REVIEW ARTICLE

CONTENTS

Current perspectives on the etiology and pathogenesis of tendinopathy - a literature review

Franciszek Grabowski[1], Dominika Ciesielska[2], Ignacy Nieściór[1], Michał Merkisz[3], Aleksandra Bełżek[4], Karolina Turżańska[5], Magdalena Sobiech[6]

[1]INTERDISCIPLINARY SCIENTIFIC GROUP OF SPORTS MEDICINE, DEPARTMENT OF SPORTS MEDICINE, MEDICAL UNIVERSITY OF LUBLIN, LUBLIN, POLAND

[2]INTERDISCIPLINARY SCIENTIFIC GROUP OF SPORTS MEDICINE, DEPARTMENT OF SPORTS MEDICINE, MEDICAL UNIVERSITY OF LUBLIN, LUBLIN, POLAND

[3]MEDICAL UNIVERSITY OF GDANSK, GDANSK, POLAND

[4]REGIONAL HOSPITAL IN POZNAN, POZNAN, POLAND

[5]DEPARTMENT OF REHABILITATION, MEDICAL UNIVERSITY OF LUBLIN, LUBLIN, POLAND

[6]DEPARTMENT OF SPORTS MEDICINE, FACULTY OF HEALTH SCIENCE, MEDICAL UNIVERSITY OF LUBLIN, LUBLIN, POLAND

ABSTRACT

Aim: Tendinopathy is a pain-dysfunction syndrome of tendons resulting from an imbalance between the processes operating within the tissue and the external factors to which it is exposed. Contemporary concepts of pathogenesis indicate the overlap of many mechanisms: from mechanical overload leading to abnormal matrix remodeling, through inflammatory processes, to intratendinous compression causing hypoxia and pathological vascularization. The aim of this study was to review information regarding the current state of knowledge about the pathogenesis and mechanisms driving tendinopathies, based on available scientific research.

Materials and Methods: A comprehensive literature review was conducted, including clinical trials, systematic reviews, and meta-analyses that investigated the mechanisms influencing the pathogenesis of tendinopathy.

Conclusions: The heterogeneity of the clinical and histological presentation confirms the multifactorial nature of tendinopathy and explains the therapeutic challenges and frequent treatment failures. The authors suggest that integrating knowledge regarding mechanotransduction, inflammation, metabolism, and the role of intratendinous pressure may provide a basis for developing more precise and effective treatment strategies for tendinopathy.

KEY WORDS: inflammation, intratissue pressure, mechanical loading, mechanotransduction

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INTRODUCTION

Tendinopathy, defined as a clinical syndrome of pain and tendon dysfunction, is one of the most common musculoskeletal disorders, exerting a significant socio-economic impact on modern society [1]. This problem is particularly acute in sports, where tendinopathies are responsible for up to one in three overload injuries [2]. However, tendinopathy also affects a wide range of the general population. Epidemiological data indicate that although the condition occurs in children and youth [3], its incidence increases with age. Studies predict that approximately 1–2% of adults will experience this lower limb problem during their lifetime [4]. The growing scale of the problem is confirmed by the EU report on the development of occupational diseases in selected musculoskeletal disorders, which shows that the number of cases of tenosynovitis and enthesopathy increased by approximately 10% between 2013 and 2019. In addition to the painful and debilitating impact on patients' daily lives, tendon disorders still constitute an underestimated financial burden [1]. The scale of costs is illustrated by data from individual countries – for example in the Netherlands the annual costs associated with lower limb tendinopathies are counted in millions of euros, making them a significant problem for the healthcare system [5].

