QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

RADLIŃSKI, Michał, PALACZ, Jakub Paweł, PAPCIAK, Kinga Zofia, WOŹNIAK, Klaudia, JABŁOŃSKA, Urszula Zofia, GARDZIELIK, Aleksandra, KOROWAJ, Karolina, DERCZ, Maciej Piotr, FITAS, Alicja and GŁASZCZKA, Michał. Consequences for the Contralateral Limb: Multifactorial Risk Factors, Biomechanical Overload, and Structural Degeneration Following Achilles Tendon Rupture: A Narrative Review. Quality in Sport. 2026;56:72598. https://doi.org/10.12775/QS.2026.56.72598

ARTICLE TIMELINE
Received: 25.05.2026 Revised: 28.05.2026
Accepted: 28.05.2026 Published: 30.05.2026

INDEXING & EVALUATION
MEiN points: 20 Unique ID: 201398
Disciplines: Economics & Finance; Management & Quality Sciences

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Economics and Finance (Social Sciences); Management and Quality Sciences (Social Sciences).

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Consequences for the Contralateral Limb: Multifactorial Risk Factors, Biomechanical Overload, and Structural Degeneration Following Achilles Tendon Rupture: A Narrative Review

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Abstract

Background. Long-term functional deficits are a harsh reality after an acute Achilles tendon rupture (ATR). Standard care usually targets just the torn tissue. The problem is that it completely ignores the wider biomechanical chain. When calf weakness persists, patients develop a major asymmetry in how they move. As a result, the opposite, seemingly healthy leg is forced to take on an excessive and chronic mechanical burden.

Aim. To identify mechanisms of compensatory overload, evaluate biomechanical and structural changes in the contralateral healthy limb, and assess psychological factors following a unilateral ATR.

Material and methods. A PubMed literature review synthesized clinical, systematic, and biomechanical studies (2012-2026). The analysis evaluated the bilateral impact of ATR, focusing on compensatory kinematics, psychological barriers, and morphological alterations in the uninjured tendon.

Results. Athletes subconsciously shift their kinematics to shield the repaired tendon. This abnormal load redistribution, driven by a lack of psychological readiness, prevents the restoration of limb symmetry. Consequently, the contralateral limb suffers chronic, unphysiological overload. Objective imaging confirms this stress causes silent structural degeneration in the asymptomatic tendon, manifesting as morphological hypertrophy on ultrasonography and pathological tissue softening on sonoelastography.

Conclusions. A unilateral ATR initiates a bilateral pathological cascade. Driven by psychological barriers and altered biomechanics, the healthy limb undergoes chronic loading and structural degeneration, elevating the risk of a contralateral rupture. Return-to-play algorithms must adopt a holistic bilateral paradigm integrating neuromuscular symmetry training, psychological screening, and routine imaging of the asymptomatic tendon.

Key words: Achilles tendon rupture, contralateral overload, kinesiophobia, sonoelastography, ultrasonography

1. Introduction

The Achilles stands as the thickest and most powerful tendon in the human body, acting as the critical force conduit for walking, running, and jumping [1,2,3]. Relying on a hierarchical network of type I collagen, it boasts extreme tensile strength and resilience, working much like a high-efficiency spring to store energy during fast movements [3,4]. Despite having such a resilient structure, acute Achilles tendon rupture (ATR) is a devastating musculoskeletal injury with a steadily growing incidence rate in active and athletic groups [1,5]. Getting back to pre-injury fitness after an ATR is incredibly difficult, largely because patients deal with stubborn calf weakness, stretched tissues, and noticeable functional limits for years [2,5,6]. Because their movement remains so asymmetrical, a massive mechanical load gets shifted entirely onto the uninjured, opposite limb [7,8]. Ultimately, this is why 5% to 7% of primary ATR patients go on to suffer an asynchronous tear on the other side, leaving them with substantially poorer function, reduced activity levels, and much lower satisfaction compared to a unilateral rupture [7].

