

Tendinopathy: Current Concepts in Pathophysiology, Clinical Presentation, and Management – An Updated Review

Dr. Saurabh Saklani^{1*}, Ruchita Sharma², Garima Kapoor¹, Jyotsna Bharti¹, Mansi Thakur¹, Surender Kumar³, Milind Thakur¹

¹School of Pharmacy and Emerging Sciences (SPES), Baddi University of Emerging Sciences and Technology, Mahnumajra, Baddi, District Solan, Himachal Pradesh - 173205, India

²GRY Institute of Pharmacy Borawan, Madhya Pradesh-451001, India

³Department of Pharmaceutical Sciences (Pharmaceutics), Lord Shiva College of Pharmacy, Sirsa, Haryana, India-125055

Corresponding Author:

Email ID : dsaklani@baddiuniv.ac.in

ABSTRACT

Tendinopathy is a common musculoskeletal disorder characterized by tendon pain, impaired function, and reduced load-bearing capacity. It affects both athletic and non-athletic populations and represents a significant cause of disability and healthcare burden worldwide. Traditionally regarded as an inflammatory condition, current evidence suggests that tendinopathy is a multifactorial disorder involving mechanical overload, extracellular matrix degradation, collagen disorganization, cellular dysfunction, oxidative stress, neurovascular ingrowth, and a failed healing response. Commonly affected tendons include the Achilles tendon, patellar tendon, rotator cuff tendons, and lateral elbow extensor tendons. Several intrinsic and extrinsic risk factors, including aging, obesity, diabetes mellitus, repetitive occupational activities, and sports-related overuse, contribute to disease development and progression. Advances in imaging modalities such as ultrasonography and magnetic resonance imaging have improved the diagnosis and monitoring of tendon pathology. Management strategies include exercise-based rehabilitation, physiotherapy, extracorporeal shockwave therapy, pharmacological interventions, platelet-rich plasma therapy, regenerative medicine approaches, and surgical treatment in refractory cases. Emerging therapies such as stem cell therapy, growth factor delivery, gene therapy, and tissue-engineered scaffolds offer promising future directions for tendon regeneration. This review provides a comprehensive overview of the epidemiology, pathophysiology, clinical manifestations, diagnosis, and current as well as emerging treatment strategies for tendinopathy, highlighting recent advances and future perspectives in tendon research and clinical practice.....

Keywords: Tendinopathy; Tendon Degeneration; Tendinosis; Achilles Tendinopathy; Patellar Tendinopathy; Rotator Cuff Tendinopathy; Tendon Healing; Extracellular Matrix; Platelet-Rich Plasma (PRP); Extracorporeal Shockwave Therapy (ESWT); Regenerative Medicine; Stem Cell Therapy; Rehabilitation; Sports Medicine; Musculoskeletal Disorders..

How to cite this article: . Dr. Saurabh Saklani, Ruchita Sharma, Garima Kapoor, Jyotsna Bharti, Mansi Thakur, Surender Kumar, Milind Thakur., Tendinopathy: Current Concepts in Pathophysiology, Clinical Presentation, and Management – An Updated Review Int J Drug Deliv Technol. 2026;16(86s):697-706. DOI: 10.25258/ijddt.16.86s.75

Source of support: Nil.

Conflict of interest: Nil.

INTRODUCTION

Tendinopathy, historically referred to as *tendinitis* or *tendonitis*, is a common musculoskeletal disorder characterized by tendon pain, swelling, impaired function, and reduced load-bearing capacity. While the term *tendinitis* implies an inflammatory condition, contemporary evidence suggests that most chronic tendon disorders are predominantly degenerative in nature and are therefore more accurately described as *tendinopathy*. The condition affects both athletic and non-athletic populations and represents a major cause of disability, reduced physical performance, and diminished quality of life¹.

Tendinopathy is one of the most common causes of sports- and work-related musculoskeletal injuries worldwide. Recent epidemiological studies indicate that tendon disorders

account for approximately 30–50% of all sports medicine consultations and contribute significantly to healthcare expenditures and work absenteeism². Globally, musculoskeletal disorders affect nearly 1.71 billion people, with tendon-related pathologies representing a substantial component of this burden^{3,51}. The most frequently affected tendons include the Achilles tendon, patellar tendon, rotator cuff tendons, and lateral elbow extensor tendons. Rotator cuff tendinopathy affects approximately 20–30% of adults⁵⁰, with prevalence increasing to more than 50% among individuals over 60 years of age. Achilles tendinopathy is reported in 6–18% of runners, while patellar tendinopathy affects nearly 14–22% of elite jumping athletes^{4,5,48,49}.