Despite significant advances in tendon tissue research in recent decades, our understanding of the mechanisms underlying the development of these pathologies remains limited. Consequently, effective treatment of this

debilitating condition remains a significant challenge for clinicians. This problem likely stems from the fact that current therapeutic approaches do not directly address all aspects of the complex course of the disease. Clinical practice guidelines recommend load management, patient education, manual therapy, and shockwave therapy as the most effective treatments for tendinopathy [6]. Unfortunately, the criteria for selecting exercises, their types, intensity, and volume, have not yet been thoroughly researched. Similarly, manual therapy is known for its nonspecific effects. Furthermore, these methods exhibit antagonistic effects in certain areas. This may explain why treatment fails to produce the intended results in many patients, suggesting the need for further research. A more detailed understanding of the mechanisms and factors moderating the development of tendinopathy seems crucial. The continuum model, developed by Professor Jil Cook, was a milestone in this regard [7]. This model notes that depending on the patient's tendinopathy phenotype, a different treatment approach may be required, targeting the actual source of pain and dysfunction. However, 10 years have passed since the publication of this model. Therefore, the authors conducted a literature review of the current state of scientific knowledge on this topic.

AIM

The aim of the study was to review information regarding the current state of knowledge about the pathogenesis and mechanisms driving tendinopathies, with particular attention paid to the role of inflammation, mechanical loading and intratendinous compression based on available scientific research.

MATERIALS AND METHODS

This work was based on a review of scientific studies from PubMed, Google Scholar, Web of Science and MEDLINE, using combinations of the following keywords: tendinopathy, inflammation, interstitial pressure, mechanical loading and mechanotransduction. The materials were collected from 2004 to 2025, beginning with Scott et al.'s significant knowledge of inflammation and continuing with the most recent available research on the etiology and pathogenesis of tendinopathy, which formed the basis of the work. Studies were selected based on the following inclusion criteria: meta-analyses, systematic reviews, clinical practice guidelines, and narrative review articles. The following were excluded from the search: conference abstracts, case reports, and articles written in languages other than English.

REVIEW AND DISCUSSION

INFLAMMATION

Contemporary literature revises the view that tendinopathy is exclusively degenerative in nature. Researchers point to inflammation as the primary etiological factor, rather than merely a secondary consequence [8], and furthermore, it is recognized that it is not a transient process, but rather reflects a dysregulated, self-perpetuating network that destabilizes tendon homeostasis [8]. The pathogenesis is coordinated by the dynamic interaction of three interrelated cellular compartments: the stroma compartment (including tenocytes and tendon stem and progenitor cells), the immune compartment (resident macrophages and mast cells), and the infiltrating compartment, which consists of recruited immune cells [8]. Recent advances in immunology and pathology research have enabled the identification of macrophages, T cells, and B cells in structures affected by chronic changes, proving that the defensive response is not suppressed at any stage of the disease [9]. Importantly, recent analyses have revealed the presence of a unique subpopulation of immunocompetent cells referred to as "tenophages," which are characterized by the expression of markers specific to tendons and macrophages [8]. Interleukin-1β (IL-1β - a pro-inflammatory cytokine) acts as a "pathological switch", meaning that it reprograms stromal cells and induces a pro-inflammatory activation state in tenocytes [8]. The cytokine IL-1β promotes overexpression of collagenases (MMP-1, MMP-13 - extracellular matrix metalloproteinases) and stromelysins (MMP-3 - extracellular matrix metalloproteinase 3) while simultaneously suppressing the response of TIMP inhibitors - tissue inhibitors of metalloproteinases, leading to the degradation of the extracellular matrix (ECM) and thus driving the progression of tendinopathy [9,10]. In addition, IL-1β alters the fate of tendon stem/progenitor cells (TSPCs) by promoting their degeneration. It inhibits the expression of tenogenic markers (tenomodulin, scleraxis) and type I collagen, promoting cell differentiation towards non-tenogenic lines [8]. At the intracellular level, these processes are driven by the NF-κB signaling pathway - nuclear factor kappa-B, which stimulates the production of further pro-inflammatory cytokines (IL-6 - interleukin 6, TNF-α - tumor necrosis factor alpha), creating a feedback loop that perpetuates inflammation [9]. Neurogenic inflammation is an important element of pathophysiology. Studies confirm the presence of nerve ingrowth markers (PGP 9.5 - protein gene product 9.5) and the involvement of the glutamatergic system (glutamate, NMDAR receptors - N-methyl-D-aspartate receptors, mGLUT