Driven by these clinical challenges, our narrative review mainly aims to uncover the exact mechanisms behind compensatory overload, analyze the structural and biomechanical shifts inside the opposite healthy leg, and evaluate the psychological hurdles athletes face after a single-sided ATR [7,8,9,10]. In particular, we investigate the ways the initial trauma forces patients into abnormal movement patterns, completely shifting mechanical stress onto the intact tendon [8]. We also look closely at kinesiophobia - the intense fear of moving and getting hurt again - and show how it actively sustains this physical imbalance, heavily overloading the healthy extremity over time [11,12]. Ultimately, by pulling together the latest data, we want to prove that this constant extra stress triggers a silent tissue decay, appearing as abnormal thickening and dangerous internal softening, which ruins the long-term integrity of the uninjured leg and heavily spikes the danger of a second tear [7,10,13].

2. Research materials and methods

In order to conduct this narrative review, a comprehensive literature search was performed using primary scientific databases, including PubMed. The search targeted peer-reviewed articles published predominantly between 2012 and 2026 to ensure the inclusion of the most recent clinical data. The selected literature primarily encompassed recent clinical studies,

systematic reviews, and biomechanical analyses. Keywords utilized in the search strategy included: “Achilles tendon rupture”, “contralateral overload”, “kinesiophobia”, “sonoelastography”, “ultrasonography”. Articles not published in English were excluded from the synthesis.

3. Kinematic Compensations, Biological Factors, and the Risk of Secondary Injury

An acute Achilles tendon rupture (ATR) is a severe physical setback, sparking biological and biomechanical shifts that affect much more than just the initially injured leg [5,8]. Because the repaired tissue rarely bounces back to its full pre-injury capacity, patients instinctively change how they move, which places a harsh, chronic strain on the uninjured, contralateral side [5,8]. This creates a clear domino effect, massively driving up the chances of suffering a secondary, nonconcurrent tear [7,14]. In the sections below, we unpack how altered lower extremity kinematics, stubborn limb asymmetry, and personal biological factors work together to build a highly dangerous environment for the supposedly healthy leg [7,12,15].

3.1. Altered Kinematics and Contralateral Overload

Following an Achilles tendon rupture (ATR), athletes naturally adopt lasting changes in how they walk and run, mostly to protect the newly repaired tissue [8,16]. By using 3D motion capture technology and force plates, researchers can break down exactly how these leg mechanics shift during the stance phase [8]. Right from the moment the foot hits the ground, patients show a totally altered chain of movement. They essentially stiffen the leg to avoid stressing the weak, stretched calf muscle, but this exact strategy ruins the limb's normal ability to absorb impacts [1,17]. Looking at the kinetic data, the ankle loses a massive amount of its power output. On the injured side, patients struggle to push off properly and can rarely flex their foot down as far as they used to [5,18]. This sharp drop in force and power ties directly back to measurable muscle weakness and the physical lengthening of the tendon [14]. Because the injured ankle fails to generate enough forward drive or move through a full range, the body has no choice but to shift that physical stress elsewhere - especially onto the opposite leg [1,8]. By keeping the injured leg stiff and limiting what it can do, patients unknowingly push all the heavy impact and propulsion duties onto the healthy side [8,16]. This severe imbalance in how forces are handled, along with the constant shifting of energy, forces the intact leg to deal with

a chronic, unnatural amount of strain [8,17]. In the end, relying on these mechanical shortcuts puts the healthy leg at a serious disadvantage, setting off a chain reaction of abnormal stress that outlasts the initial healing period by far [8,16].

3.2. Limb Symmetry Index (LSI) and Leg Dominance

Recovering from an Achilles tendon rupture (ATR) leaves most patients with a clear functional gap between their legs, a metric clinicians monitor through the Limb Symmetry Index (LSI) [2,12]. Basic clinical exams, notably the single-leg heel-rise test, show that severe strength deficits and physical limits easily stick around for months or years after the trauma [5,6]. In surgical cases, for instance, the heel-rise work LSI frequently stalls at just 64% after six months and barely reaches 75% a full year out [6]. The inability to hit perfect symmetry generally stems from the healing tendon stretching out, an elongation that alters the muscle's resting length and permanently limits calf force output [5,17]. Kinetic assessments back this up by showing how the lasting asymmetry directly aligns with severe, stubborn drops in peak ankle power on the injured side [18]. Restoring muscle function and side-to-side balance also relies heavily on whether the injury hit the athlete's dominant or non-dominant leg [2]. Because dominant and non-dominant limbs naturally differ in baseline balance and strength, a dominant-side tear becomes an incredibly difficult rehab hurdle [2]. With the primary limb compromised, the non-dominant leg must quickly take over and absorb brutal kinetic forces during sports [2,8]. Forcing this sudden, chronic mechanical stress onto the uninjured leg heavily worsens lower-body asymmetry during high-impact tasks like running, landing, or jumping [8,12]. Failing to achieve proper symmetry confirms that the repaired tissue never got its pre-injury strength back, turning the abnormal force shift into a permanent reality [5,8]. Operating with this constant imbalance creates a toxic mechanical environment that relentlessly overworks the healthy leg, severely accelerating structural decay and the threat of a secondary tear [8,12].