Traditionally, tendon pain was believed to result primarily from inflammation. However, advances in

*Author for Correspondence: dsaklani@baddiuniv.ac.in

histopathological, molecular, and imaging studies have fundamentally changed the understanding of tendon pathology. Current evidence demonstrates that tendinopathy is characterized by collagen disorganization, extracellular matrix degradation, increased tenocyte proliferation, neovascularization, apoptosis, and impaired tendon healing responses rather than classical inflammation alone. Recent research has identified the involvement of inflammatory mediators such as interleukin-1 β (IL-1 β), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF- α), matrix metalloproteinases (MMPs), and oxidative stress pathways, highlighting the complex interaction between degenerative and inflammatory mechanisms in disease progression^{6,7}.

The incidence of tendinopathy continues to rise due to increasing participation in recreational sports, aging populations, obesity, diabetes mellitus, and repetitive occupational activities. Emerging evidence suggests that individuals with obesity have a 1.5–2.5-fold higher risk of developing lower-limb tendinopathies compared to normal-weight individuals. Similarly, metabolic disorders such as diabetes mellitus have been associated with altered collagen cross-linking, decreased tendon elasticity, and delayed tendon healing, emphasizing the systemic nature of tendon pathology^{8,9}.

Recent technological advances have improved the diagnosis and management of tendon disorders. High-resolution ultrasonography, power Doppler imaging, ultrasound elastography, and magnetic resonance imaging (MRI) have enhanced the detection and monitoring of tendon abnormalities. Furthermore, novel therapeutic approaches including eccentric exercise programs, platelet-rich plasma (PRP), extracorporeal shockwave therapy (ESWT), mesenchymal stem cell therapy, growth-factor-based treatments, and gene-mediated regenerative strategies are being actively investigated to improve tendon healing and functional recovery^{10,11,53}.

Despite significant advances in understanding tendon biology, the precise pathophysiological mechanisms underlying tendinopathy remain incompletely understood, and no universally accepted gold-standard treatment exists. Therefore,

this review aims to provide a comprehensive and updated overview of the epidemiology, risk factors, pathogenesis, clinical manifestations, diagnostic approaches, and current as well as emerging therapeutic strategies for tendinopathy, highlighting recent scientific developments and future research directions.

Epidemiology of tendinopathy

Tendinopathy is a common musculoskeletal disorder accounting for approximately 30–50% of all sports-related injuries and consultations. Although traditionally associated with athletes, its prevalence is increasing among non-athletic populations due to aging, obesity, diabetes, and repetitive occupational activities¹². Achilles tendinopathy affects 2–5% of the general population and 6–18% of runners, while rotator cuff tendinopathy affects nearly 20–30% of adults, increasing to over 50% in individuals older than 60 years¹³. Patellar tendinopathy is reported in approximately 14–22% of elite athletes, particularly those involved in jumping sports. Recent studies have also identified obesity as a significant risk factor, increasing the risk of lower-limb tendinopathies by 1.5–2.5 times. These findings highlight the growing global burden of tendinopathy and the need for effective prevention and management strategies^{14,51,52}.

Signs and Symptoms of Tendinopathy

Tendinopathy is a chronic tendon disorder characterized by tendon degeneration, thickening, and impaired tendon structure, leading to pain and reduced functional capacity. Clinical manifestations vary depending on the affected tendon and severity of the condition. Common signs and symptoms include^{15,16}:

- Localized pain and tenderness over the affected tendon
- Burning sensation and swelling around the tendon
- Pain that worsens during or after physical activity
- Morning stiffness or stiffness after periods of inactivity
- Restricted joint movement and reduced flexibility
- Decreased muscle strength and functional performance
- Tendon thickening or palpable nodules in chronic cases
- Persistent pain lasting for weeks or months, affecting daily activities and sports performance

