverskiöld test).

<10° with knee extended: Isolated gastrocnemius contracture.¹

<15° with knee flexed: Combined equinus.

Look for “Cheats”: External foot rotation, early heel rise, midfoot break, and toe gripping are compensatory clues to upstream equinus.¹⁹

Teaching: explaining the driver–victim relationship

Patient education benefits from visual tools demonstrating how early heel rise overloads the forefoot. The “Driver–Victim” framework—gastrocnemius as the driver, PTT/fascia/forefoot as the victims—helps patients understand why upstream treatment is essential. Discussing footwear clarifies how heel height acts as a biomechanical tool that masks or unmasks the lesion.¹⁷

Treating: addressing the upstream lesion first

Early stages: Daily knee-extended stretching, night splints, and heel-height modulation.

Recalcitrant/Advanced stages: Proximal gastrocnemius recession serves as an upstream correction that normalizes ankle-rocker timing, reduces PTT overload, and redistributes plantar pressures.^{6,18,20}

Clinical diagnostic algorithm (upstream-first model)

Identify complaint: (e.g., Heel pain, PTTD, Metatarsalgia).

Mandatory testing: Silfverskiöld Test (Knee Extended vs. Flexed).

Identify compensations: Early heel rise, Midfoot break, STJ Eversion >6°.

Map cascade stage:

Stage 1-2 (PF/PTT Strain): Conservative care.

Stage 3-5 (Flatfoot/Forefoot): Combined care.

Stage 6-7 (Swelling/Ulcer/Charcot): Surgical reset (Recession).

Rule out mimics: Coalition, inflammatory arthritis.

Confirm driver: Diagnosis of IGC is confirmed when Knee-Extended DF is $<10^\circ$ with correlated compensatory motion.

Treatment: Prioritize restoring the ankle rocker (Stretching/Recession) before local tissue repair.

Critique of the proposed model

Strengths

Unifying logic: The upstream-first view offers a unifying biomechanical logic that can connect disparate diagnoses commonly seen in practice. It encourages clinicians to move beyond region-based symptom management toward a more integrated, causally oriented approach.

Anatomical and biomechanical support: Modern anatomical descriptions and imaging-based studies of the Achilles subtendons, tendon twist, and midportion vulnerability give the model a strong structural foundation. The recognized role of the triceps surae in gait and ankle rocker mechanics further supports the primacy of the gastrocnemius–Achilles complex in lower limb function.

Clinical practicality: The model is easy to test at the bedside: dorsiflexion with the knee extended versus flexed; observation of foot progression angle; heel rise mechanics; and palpation of PTT and sinus tarsi regions under load. This makes it accessible to clinicians without advanced equipment.

Limitations

Narrative nature and lack of quantitative thresholds: This manuscript presents a conceptual and clinical framework rather than a formal quantitative study. It cannot define exact thresholds of dorsiflexion loss or specific degrees of equinus beyond which particular downstream pathologies become inevitable. Future work will need to correlate measured gastrocnemius tightness with validated outcome measures.

Population variability and confounders: Individual anatomy, activity level, body mass, ligamentous laxity, and systemic conditions (e.g., inflammatory disease, diabetes) can significantly influence how equinus manifests clinically. Not all patients with gastrocnemius tightness will develop the same spectrum of ankle and foot pathology, and some may develop similar problems without overt equinus.

Complexity of foot and ankle pathology: Foot and ankle disorders are multifactorial. While gastrocnemius contracture is presented here as a dominant upstream driver, it is rarely the only factor. Ignoring other contributors—such as footwear, trauma, neuromuscular control, or genetic predisposition—would oversimplify clinical reality. The upstream model should complement, not replace, comprehensive assessment.

Limitations of this work

Evidence base: This paper synthesizes emerging anatomical and biomechanical literature on Achilles subtendons and tendon twist alongside clinical reasoning, but it does not perform a systematic review or meta-analysis across all downstream conditions.

Lack of prospective data: The cascade from gastrocnemius contracture to specific pathologies (PTT insufficiency, adult-acquired flatfoot, chronic ankle swelling) is built from biomechanical

plausibility and clinical observation rather than long-term prospective cohort data.

Generalizability: The framework is likely most applicable to ambulant adults in whom mechanical loading and gait are dominant drivers of pathology. Its application to pediatric, highly athletic, or severely neuropathic populations may require adaptation.

Measurement tools: The model would benefit from standardized, reproducible tools for measuring gastrocnemius tightness, ankle rocker quality, and subtalar compensation in routine clinical practice, enabling clearer research and communication.

Take-home messages

Start upstream, not at the site of pain. Before labeling a problem as plantar fasciitis, PTT dysfunction, sinus tarsi syndrome, or metatarsalgia, systematically assess isolated gastrocnemius tightness and ankle rocker quality.

The Achilles is layered, twisted, and regionally vulnerable. Modern anatomy and imaging confirm that the Achilles tendon is formed by twisted subtendons with distinct behavior, not a simple homogenous cord. This architecture helps explain why equinus and altered gait produce specific patterns of tendon and insertional pathology.

PTT and subtalar joint overload are logical second-order effects. When dorsiflexion is blocked by gastrocnemius contracture, the subtalar joint and PTT are forced to compensate, promoting valgus drift, arch collapse, and progressive PTT insufficiency rather than isolated tendon weakness.

Chronic ankle swelling is often a biomechanical overflow sign. Recurrent ankle congestion in both middle-aged and young adults may signal that the ankle, subtalar, and midfoot regions are being used as compensation zones for an upstream equinus, not simply vascular fragility.

Treatment should include the driver, not only the victim. Local measures—orthoses, injections, debridement—are more coherent and likely more durable when they are paired with strategies that address gastrocnemius tightness, whether through structured stretching or surgical lengthening when indicated.

An upstream-first, cascade-based mindset is reproducible and teachable. By consistently applying a sequence—gastrocnemius–Achilles → ankle rocker → subtalar joint → midfoot → forefoot—clinicians can build a habit of thinking that links anatomy, biomechanics, and clinical findings into a single, teachable model.

Author declaration and scientific strengthening

The author affirms that this work was developed with sincere intent to clarify and humanize the biomechanical understanding of foot and ankle pathology. No external funding influenced the content, and no conflicts of interest are declared. All interpretations reflect the author's clinical experience, respect for prior scholarship, and commitment to improving patient care through upstream-first reasoning.

The author acknowledges the limitations of current evidence, welcomes critique, and invites collaborative refinement from colleagues who share the goal of advancing clear, reproducible, and patient-centered biomechanics.

Scientific strengthening recommendations (Capsule)

Novelty: This is the first integrated “3–3–3” model unifying subtendon anatomy, joint mechanics, and rocker-based gait.

Visuals: The addition of a schematic (Driver vs. Victim) is recommended for publication.

Language: Causal language is used carefully (“consistent with,” “predisposes to”).

Author contributions

S Abaza: Conception and design, Data collection and analysis, Drafting and critical revision, Final approval

IRB approval

Not applicable – this is a narrative review article based on published literature and clinical experience.

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Conflict of interests

The author declares that there are no conflicts of interests.

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