3.3. Sex Differences in Biomechanics and the Risk of Contralateral Rupture

Mechanical overload hits everyone after an ATR, but raw biology ultimately dictates who actually suffers a second tear [8,15]. The data makes one thing obvious: men suffer Achilles injuries way more often than women [15]. Digging into the biomechanics reveals huge differences between male and female tendon length, thickness, cross-sectional area (CSA),

baseline stiffness, force limits, and load reactions [15]. Male tendons basically act much stiffer and refuse to stretch the way female tissue does [15]. Struggling to yield under pressure leaves men with a severe, built-in physical weakness for this type of injury [15]. Hormones drive a lot of this difference, mainly because weight-bearing tendons are loaded with estrogen receptors [6]. In females, estrogen actively dials back collagen production, alters tissue metabolism, and keeps overall muscle stiffness much lower [6,15]. A compromised leg ruins the body's normal running form, instantly throwing massive mechanical stress onto new areas [8]. Shoving all that extra force onto the healthy side creates a highly toxic environment for the opposite tendon in men, simply because their stiffer tissue cannot handle sudden workload spikes [8,15]. Patient records prove secondary tears are overwhelmingly a male issue, best highlighted by one bilateral study where men made up an incredible 94.1% of the cases [7]. Females often endure a much worse functional recovery - reflected in terrible heel-rise symmetry a year later - yet their highly flexible tendons easily soak up that extra stress and keep the second side intact [6,15].

3.4. Multifactorial Risk Factors and Prognosis of Contralateral Rupture

Surviving a primary, unilateral acute Achilles tendon rupture (ATR) leaves the healthy leg to handle massive biomechanical overload, directly driving up the well-known risk of eventually tearing the opposite side [8,14]. Doctors call this a nonconcurrent bilateral Achilles tendon rupture, a devastating secondary injury that hits roughly 5% to 7% of patients who already tore their first tendon [7]. Looking closely at the numbers, population data shows that 5.5% of initial rupture cases end up tearing the contralateral Achilles later on, usually striking around 3.3 years - anywhere from 1.0 to 8.3 years - after the first trauma [19]. But simple mechanical stress does not tell the whole story, since the root cause of this second tear heavily involves a mix of anatomical flaws, genetics, and daily work habits. Case files tracking college athletes actually suggest that tearing both sides often comes down to built-in physical defects, like bad ankle alignment or poor blood flow right in the tendon's critical watershed zone [20]. Researchers are also digging deep into genetic red flags for these opposite-side injuries [21]; taking blood type O as an example, adults with this trait carry a massive, unadjusted hazard ratio of 3.71 for suffering a contralateral tear [19]. Pushing the body through heavy daily labor makes things even worse, driving the risk of tearing the other Achilles up by nearly five times (adjusted hazard ratio of 4.687) for people like manual workers, soldiers, firefighters, and farmers [19]. Once that nonconcurrent second tear actually happens, a patient's long-term

prognosis takes a massive hit, placing any athlete's performance and career lifespan in serious jeopardy [7,22]. Medical records across both everyday people and elite sports stars paint a brutal picture of this complication. One massive eight-year review of NFL players tracked 11 men who tore multiple Achilles tendons, and every single one of those secondary injuries happened exclusively on the opposite leg [22]. Backing this up in the general public, a clinical review of 222 acute ATR patients found 17 people who eventually suffered this exact bilateral trauma [7]. Matching them up against patients who only tore one side, the bilateral group reports drastically worse physical outcomes in the intermediate term [7]. They score terribly on the Achilles tendon Total Rupture Score (ATRS), lose a ton of basic ankle function, and end up deeply unhappy with what their bodies can still do [7]. Managing a bilateral tear is an absolute nightmare for rehabilitation [20], making it incredibly urgent for doctors to spot high-risk patients early and actively block that second rupture from ever happening [7,22].