Table 1. Common Clinical Features of Tendinopathy

Clinical feature	Description
Localized pain and tenderness	Pain or tenderness over the affected tendon
Burning and swelling	Burning sensation and swelling around the tendon
Activity-related pain	Pain worsens during or after physical activity
Stiffness	Morning stiffness or stiffness after inactivity
Restricted movement	Reduced joint movement and flexibility
Reduced strength	Decreased muscle strength and functional performance
Structural changes	Tendon thickening or palpable nodules in chronic cases
Persistent symptoms	Pain may persist for weeks or months and affect daily activities

Causes and Risk Factors of Tendinopathy

Tendinopathy is a multifactorial condition resulting from the interaction of mechanical, biological, and systemic

factors. The most common cause is repetitive overloading of the tendon, leading to microtrauma and a failed healing

response. It may also occur following acute injuries such as falls, direct trauma, or sports-related accidents^{17,18}.

Major risk factors include:

Repetitive movements and overuse during sports or occupational activities

Sudden increase in training intensity, frequency, or duration

Poor biomechanics and inadequate conditioning

Physical trauma or tendon injury

Increasing age and age-related tendon degeneration

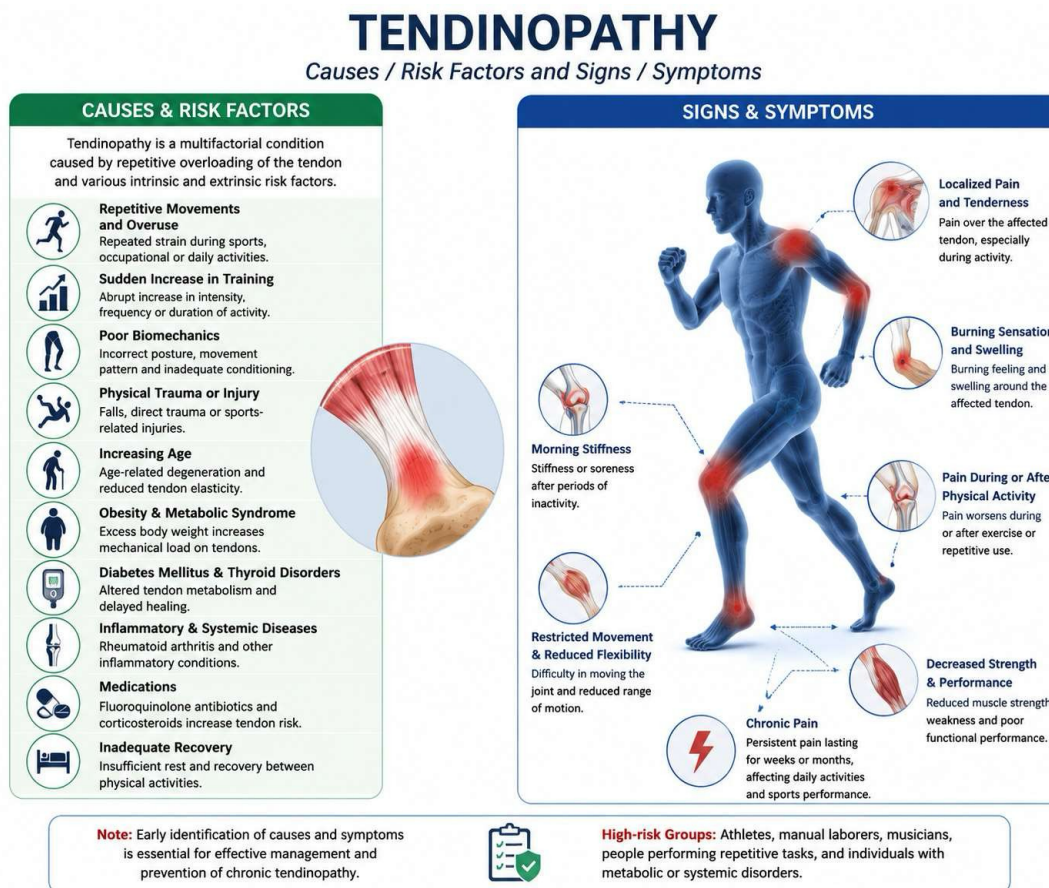
Obesity and metabolic syndrome

Diabetes mellitus and thyroid disorders

Rheumatoid arthritis and other inflammatory joint diseases

Certain medications, particularly fluoroquinolone antibiotics and corticosteroids

Inadequate recovery time between physical activities



Athletes, manual laborers, musicians, and individuals performing repetitive tasks are particularly susceptible to developing tendinopathy. Recent evidence also highlights the role of systemic metabolic disorders in increasing tendon vulnerability and delaying healing.

