Methodological and Interpretative Limitations in a Case Report of Achilles Tendon Rupture Following Extracorporeal Radial Pressure Wave Therapy

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Abstract

This commentary critically examines a case report attributing Achilles tendon rupture to shock wave therapy. The manuscript presents significant conceptual and methodological flaws, particularly the conflation of extracorporeal shock wave therapy, radial pressure wave therapy, and percussive ultrasound, despite their distinct physical and biological characteristics. The proposed mechanism of cumulative microtrauma lacks supporting evidence and contradicts current mechanotransduction-based models. In addition, insufficient reporting of treatment parameters limits reproducibility. The authors fail to consider well-established risk factors for tendon rupture, relying instead on temporal association, which constitutes a post hoc fallacy. Overall, the conclusions are speculative, unsupported, and not aligned with existing literature.
Keywords: Shock waves, Radial pressure waves, Achilles tendinopathy, Achilles rupture

Introduction

Commented article "A Case Report of Achilles Tendon Distractive Rupture after Shock Wave Therapy" [1], published in the Journal of Surgical Case Reports.

This manuscript describes a single case of Achilles tendon rupture allegedly associated with prior radial pressure wave treatment, despite the title indicating that shock waves were used. Although the topic is, in principle, clinically relevant, the report exhibits significant methodological, conceptual, and interpretative weaknesses that substantially limit its scientific value.

Confusion of Therapeutic Modalities and Their Mechanisms of Action

A major conceptual flaw lies in the confusion between "percussive ultrasound therapy," as described by the authors, with "extracorporeal radial pressure wave therapy (RPWT)" and "extracorporeal shock wave therapy (ESWT)." These approaches differ substantially in their physical characteristics, depth of penetration, and biological effects [2,3,4]. The manuscript refers to these three modalities interchangeably, raising serious concerns about both the accuracy of the intervention administered and the authors' understanding of the treatment. Even some articles cited by the authors reflect similar inconsistencies in terminology [5,6].

It is essential to distinguish shock waves from radial pressure waves (Fig. 1). The peak positive pressure of shock waves used in ESWT may reach up to 80 MPa. Their rise time, defined as the interval required for the pressure to increase from 10% to 90% of its maximum value, typically ranges from 2 to 500 ns. The extremely short rise time and brief duration of the positive pressure phase, together with a peak positive pressure and a tensile (negative-pressure) phase reaching amplitudes of several tens of megapascals, give rise to effects that differ markedly from those produced by ballistic devices, which exhibit peak compressive pressures of up to approximately 30 MPa and much longer rise times (on the order of 3 μs). A clear example of a shock wave-induced phenomenon is acoustic cavitation and the subsequent formation of high-velocity microjets, which may induce mechanical and biological effects. Furthermore, in addition to the well-known difference in penetration depth between shock waves and radial pressure waves, it is worth noting that, unlike shock waves, radial pressure waves also include shear and elastic components that may influence regenerative processes [7].

There is also confusion regarding the mechanism of action of therapeutic mechanical waves. The hypothesis that repetitive ESWT induces cumulative microtrauma leading to rupture is presented without any supporting experimental, biomechanical, or clinical evidence, and remains inconsistent with the current understanding of ESWT's biological effects [8,9].

Rather than causing structural damage at the tissue level, the effects of ESWT are primarily mediated through biological pathways triggered by acoustic stimulation, a process known as mechanotransduction – the conversion of a mechanical stimulus into a cellular response without producing structural damage. Accordingly, as noted by D'Agostino et al.

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[8], the mechanism of action in musculoskeletal applications is not based on direct mechanical effects, such as those observed in urological lithotripsy, but on biologically mediated responses; hence, the traditional "mechanical model" has been replaced by a "biological model" in the treatment of tendons [2,8,9]. This is because mechanotransduction is initiated by the deformation of cells through stretching, compression, and shear forces that arise when shock waves interact with and are reflected at interfaces between tissues with different acoustic impedances [10].

In the context of extracorporeal pressure wave therapy, it should be emphasized that shock waves are not inherently more effective or "superior" to radial pressure waves. They are fundamentally different and, with few exceptions, should be used to treat distinct conditions. The direct transfer of parameters such as pressure, energy density, or frequency from one approach to the other is not valid.

Finally, it is worth noting that in "percussive ultrasound therapy," waves with amplitudes on the order of 0.1–1 MPa and frequencies of approximately 1–3 MHz are applied. These characteristics differ substantially from those of ESWT or RPWT and, therefore, lead to different physical interactions and biological effects.