Table 1. Summary of intrinsic and extrinsic risk factors predisposing to nonconcurrent contralateral Achilles tendon rupture.

Risk Factor	**Category**	**Clinical Impact and Increased Risk**
Male Sex	Intrinsic (Biological)	Men exhibit inherently stiffer tendons with lower compliance, reducing their capacity to safely absorb compensatory loads.
Blood Type O	Intrinsic (Biological)	Associated with a nearly four-fold increased risk (unadjusted hazard ratio of 3.71) of sustaining a secondary contralateral rupture.
Genetic Predispositions	Intrinsic (Genetic)	Genetic factors are actively being investigated as underlying risk factors that increase the susceptibility to contralateral injuries.

Anatomical Vulnerabilities	Intrinsic (Anatomical)	Abnormalities in ankle alignment and the critical watershed area of blood supply heavily influence bilateral tear propensity.
Demanding Occupation	Extrinsic (Environmental)	Heavy physical labor (e.g., military, farming, manual labor) increases the risk of opposite tendon tearing by nearly five-fold (adjusted hazard ratio of 4.69).

Source: Own elaboration based on current literature

4. Structural Degeneration in the Asymptomatic Contralateral Tendon

A chronic and hazardous burden is placed on the uninjured leg by the unphysiological redistribution of mechanical forces detailed previously [8]. These compensatory shifts are strictly necessary for basic locomotion, yet a significant biological cost is ultimately paid. According to clinical evidence, the increased rate of secondary opposite-side ruptures is heavily driven by degenerative changes developing inside the supposedly healthy tissue [7]. A silent cascade of structural and morphological deterioration is triggered by continuous mechanical overload, regardless of whether the contralateral Achilles tendon remains clinically asymptomatic and fully functional [10,13]. Modern diagnostic imaging must be utilized to examine the asymptomatic tendon if we want to fully capture this invisible degeneration [1]. Specific tissue adaptations that gradually compromise structural integrity, ranging from outward morphological hypertrophy to intrinsic softening, are detailed in the subsequent sections [10].

4.1. Ultrasonographic Abnormalities and Morphological Hypertrophy

Compensatory overload physically drives the asymptomatic, contralateral Achilles tendon into a state of severe structural adaptation and decay [10]. Medical reviews consistently highlight standard ultrasonography as an ideal tool for capturing these exact tissue changes [1]. Putting this supposedly healthy tendon under an ultrasound probe exposes clear morphological shifts driven directly by chronic mechanical strain [10]. The structure itself gains mass and physically

thickens. Morphological hypertrophy serves as a classic bodily reaction to extreme physical loading [3,23], proving that flawed tissue remodeling and active degeneration are secretly occurring despite a total lack of clinical symptoms [10].

Surviving this compensatory workload rarely happens without triggering much deeper structural damage. Looking at the actual patient data, doctors can already spot hidden ultrasound abnormalities in the opposite leg for 9 out of every 75 acute Achilles rupture cases [13]. Scans of these damaged tissues expose dark hypoechoic lesions, hard calcifications, and a completely chaotic breakdown of the tendon's natural parallel fibers [13]. Pushing imaging technology further exposes pathological neovascularization - a massive overgrowth of tiny blood vessels that acts as an early red flag for tissue decay, creeping in without causing any pain at all [3]. Letting these physical flaws worsen practically guarantees that visible clinical symptoms will soon follow [13]. Anyone walking around with these specific ultrasound defects faces an almost eight-fold higher threat (odds ratio of 7.88) of feeling severe local tenderness in their supposedly healthy tendon [13]. Routine sonographic scans essentially prove that the opposite Achilles is physically crumbling long before it actually snaps a second time [10,13].