Table 2. Major Risk Factors Associated with Tendinopathy

Category	Risk Factors	Possible Contribution
Mechanical	Repetitive movements, overuse, sudden increase in training load	Tendon overload and microtrauma
Biomechanical	Poor biomechanics, inadequate conditioning	Abnormal tendon loading
Age-related	Increasing age	Tendon degeneration and reduced healing capacity
Metabolic	Obesity, metabolic syndrome	Increased tendon vulnerability
Endocrine	Diabetes mellitus, thyroid disorders	Altered collagen metabolism and delayed healing
Inflammatory	Rheumatoid arthritis and other inflammatory joint diseases	Altered tendon biology

Traumatic	Falls, direct trauma, sports injuries	Acute tendon damage
Medication-related	Fluoroquinolone antibiotics, corticosteroids	Increased tendon injury risk
Recovery-related	Inadequate recovery between activities	Persistent mechanical stress

Pathophysiology of tendinopathy

As of 2018, the pathophysiology of tendinopathy remained incompletely understood, with several competing models attempting to explain the relationship between tendon structural changes, inflammation, and pain generation. The pathophysiology of tendinopathy is complex and multifactorial, involving mechanical overloading, cellular dysfunction, extracellular matrix (ECM) degradation, inflammation, and a failed healing response. Traditionally, tendinopathy was considered an inflammatory condition (*tendinitis*); however, current evidence suggests that chronic tendinopathy is primarily characterized by tendon degeneration with a limited and dysregulated inflammatory component¹⁹.

The most widely accepted mechanism underlying tendinopathy is repetitive mechanical overloading of the tendon, which exceeds its physiological capacity for repair. Excessive tensile, compressive, or shear forces result in micro-trauma within the tendon matrix, leading to disruption of collagen fibers and activation of resident tendon cells (tenocytes). In response to injury, tenocytes release inflammatory mediators such as interleukin-1 β (IL-1 β), interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), and prostaglandin E2 (PGE2), which contribute to extracellular matrix degradation and impaired tendon healing^{20,21}.

At the molecular level, tendinopathy is associated with increased activity of matrix metalloproteinases (MMPs) and reduced synthesis of type I collagen, the primary structural protein of healthy tendons. Simultaneously, there is an increased production of type III collagen, which is mechanically weaker and less organized. This imbalance results in collagen disorganization, reduced tensile strength, and progressive tendon degeneration. Histopathological examination typically reveals collagen fiber separation, increased ground substance, tenocyte proliferation, and disruption of normal tendon architecture^{22,23}.

