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EDITORIAL

TREATING ACHILLES TENDINOPATHY: CONFRONTING DECADES OF SCIENTIFIC CONFUSION

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Clinicians who treat Achilles tendinopathy encounter three problems: (i) deficit of knowledge, (ii) denial of knowledge, and (iii) misinterpretation of knowledge.

Indeed, there is still an incomplete understanding of the underlying biology, a stubborn habit of pooling two distinct conditions into one, and a publish or perish trinity of authors, reviewers and publishers producing literature that too often mistakes volume for progress.

None of these issues is new, none is unique for extracorporeal shockwave therapy (ESWT) or any other treatment concept currently in use. Together, however, they explain why clinicians are still unable to give patients a confident answer about what actually works.

Deficit of knowledge

The physiopathology of mechanisms driving tendon pain remains, at best, vague[1,2]. Histological hallmarks long assumed to explain chronic painful Achilles tendinopathy, including collagen disorganisation, increased type III collagen, increase of extracellular matrix, and blood vessel ingrowth, correlate poorly with symptoms: these tissue pathologies may also be present in asymptomatic tendons, and patients can recover clinically without any of these features reversing.

Excessive ingrowth of sensory nerves, with tendon-specific upregulation of pain receptors and mast-cell receptors for pain mediators, produces a nerve-mast cell axis of neurogenic inflammation. Of the treatments discussed, only eccentric loading and, as co-therapy, extracorporeal shockwave were credited with the strongest scientific evidence for targeting this peripheral neoinnervation and promoting nerve regeneration[1].

Denial of knowledge

The clear distinction between insertional (IAT) from non-insertional Achilles tendinopathy (NAT) has been proposed from more than three decades[3], and more recently reinforced as clinically meaningful[4]. A consensus nomenclature subsequently formalised these definitions, anchoring the diagnosis of insertional and mid-portion tendinopathy to anatomical location, symptoms, clinical findings and histopathology[5]. Yet, large parts of the clinical trial literature continue to lump the two together.

The tendon continuum model recognized reactive

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tendinopathy, tendon disrepair, and degenerative tendinopathy as distinct stages within a continuum rather than a single disease entity[6]. These stages were likely to differ in their biological characteristics and might not respond similarly to various treatment efforts.

For clinical studies, tendon pathology should be divided into two clear groups: reactive/early tendon disrepair and late tendon disrepair/degenerative tendinopathy. This division of the continuum into two categories would allow to have a nominal threshold beyond which tendons will not fully return to normal structure. This grouping has, to our knowledge, not been applied in clinical trials.

Unfortunately, the expression "extracorporeal shock wave therapy" (ESWT) has come to include both shock wave therapy and treatments using radial pressure waves[7,8]. In this context, the word "wave" refers to a pressure pulse consisting of a positive phase followed by a negative or rarefaction phase. In other words, it includes half a positive cycle and half a negative cycle. When the first ballistic devices entered the market, many authors referred to them as radial shock wave generators. This is incorrect.

Shock waves and radial pressure waves, both mechanical waves with therapeutic applications, behave differently as they travel through tissue. Only shock waves can deliver a significant amount of energy several centimetres deep into the body. Radial pressure waves act almost exclusively at the surface. Using radial pressure waves to treat deep seated conditions is senseless.

To avoid the incorrect use of the acronym ESWT, we use the term "extracorporeal shock wave therapy" (ESWT) when referring to the therapy in general, and "radial pressure wave therapy" (RPWT) for treatments performed exclusively with ballistic devices[7,8].

Nevertheless, it must be crystal clear that the major obstacle to comparing results, designing reproducible protocols, and avoiding errors in both ESWT and RPWT, is the lack of uniformity in device settings. Manufacturers have implemented different adjustment parameters, such as voltage, intensity, energy level, and pressure, which vary from one system to another. These parameters are device-dependent: hence, comparisons are only valid when made between systems of the same brand and model.

Yet, large parts of the clinical trial literature continue to lump ESWT and RPWT together and ignore device-dependent treatment parameters.

Misinterpretation of knowledge

A 2021 network meta-analysis screened 5154 records and retained just 29 randomised trials evaluating the comparative effectiveness of any treatment for Achilles tendinopathy, without separating insertional from midportion disease. Not a single level-of-evidence grade 1 trial addressing surgery was identified[9]. Its headline conclusion that, for midportion Achilles tendon disease, no active treatment shows a clinically relevant advantage over any other at 3 or 12 months, is a genuine finding, not an artefact of poor methodology. But it remains to be analysed critically how a "comparative effectiveness" question could be asked and answered at all without engaging the anatomical distinction that most plausibly determines which patients respond to which intervention: For two decades ago, in non-insertional Achilles tendinopathy (NAT) adding radial pressure wave therapy (RPWT) to eccentric loading had produced an additional benefit over eccentric loading alone[10]. And extending the exactly same treatment parameters (ballistic device of the same brand and model) to insertional Achilles tendinopathy (IAT), RPWT had demonstrated superior results over eccentric loading alone[11].