- metabotropic glutamate receptors) and sympathetic systems (NPY - neuropeptide Y, adrenoreceptors) in diseased tendons [11]. Substance P and CGRP (calcitonin gene-related peptide), released from nerve endings, can indirectly stimulate mast cell degranulation and histamine release, which intensifies vasodilation, edema, and angiogenesis [11,12]. Excessive stimulation of nociceptors by these mediators is directly related to the sensation of pain [11]. Other causes that may contribute to the development of tendinopathy include diabetes and hypercholesterolemia [13]. Chronic hyperglycemia induces the accumulation of advanced glycation end-products (AGEs), which destabilize the collagen architecture, reducing tendon elasticity [13]. Obesity also correlates with the development of tendinopathy, due to systemic inflammation and increased mechanical stress [13]. Metabolic danger patterns (DAMPs - signaling molecules released as a result of cell damage), such as LDL cholesterol - low-density lipoprotein, fatty acids, or hyperglycemia, induce the phenomenon of trained immunity - they permanently regulate the activity of the innate immune system [10]. Chronic low-grade inflammation is a risk factor for healing failure [12]. Tenocytes can retain "inflammatory memory" through epigenetic changes, showing hyperresponsiveness to cytokine stimuli even years after the initial injury has resolved [8]. A key therapeutic challenge is restoring the balance in macrophage polarization. In normal healing, proinflammatory (M1) macrophages should give way to a repair phenotype (M2). Disruption of this switch leads to fibrosis and chronic degeneration [12]. The understanding that inflammation, mechanical stress, and vascularization form a self-perpetuating "tendinopathic loop" suggests the need for targeted therapies that precisely modulate, rather than completely suppress, the inflammatory response [10].

MECHANICAL LOAD

The etiopathogenesis of tendinopathy is closely correlated with disruption of mechanical homeostasis within the extracellular matrix (ECM). Contemporary biomechanical models point to the "Goldilocks zone" as the optimal loading window, which is essential for maintaining tissue anabolic properties [14, 15]. Inappropriate mechanical loading – including both chronic overload and mechanical underload – induces tenocytes to enter a catabolic state. This leads to progressive dysregulation of the molecular composition of the matrix and loss of its structural integrity [14, 16]. Mechanosensitivity and mechanotransduction are key processes that integrate external mechanical stimuli with intracellular signaling pathways, determining cellular phenotype and tissue architecture [17]. This is a dynamic interface that allows tenocytes to adaptively remodel the matrix in response to a variable load profile, determining tenogenic differentiation and proliferation [1, 17]. This process depends on the integrity of the actin cytoskeleton and integrin complexes, and PIEZO1 ion channels play a key role in detecting shear stress [18]. Their activation determines a transient influx of calcium ions (Ca2+), modulating the expression of enzymes responsible for collagen structure stabilization [17, 18]. Under pathological conditions, excessive mechanical stimulation induces nonphysiological expression of proinflammatory cytokines (IL-1β), which, through autocrine pathways, increases the activity of matrix metalloproteinases (MMPs) discussed in the previous paragraph. This results in accelerated proteolysis of structural proteins and initiation of the inflammatory cascade [14, 16, 19]. At the ultrastructural level, pathological loading leads to disorganization of the parallel arrangement of type I collagen fibers and their substitution by thinner fibrils with lower mechanical strength [16, 20]. These changes lead to the pathological accumulation of glycosaminoglycans (GAGs), which drastically alters the biomechanical properties of the tissue: fibrosis processes correlate with increased stiffness, while the degenerative phase is characterized by the loss of the ability to effectively transfer loads [16, 21]. ECM stiffening observed in the course of fibrosis (scarring) may disturb the response of cells to physical stimuli, leading to changes in their motility, proliferation, adhesion, and differentiation [17, 18]. An important phenomenon in this context is the stress shielding mechanism, resulting from the uneven distribution of stresses within the damaged structure [21]. As a result of local fiber disorganization, healthy fibrils absorb excessive loads, while adjacent pathological areas are paradoxically excluded from force transmission. This lack of physiological stress in the shielded areas induces tenocyte apoptosis and further matrix degradation, completing a vicious cycle of tissue degeneration [16, 18]. The degradation process is driven by cumulative microtrauma that exceeds the metabolic repair capacity of tenocytes [14]. This process drastically increases with age and metabolic disorders, which lead to loss of matrix elasticity and pathological stiffening. In this context, a critical etiological factor is compressive forces, occurring at specific anatomical points where the tendon changes direction, wrapping around bony structures [1, 14]. It is in these zones that ischemic and mechanical stresses accumulate, which, combined with the limited biomechanics of the stiffened matrix, becomes a trigger for degenerative processes.