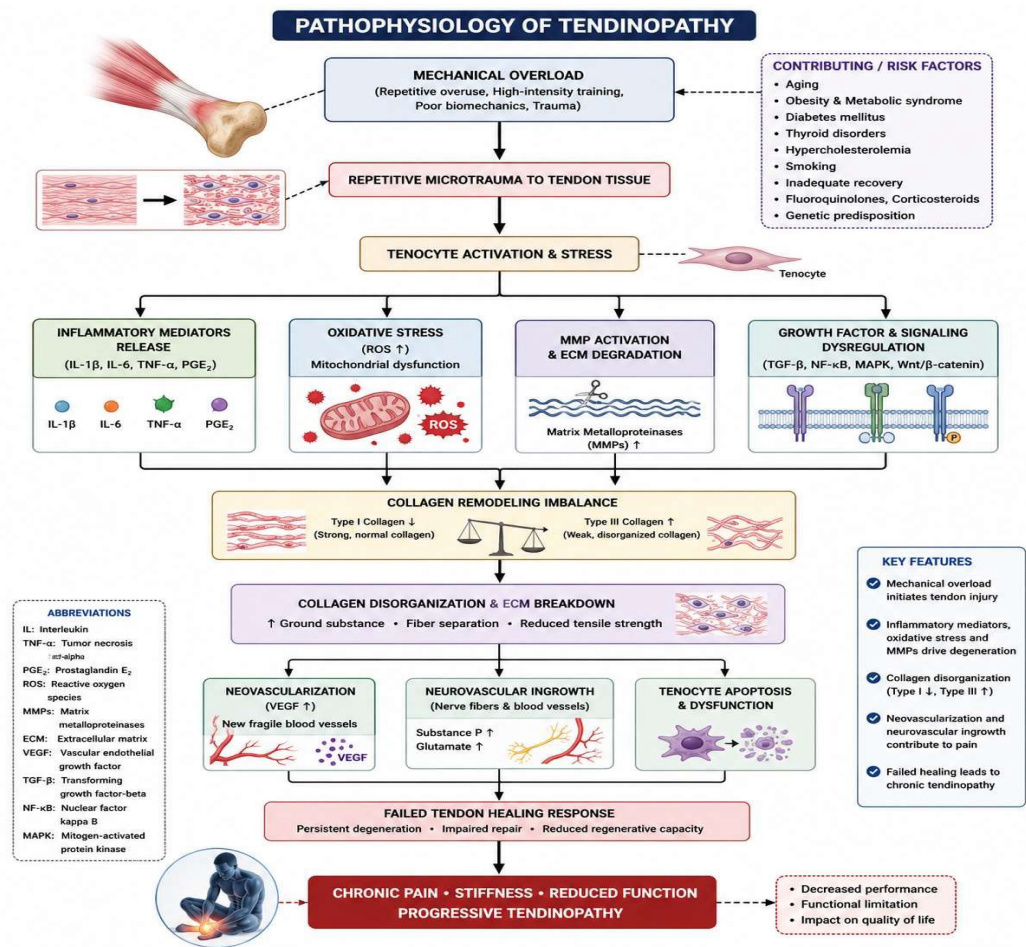
Recent studies have identified oxidative stress and mitochondrial dysfunction as important contributors to tendon

pathology. Excessive generation of reactive oxygen species (ROS) can induce tenocyte apoptosis, impair collagen synthesis, and accelerate matrix degradation. Furthermore, activation of intracellular signaling pathways such as NF- κ B, TGF- β , MAPK, and Wnt/ β -catenin pathways plays a crucial role in regulating inflammation, tissue remodeling, and tendon degeneration^{24,25}.

A characteristic feature of chronic tendinopathy is neovascularization and neurovascular ingrowth. New blood vessels and sensory nerve fibers infiltrate the degenerative tendon tissue, often mediated by increased expression of vascular endothelial growth factor (VEGF). These neurovascular changes are believed to contribute significantly to chronic pain and tendon dysfunction. Increased concentrations of neurotransmitters such as glutamate and substance P have also been identified in tendinopathic tendons, further supporting the role of neural mechanisms in pain generation^{26,27}.

Metabolic and systemic factors also influence tendon pathology. Obesity, diabetes mellitus, hypercholesterolemia, thyroid disorders, and aging can alter tendon metabolism and reduce its regenerative capacity. Advanced glycation end products (AGEs), particularly in diabetic patients, accumulate within tendon collagen, increasing stiffness and susceptibility to injury²⁸.

According to the Continuum Model of Tendon Pathology, tendinopathy progresses through three stages: reactive tendinopathy, tendon disrepair, and degenerative tendinopathy. Reactive tendinopathy represents a short-term adaptive response to acute overload. Continued excessive loading leads to tendon disrepair, characterized by matrix breakdown and cellular changes. If the pathological stimulus persists, degenerative tendinopathy develops, involving extensive collagen disruption, cell death, neovascularization, and a markedly reduced healing capacity²⁹.