When literature analysis is restricted to ESWT/RPWT alone, and the insertional/midportion split is respected, a more coherent, if modest, picture emerges. Paantjens and colleagues, in 2022, screened 3558 publications and retained only seven randomised trials meeting rigorous inclusion criteria, one of which was subsequently excluded for high risk of bias arising from randomisation[12]. Of the remaining six, three addressed NAT and three addressed IAT. For NAT, RPWT combined with eccentric loading outperformed a wait-and-see strategy. For IAT, with ESWT- and RPWT- trials lumped together, no superior treatment effect was found.

The conclusions were appropriately guarded: moderate evidence for RPWT added to a tendon-loading programme in NAT, and a lack of supporting evidence for ESWT/RPWT in IAT. That "moderate" rating for NAT rested on only three trials, which is not a solid scientific foundation by any standard.

Pragmatic, registry-level data respecting the same anatomical split point in a more encouraging direction: prospective, multicentre follow-up of the UK ASSERT database found significant improvement in pain, patient-reported function, and several quality-of-life domains over 24 months in both NAT and IAT treated with standardised EPWT/RPWT protocols, although the uncontrolled, level IV design cannot exclude natural history, regression to the mean, or placebo effects[13,14].

Recent subsequent research has offered only qualified support for NAT. It has not strengthened the case for IAT.

In a recent randomised trial, 46 runners with NAT received exercise alone, exercise plus RPWT, or exercise plus RPWT plus photobiomodulation. The combination of exercise and RPWT produced the greatest early reduction in symptoms, but all three arms achieved clinically meaningful improvement, underscoring that exercise remains the active ingredient regardless of what is added to it[15]. For IAT, 76 participants were randomised to RPWT or sham treatment, both alongside exercise, and no between-group difference in the primary outcome was reported at 6 or 12 weeks[16].

A 2026 systematic review and meta-analysis by Korakakis and colleagues, pooling nine RCTs with 557 participants, mixing up ESWT- and RPWT-trials, reported no clinically

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meaningful benefit over sham for either insertional (IAT) or midportion (NAT) disease, concluding that shockwave therapy should not, at present, be considered a routine treatment for either entity[17]. The authors provocatively coined their publication “Shockwave therapy for midportion and insertional Achilles tendinopathy: a nail in the coffin?” Given the acknowledged low- and very-low-certainty ratings assigned to much of the available evidence within the review itself, such categorical wording appears stronger than warranted by the underlying data[18].

Perspectives

Do all these findings mean ESWT/RPWT for Achilles tendinopathy is finished? Are we riding a dead horse? Not necessarily, but the horse is undeniably in danger, as indeed is any other conservative or operative approach to Achilles tendinopathy.

The honest reading of the cumulative evidence is that RPWT, after all, when added to a structured loading programme may retain a modest, disease-specific role in non-insertional (NAT) disease. In insertional (IAT) disease the jury is still out, and level I evidence is called for.

The overall evidence level for ESWT/RPWT for all forms of Achilles tendinopathy remains depressingly low. What is missing is not more small trials repeating the same design flaws that undermine comparability across studies. It is necessary to plan one adequately powered, blinded, multicentre trial that, from the outset, separates insertional from midportion disease[3-5]. distinguishes reactive/early tendon disrepair from late tendon disrepair/degenerative tendinopathy patients[6], uses a credible sham comparator, observes device-dependency with regard to brand and model[7,8] and follows patients for years rather than weeks.

The orthopaedic research community has recently proven that this kind of collaborative ambition is achievable. The ten-year follow-up of the FIDELITY trial, comparing arthroscopic partial meniscectomy with sham surgery across five hospitals, found no benefit, but possible harm, from a procedure performed for decades on the strength of belief rather than evidence[19]. More than 90% of the original cohort remained in follow-up after ten years, showing that patients will commit to long, rigorous science when investigators ask them to.

Achilles tendon tendinopathy research deserves the same ambition. ESWT/RPWT has yet to prove beyond reasonable doubt to be a genuine bridge between mechano-transduction and tendon biology, targeting neurogenic inflammation, alone or in combination with heavy load eccentric or concentric loading. That claim will only be settled by a single, sufficiently large, long-term multicentre trial, designed and delivered by the generation of clinicians currently training the next one.

The evidence gathered so far is dubious regarding the routine use of ESWT or RPWT for insertional Achilles tendinopathy, but advocates its cautious, exercise-combined use in midportion disease, and leaves the definitive answer to a trial that has not yet been run.

That trial should start before we run out of time to run it. Patients and clinicians are waiting.

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