INTRATENDINOUS COMPRESSION

Scientific analyses emphasize the role of disturbances in intratissue pressure homeostasis in the pathogenesis of tendinopathy. Classical mechanical models ignored

this factor, focusing primarily on tensile forces. Current evidence is changing the perspective on the mechanisms of pathological changes [2]. Each structure in the body has a specific total tissue pressure (TTP), which is the sum of interstitial fluid pressure (IFP) and the solid stress (SSR) exerted by matrix components such as collagen, cells, and proteoglycans [2]. Under physiological conditions, TTP remains low (below 10 mm Hg); its increase is observed in many pathologies, such as pressure-induced neuropathies, osteoarthritis, and cancer [2]. Pressure imbalance within the tendon is currently considered a significant factor influencing the cellular response of tenocytes and the progression of degenerative processes [2, 22]. Increased intratendinous resting pressure (IRP) in tendinopathy is referred to in the literature as "miniature compartment syndrome." This term describes a phenomenon in which the volumetric expansion of the extracellular matrix encounters mechanical resistance from surrounding low-compliance structures. In the case of a tendon, the limiting "walls" of the compartment are the perimysium (epitenon), which surrounds the entire tendon, and the intratendonium (endotenon/IFM), which surrounds individual fascicles. The resistance offered by these sheaths to the swelling tissue induces internal pressure, analogous to the mechanism observed in compression neuropathies, where fluid accumulates under the impermeable epineurium [2, 22]. The main factor in swelling is the pathological accumulation of hydrophilic glycosaminoglycans (GAGs) and proteoglycans, which increase the so-called solid stress [22]. This was confirmed by ex vivo experimental studies on human Achilles tendons, providing quantitative evidence of this relationship. It was shown that infusion of 2 ml of physiological fluid into the native tendon caused an approximately 2.2-fold increase in resting pressure – from a mean value of 18.87 mmHg to 41.02 mmHg ($p<0.001$) [22]. A similar effect was observed in the case of changes in biochemical composition: injection of 80 mg of glycosaminoglycans (GAGs) resulted in an increase in resting pressure to 40.74 mmHg [22]. The final pressure value is determined by the location [2]. The internal compartment, i.e. the tendon fascicle, is characterized by a much smaller diffusion space than the surrounding interfascicular matrix (IFM) [2]. By analogy with studies on nerves, it has been suggested that even a small increase in the volume of fluid enclosed within the fascicle is associated with a drastic increase in pressure (reaching up to 750 mmHg in nerves), while the same volume in the looser IFM tissue generates a much lower pressure (up to 60 mmHg) [2]. This means that the measured mean pressure across the entire tendon may mask critically high pressure peaks occurring locally within individual fascicles [2]. These disturbances in resting homeostasis become critical under dynamic loading conditions. One of the protective mechanisms of a healthy tendon is its ability to reduce its volume under stretching. As demonstrated by Ahmadzadeh et al. [23], the Poisson's ratio for human tendons often exceeds 0.5, which forces radial fluid exudation from the matrix outward. This phenomenon manifests clinically as an immediate reduction in the tendon's cross-sectional area after exercise [24]. However, in tendinopathy, this mechanism is drastically impaired. The pathological accumulation of water-binding GAGs and PGs reduces the hydraulic permeability of the matrix [2, 25]. As a result, instead of being safely squeezed out, fluid becomes trapped within the intractable tendon bundles. Cyclic loading then leads to rapid increases in intratendinous dynamic pressure (IDP) [2]. It is speculated that increased IDP induces a state of functional ischemia by compressing the intratendinous microcirculation. Consequently, pathological tendons—unlike healthy tendons—do not exhibit physiological thickness reduction after exercise, indicating the perpetuation of an intramatrix "compartment syndrome" [2, 25]. This creates a destructive vicious cycle: any attempt to load the tendon exacerbates the pathology by generating pressures exceeding the physiological adaptive threshold of tenocytes. It should be emphasized that the presented mechanism constitutes a novel research paradigm. The authors emphasize the conceptual nature of these considerations [2], pointing out that the role of fluid dynamics and intratendinous pressure is still in the process of being discovered, which may open new avenues for therapeutic strategies to improve tendon healing.