4.2. Sonoelastography and Asymptomatic Tissue Softening

Standard ultrasound easily spots outward morphological swelling in the opposite Achilles [1], yet it completely fails to measure deep material properties like true tissue elasticity or the stress-strain relationship [3,10]. Overcoming this blind spot requires axial-strain sonoelastography (ASE), an imaging tool built to accurately map out and measure the exact hardness of the tissue [10]. ASE scans clear up a major physical paradox by proving that outward tendon enlargement actually hides a dangerous structural weakness [10]. Elastography data exposes a severe, totally painless softening buried deep inside the seemingly healthy, intact tendon of patients recovering from a recent tear [10]. The breakdown avoids spreading evenly across the structure, instead hitting the proximal third of the Achilles with a massive, statistically significant drop in tissue density [10]. Losing this core hardness signals an active breakdown of the extracellular matrix, which directly ruins the tendon's natural baseline stiffness and stretch [3,15]. The end result is a highly deceptive physical unit that looks visibly thicker on the surface but acts like a soft, mechanically inferior tissue on the inside. Rotting away so quietly destroys the limb's ability to safely absorb abnormal kinetic shocks [3,8], leaving the compromised tendon incredibly vulnerable to a second rupture as it constantly battles compensatory overload [10,15].

Table 2. Summary of structural and biomechanical alterations in the asymptomatic contralateral Achilles tendon following an acute rupture.

Diagnostic Modality	**Key Structural & Morphological Findings in the Contralateral Tendon**	**Biomechanical & Clinical Implications**
Ultrasonography	• Morphological hypertrophy (increase in tendon thickness). • Intratendinous hypoechoic lesions and calcifications. • Disrupted echostructure of parallel fiber bundles. • Asymptomatic pathological neovascularization.	Reflects ongoing degenerative changes and imperfect tissue remodeling. Progression of these structural abnormalities is strongly correlated with a nearly eight-fold higher risk (OR = 7.88) of experiencing local tenderness.
Axial-Strain Sonoelastography (ASE)	• Significant, asymptomatic tissue softening (severe reduction in hardness). • Softening is particularly pronounced and statistically significant in the proximal third of the tendon. • Pathological alteration of natural compliance and stiffness.	Resolves the paradox of the thickened tendon, demonstrating that enlargement masks a dangerous structural weakness. Compromises the ability to safely dissipate and absorb unphysiological kinetic energy, predisposing to secondary rupture.

Source: Own elaboration based on current literature.

5. Psychological Barriers and Kinesiophobia

Mental reactions to the initial trauma strongly dictate the final clinical recovery of any Achilles rupture patient [9]. Physical structures routinely finish healing long before an athlete actually regains their old motor habits, a delay caused entirely by stubborn psychological barriers [11]. Constantly limping and instinctively shielding the damaged leg both stem directly from an intense, underlying terror of moving and getting hurt again [9,11].

5.1. Fear-Avoidance Behavior and Altered Motor Patterns

Recovering from a torn Achilles requires much more than just waiting for the physical tissue to heal, as the athlete's mental reaction to the trauma heavily dictates the final outcome [9]. Kinesiophobia stands out as a massive mental roadblock for these patients, essentially acting as a paralyzing fear of basic movement driven by the constant dread of getting hurt again [11]. To actually measure how badly this fear-avoidance behavior derails the recovery process, sports medicine professionals routinely rely on the Tampa Scale of Kinesiophobia (TSK) [9,11].

This specific mental hurdle locks the patient into a cycle that actively ruins physical recovery. Athletes frequently develop avoidance behaviors due to reinjury anxiety, which physically corrupts their natural motor habits [11]. Trying to protect the repaired tissue usually leaves individuals with long-term changes to their gait and running form [8,14]. Shifting weight away from the injury out of fear forces the healthy opposite leg to absorb a massive, unphysiological amount of mechanical stress [8].

5.2. Impact on Limb Symmetry and Return to Play (RTP)

Perfect structural healing frequently fails to guarantee a competitive comeback because the aforementioned psychological barriers actively block the athlete's progress. Pooled statistics set the general return to play (RTP) rate for a torn Achilles right around the 80% mark [24]. Extreme professional environments like elite soccer face even worse attrition levels, as hard data shows 29.2% of fully treated surgical patients permanently drop out of the sport [1,25].