Insufficient Description of Treatment Parameters

The description of the protocol is incomplete and inadequate for proper interpretation or replication. The authors report that the patient presented to the emergency department with a ruptured Achilles tendon. Although they provide information on the number of sessions and the applied frequency, which clearly do not correspond to recommended treatment protocols [2,11], other essential parameters are not specified. Moreover, the timeline of events is not clearly established. In particular, the interval between the treatments and the tendon rupture is not reported, nor is the time elapsed between the completion of the sessions and the initiation of rehabilitation.

In addition to the importance of correctly using the terms ESWT and Extracorporeal RPWT, any clinical protocol or report should, from a physical perspective, include at least the following information:

- Number of pulses per session
- Pulse repetition frequency
- Number of sessions
- Interval between sessions
- Coupling method and coupling medium

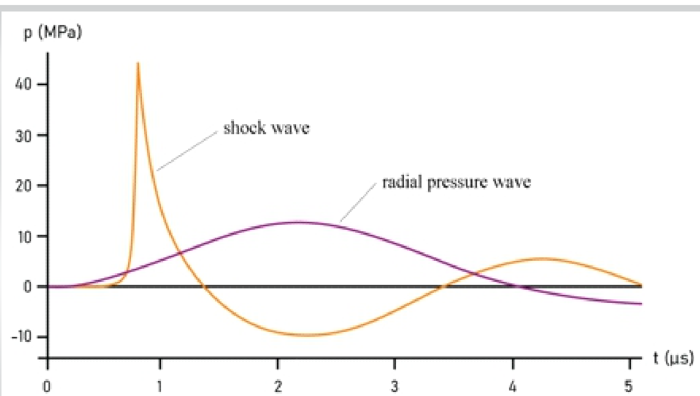

Figure 1: Pressure–time profiles measured at the focus of a shock wave source and in the near field of a ballistic applicator. Adapted from the ISMST Guidelines (2023) (https://shockwavetherapy.org/ismst-guidelines/).

- Model and manufacturer of the equipment used
- "Intensity" level or other device-specific settings
- Peak positive pressure
- Energy flux density
- Total applied energy
- Type of applicator for ballistic devices
- Air pressure for pneumatic devices
- Impulse for ballistic devices.

Without this information, comparisons between treatments should not be made, and previously used protocols cannot be reliably reproduced. When such data are available, additional characteristics of the pressure field may be inferred. This is particularly relevant because no single parameter can be directly linked to a specific biological outcome.

The magnitude of key quantities, such as peak positive and negative pressure, rise time, pulse duration, and energy flux density, depends on factors including the design of the source and the underlying generation principle. These variables are essential for developing treatment protocols, evaluating methodologies, and reporting results. However, clinicians are not expected to determine them experimentally. Instead, they should clearly document the device manufacturer, model, and all technical specifications provided by the manufacturer.

Failure to Provide a Balanced Differential Explanation

The manuscript does not adequately consider alternative explanations for the tendon rupture. The Achilles tendon is the most frequently injured tendon in the human body [12,13,14], and in most cases, patients have not previously undergone treatment with radial pressure waves.

Several relevant factors have not been properly assessed. Based on the authors' description, the patient presented at least three established risk factors for spontaneous rupture [1]: her age [13], the chronicity of her condition [13], and her thyroid disorder [14,15,16,17]. Her body mass index, which may also represent a contributing factor [18], is not reported.

Beyond the numerous factors that contribute to tendinopathy, the most common anatomopathological finding is degenerative change, which may lead to reduced tensile strength and an increased predisposition to rupture [14,19]. The absence of a structured differential analysis suggests a biased interpretation aimed at supporting a pre-conceived conclusion.

Unsupported Causal Inference

The main claim of the manuscript is not supported by the evidence presented. The authors rely solely on temporal association while failing to adequately account for well-established risk factors, including advanced age and pre-existing tendinopathy. This constitutes a clear example of *post hoc ergo propter hoc* reasoning – a logical fallacy that assumes that if one event follows another, the first must have caused the second. However, temporal sequence alone does not establish causality. This conceptual error is frequently used to attribute injuries to pressure wave therapies.

No mechanistic data, comparative analysis, or supporting literature is provided to substantiate the claim of treatment-induced rupture. Consequently, the conclusions are speculative and overstated.

Overstatement of Novelty and Literature Context

The authors claim that their case represents the first report of Achilles tendon rupture following what they describe as shock wave therapy. However, Costa et al. reported two cases of Achilles tendon rupture after treatment with focused shock waves in a study published in 2005 [20].

Conclusion

The central premise of the manuscript – that ESWT contributed to or caused the Achilles tendon rupture – is not supported by the evidence presented. Regardless of the confusion between shock waves and radial pressure waves, the authors rely solely on temporal association while failing to adequately account for well-established risk factors, including advanced age and pre-existing tendinopathy. This constitutes a clear case of *post hoc ergo propter hoc* reasoning.

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