CONCLUSIONS

A review of the scientific literature indicates the direction of modern tendinopathy treatment. Research by renowned medical experts demonstrates that the inflammatory process is not merely a consequence of injury but serves as the primary etiological factor in a self-perpetuating cascade. A key element of this dynamic is ECM degeneration and the inhibition of tendon repair processes. However, inflammation remains closely correlated with impaired mechanotransduction. Therefore, optimal mechanical loading is essential for maintaining anabolic tissue homeostasis. Both excessive and insufficient loading trigger a vicious cycle of degeneration, leading to pathological stiffening of the ECM. Such a tendon is characterized by an impaired physiological response to physical stimuli. Systemic factors such as obesity and diabetes can further fuel these

processes. This highlights the importance of patient education regarding changes in habits, lifestyle, and diet. The concept of the role of intratendinous pressure and compression, while very interesting, seems insufficiently researched to be currently used in treating tendinopathies. However, the authors do not rule out a change in this stance in the future, as this model helps explain many unknowns in the pathogenesis of tendinopathies. Clinicians aware of the mentioned mechanisms should focus their treatment on interrupting the "tendinopathic loop" through precise load management and, when necessary, suppressing active inflammation. The review authors suggest that rehabilitation teams should focus on kinesiotherapy, which utilizes mechanotransduction processes to stimulate matrix remodeling. This is crucial for a very large group of patients, such as athletes. For them, the treatment outcome should be not only improved pain but, above all, rapid return of highly specialized function, which determines success in their field. Therefore, further research on tendinopathies is needed to precisely manage treatment methods and effectively prevent them.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

CORRESPONDING AUTHOR

Franciszek Grabowski
Interdisciplinary Scientific Group of Sports Medicine,
Department of Sports Medicine
Medical University of Lublin,
Lublin, Poland
e-mail: franc.grabb@gmail.com

ORCID AND CONTRIBUTIONSHIP

Franciszek Grabowski: 0009-0000-9363-4085 A B D
Dominika Ciesielska: 0009-0007-8548-1112 B D
Ignacy Nieściór: 0009-0009-8886-5706 B D
Michał Merkisz: 0009-0007-0198-2244 B D
Aleksandra Bełżek: 0000-0001-5543-877X B D
Karolina Turżańska: 0000-0001-7359-9622 E F
Magdalena Sobiech: 0000-0003-4923-5444 E F

A – Work concept and design, B – Data collection and analysis, C – Responsibility for statistical analysis, D – Writing the article, E – Critical review, F – Final approval of the article

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