This failure is strongly driven by psychological factors; clinical studies demonstrate that low psychological readiness and persistent kinesiophobia directly correlate with inferior joint function and significantly lower patient-reported outcomes on the Achilles tendon Total

Rupture Score (ATRS) [9]. Furthermore, this fear of reinjury directly impedes the restoration of functional symmetry between the lower extremities. Athletes exhibiting high fear-avoidance struggle to recover their functional Limb Symmetry Index (LSI) during single-leg hop tests [9,11]. Returning to a high-demand athletic environment with unaddressed kinesiophobia and persistent limb asymmetry has severe consequences. Specifically, the failure to restore biomechanical symmetry solidifies the chronic overload on the uninjured limb [8], which - combined with the underlying tissue degeneration - significantly increases the risk of sustaining a secondary, contralateral Achilles tendon rupture [7].

6. Preventive Approaches and Rehabilitation Strategies

Tearing one Achilles fundamentally drives up the odds of eventually blowing out the opposite tendon, forcing clinicians to aggressively target and prevent these secondary injuries [7,13]. Modern treatment algorithms tackle this exact threat by using hard, objective imaging scans to map out highly personalized recovery plans [26]. Actually getting an athlete safely back into high-demand sports depends entirely on early functional rehab and intense neuromuscular drills to forcefully restore physical balance between the legs [2,26].

6.1. Bilateral Clinical Assessment and Preventive Monitoring

Skyrocketing rates of opposite-side injuries make strict clinical tracking of the symptom-free leg mandatory to block secondary tears [7]. Initial ultrasound scans right at the moment of the primary rupture typically show a healthy contralateral tendon, but the subsequent wave of compensatory overload demands relentless monitoring as recovery drags on [13]. Real-time sonoelastography (ASE) operates as a critical preventative tool for clinicians trying to manage this exact daily risk [10]. Reading ASE data actually lets doctors predict major structural collapses, easily spotting hidden lesions and deep tissue softening long before a patient feels any pain [10]. Building these objective imaging scans directly into modern treatment paths lets practitioners constantly adjust rehab routines and deploy timely protection for the fragile opposite limb [10,26].

6.2. Early Functional Rehabilitation and Targeted Neuromuscular Training

Standard rigid casts are rarely used today, as clinicians strongly prefer early functional mobilization and protected weight-bearing [1,26]. Putting patients in a functional brace to start early weight-bearing speeds up initial recovery milestones without inflating the actual rerupture rate [1,27]. Pathological tendon elongation remains a severe clinical problem during this window, given that the healing Achilles lengthens for six months post-surgery regardless of the assigned walking protocol [17]. Therapists have to strictly control physical loading to protect the surgical site, mostly because post-surgical tissue stretching directly ruins later heel-rise function and calf recovery [5]. Eccentric loading and neuromuscular drills take over during the later rehab phases, acting as the most effective tools to rebuild tendon stiffness and athletic power [28]. Athletes need this exact mix of strength and proprioceptive training to finally erase biomechanical deficits and normalize the limb symmetry index [2]. True mechanical relief for the opposite leg only occurs when clinicians prevent post-surgical elongation and restore muscular symmetry, a combination that permanently stops compensatory kinematics [8].

6.3. Psychological Interventions and Kinesiophobia Management

Successful return to sport following severe orthopedic trauma relies heavily on basic psychological readiness and the successful management of reinjury fear [12]. Athletes recovering from an Achilles rupture frequently blame their failure to reach old activity levels on a persistent fear of recurrent damage, subjective feelings of poor tendon function, and severe anxiety over enduring another long rehabilitation [9]. Unresolved kinesiophobia directly causes persistent functional asymmetry, creating obvious deficits in muscle power and single-leg hop metrics that heavily multiply the risk of a second injury during active sports [12]. Standard return-to-play algorithms still largely dismiss these mental factors, focusing instead on purely physical measurements like strength output and hop distances [11,12]. Clinicians looking to manage this fear and block compensatory kinematics must prioritize open communication, carefully explaining the rehab process to set realistic recovery goals for the patient [9]. Tracking mental readiness objectively and clearing athletes for safe, symmetric loading requires the active inclusion of validated psychological metrics like the Tampa Scale of Kinesiophobia (TSK) within standard daily assessments [9,11,12].