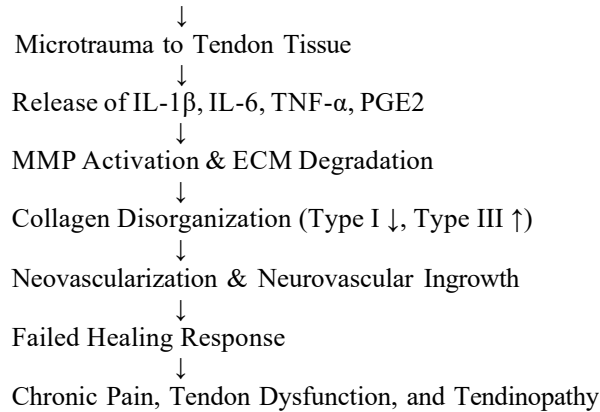


Overall, tendinopathy is now recognized as a multifactorial disorder involving the interaction of mechanical overload, inflammation, matrix degradation, oxidative stress, neurovascular changes, and failed tissue repair. Understanding these mechanisms is essential for developing effective therapeutic and regenerative treatment strategies.

Table 3. Major Pathophysiological Mechanisms in Tendinopathy

Mechanism	Key changes	Potential consequence
Mechanical overload	Excessive tensile, compressive, or shear forces	Microtrauma and activation of tenocytes
Inflammatory signaling	IL-1β, IL-6, TNF-α, PGE2	Matrix degradation and impaired healing
ECM remodeling	Increased MMP activity and extra-cellular matrix degradation	Loss of normal tendon architecture
Collagen alteration	Reduced type I collagen and increased type III collagen	Reduced tensile strength and disorganization
Oxidative stress	Increased ROS and mitochondrial dysfunction	Tenocyte apoptosis and impaired collagen synthesis
Neurovascular changes	Neovascularization and sensory nerve ingrowth	Contribution to chronic pain and dysfunction
Systemic/metabolic effects	Obesity, diabetes, hypercholesterolemia, thyroid disorders, aging	Reduced regenerative capacity
Failed healing	Persistent pathological stimulus and inadequate repair	Progression to chronic/degenerative tendinopathy

Figure Concept
Mechanical Overload / Risk Factors



Treatment of tendinopathy

The primary goals of tendinopathy management are to reduce pain, restore tendon function, promote tissue healing, and prevent recurrence. Treatment approaches can be broadly categorized into non-pharmacological, pharmacological, interventional, and surgical modalities. The choice of treatment depends on the severity, duration, and location of the tendon pathology.

Table 4. Current Treatment Modalities for Tendinopathy

Treatment modality	Role/approach	Key consideration
Activity modification	Reduce or modify activities that overload the tendon	Complete immobilization is generally discouraged
Exercise therapy	Eccentric loading and heavy slow resistance	Cornerstone of rehabilitation; improves pain and function
Stretching/flexibility	Improve flexibility, joint mobility, and muscle balance	Useful for stiffness and mobility
Physiotherapy	Exercise, manual therapy, soft-tissue techniques, biomechanical correction	Individualized rehabilitation approach
Bracing/taping/orthotics	Reduce tendon loading and support biomechanics	Generally provides temporary symptom relief
ESWT	Acoustic pressure waves applied to affected tendon	Used particularly in chronic tendinopathies
NSAIDs/paracetamol	Symptomatic pain management	Long-term benefit in chronic tendinopathy is limited
Corticosteroid injection	Short-term pain and swelling reduction	Repeated use may weaken tendon and increase rupture risk
PRP	Autologous platelet concentrate to support tissue responses	Promising but outcomes vary
Prolotherapy	Hypertonic dextrose or irritant solution to stimulate healing	Interventional treatment
Surgery	Debridement, repair, reconstruction, or removal of calcific deposits	Generally reserved for refractory cases

Non-Pharmacological Management

Non-pharmacological interventions are considered the first-line treatment for most patients with tendinopathy.

Activity Modification and Relative Rest:

Patients are advised to reduce or modify activities that overload the affected tendon. Complete immobilization is generally discouraged because controlled mechanical loading is essential for tendon healing. Gradual return to activity is recommended as symptoms improve³⁰.

Exercise Therapy:

Exercise-based rehabilitation remains the cornerstone of tendinopathy treatment. Eccentric loading exercises and heavy slow resistance training have demonstrated significant benefits in reducing pain, improving tendon structure, and restoring functional capacity. These exercises stimulate collagen synthesis and enhance tendon remodeling³¹.

Stretching and Flexibility Training:

Regular stretching exercises improve tendon flexibility, joint mobility, and muscle balance while reducing stiffness associated with chronic tendinopathy³².