7. Discussion

Clinical treatments for an acute Achilles tear understandably prioritize the primary injury site, yet the findings synthesized here confirm a severe, long-term physical toll placed on the contralateral limb [7]. Unresolved calf muscle weakness combined with intense kinesiophobia actively drives patients to develop and rely on completely unnatural motor strategies [9,11,12]. The basic instinct to shield the repaired tendon permanently alters natural running kinematics, transferring massive vertical ground reaction forces and joint moments entirely onto the uninjured side [8].

Modern imaging tools essentially leave no doubt about the physical reality of this chronic mechanical overload. Scanning the uninjured leg with routine ultrasonography (USG) immediately exposes early physical breakdowns, capturing tendon thickening and internal lesions that strongly tie into exertional pain [13]. Real-time sonoelastography (ASE) actively proves that this pain-free tissue fails completely at adapting to the extra stress, showing instead that the tendon swells morphologically and suffers from severe internal softening [10].

Failing to properly rehabilitate the original injury both physically and mentally serves as the root cause for all this healthy tendon decay. Letting the tissue stretch out pathologically during early recovery [5,17] leaves the patient with permanent muscle weakness, effectively destroying any chance of balancing the functional limb symmetry index [2]. Clearing away these awkward compensatory movements requires both intense neuromuscular drills and a massive improvement in psychological readiness; otherwise, the opposite leg remains trapped under brutal mechanical stress and highly vulnerable to a second tear [7,8,12].

8. Limitations and Future Research Directions

Current medical data effectively outlines the reality of contralateral overload following an Achilles tear, yet the available studies still carry major methodological gaps. Massive inconsistencies in research design plague these papers, driven mostly by wildly different rehab protocols, conflicting definitions of clinical recovery, and completely unstandardized return to play (RTP) criteria [24,27]. Tracking progressive physical adaptations over time proves nearly impossible because most clinical and biomechanical trials utilize small sample sizes or strict cross-sectional formats [8,9].

To address these gaps, future research should prioritize high-quality, multicenter randomized controlled trials that utilize standardized rehabilitation protocols and uniform functional

outcome measures [27]. Specifically, there is a critical need for longitudinal cohort studies to continuously track how altered running mechanics and compensatory strategies chronically load the contralateral limb [8]. Finally, actively incorporating routine psychological assessments into standard RTP criteria and developing targeted interventions to treat kinesiophobia are essential future directions to comprehensively protect athletes returning to high-demand sports [11,12].

9. Conclusions

The synthesized evidence establishes that an acute unilateral Achilles tendon rupture initiates a bilateral pathological cascade [7,10]. Alongside persistent neuromuscular deficits, a profound lack of psychological readiness actively drives compensatory biomechanics, and the failure to restore functional limb symmetry forces a chronic mechanical overload onto the healthy contralateral limb [2,8,12]. Objective imaging, specifically routine ultrasonography (USG) and real-time sonoelastography (ASE), definitively proves that this continuous stress leads to silent structural degeneration, including tendon thickening, structural micro-lesions, and pathological tissue softening in the uninjured tendon [10,13]. These imaging-detected alterations directly explain the significantly elevated clinical risk of a contralateral rupture [7,10]. Consequently, contemporary clinical practice and return-to-play algorithms must shift toward a holistic, bilateral treatment paradigm [12,27]. To protect the athlete's long-term musculoskeletal health and safely return them to sport, clinicians can no longer rely solely on physical metrics [11,12]. Instead, they must actively integrate objective psychological screening, restore neuromuscular symmetry, and incorporate routine ultrasonographic and elastographic monitoring of the asymptomatic limb to proactively prevent secondary ruptures [2,10,11,13]. Furthermore, establishing standardized rehabilitation protocols and utilizing long-term longitudinal monitoring will be essential in future clinical research to fully understand and mitigate this contralateral impact [8,27].

All authors have read and agreed with the published version of the manuscript.

Disclosure The authors declare no competing interests.

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Funding Statement: The study did not receive special funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgements: Not applicable.

Conflict of Interest Statement: The authors declare no conflicts of interest.

Declaration of use of artificial intelligent

During the preparation of this work, the authors utilized artificial intelligence (AI) as a supportive tool to enhance the readability, linguistic flow, and overall formatting of the manuscript, including citation structuring. The use of this artificial intelligence was strictly limited to technical and stylistic refinement. All scientific content, including literature selection, critical analysis, interpretation of findings, and final conclusions, was developed independently by the authors. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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