Physiotherapy and Rehabilitation:

Comprehensive physiotherapy programs may include therapeutic exercises, manual therapy, soft tissue mobilization, biomechanical correction, postural training, and progressive tendon loading protocols³³.

Massage Therapy:

Massage techniques such as deep transverse friction massage and soft tissue mobilization are frequently used as adjunctive treatments. These methods may enhance local blood circulation, reduce muscle tension, improve tissue flexibility, and provide symptomatic pain relief when combined with exercise therapy³⁴.

Bracing, Taping, and Orthotic Devices:

The use of braces, straps, kinesiology tape, and orthotic supports can help reduce tendon loading, improve biomechanics, and provide temporary symptom relief during rehabilitation³⁵.

Extracorporeal Shockwave Therapy (ESWT):

ESWT involves the application of acoustic pressure waves to the affected tendon. It promotes tissue regeneration, increases local blood flow, stimulates collagen production, and has shown favorable outcomes in chronic Achilles, patellar, and plantar tendinopathies³⁶.

Nutritional Support

Emerging evidence suggests that vitamin C, collagen peptide supplements, and curcumin may support tendon healing by enhancing collagen synthesis and reducing oxidative stress³⁷.

Pharmacological Management

Pharmacological therapies are primarily aimed at pain control and symptom management.

Paracetamol (Acetaminophen):

Paracetamol is commonly used for mild to moderate pain relief at a typical adult dose of **500–1000 mg every 6–8 hours**, with a maximum daily dose of 4 g³⁸.

Non-Steroidal Anti-Inflammatory Drugs (NSAIDs):

NSAIDs such as **Ibuprofen (400–600 mg every 6–8 hours)**, **Naproxen (250–500 mg twice daily)**, and **Diclofenac (50 mg two to three times daily)** are frequently prescribed for short-term pain relief. However, their long-term effectiveness in chronic tendinopathy is limited because tendon degeneration rather than inflammation predominates in most cases³⁹.

Topical NSAIDs:

Topical diclofenac gel may provide localized pain relief with fewer systemic adverse effects and is commonly used in superficial tendon disorders⁴⁰.

Interventional Therapies

Corticosteroid Injections:

Local corticosteroid injections, such as **Triamcinolone Acetonide (10–40 mg)** or **Methylprednisolone Acetate (20–40 mg)**, may provide rapid short-term pain relief and reduction in swelling. However, repeated injections can impair collagen synthesis, weaken tendon tissue, and increase the risk of tendon rupture. Therefore, their use should be limited and carefully monitored⁴¹.

Platelet-Rich Plasma (PRP) Therapy:

PRP therapy involves injecting concentrated autologous platelets into the affected tendon. Platelet-derived growth factors stimulate cellular proliferation, collagen production, angiogenesis, and tissue regeneration. PRP has shown promising results in chronic and treatment-resistant tendinopathies⁴².

Prolotherapy:

Prolotherapy involves the injection of hypertonic dextrose or other irritant solutions to stimulate the body's natural healing response and promote tendon repair⁴³.

Biological and Regenerative Therapies:

Recent advances include mesenchymal stem cell therapy, growth factor administration, gene-based therapies, and tissue-engineered scaffolds. These approaches aim to restore tendon structure and function and are currently under active clinical investigation⁴⁴.

SURGICAL MANAGEMENT

Surgical intervention is generally reserved for patients who fail to respond to conservative treatment after six to twelve months. Surgical procedures may include debridement of degenerative tendon tissue, removal of calcific deposits, tendon repair, tendon reconstruction, and minimally invasive tendon surgery. The primary objectives of surgery are to relieve pain, improve function, and facilitate tendon healing⁴⁵.

FUTURE PERSPECTIVES

Emerging regenerative approaches such as stem cell therapy, growth factor-based treatments, gene therapy, and biologically engineered scaffolds hold considerable promise for enhancing tendon regeneration and improving long-term clinical outcomes. Further high-quality clinical studies are required to establish their safety, efficacy, and optimal therapeutic protocols^{46,47}.

Notable Athlete Examples of Tendinopathy^{49,51,53}

Tendinopathy is frequently encountered among elite athletes due to repetitive loading, overuse, and high-intensity training. Several well-known athletes have experienced tendon disorders during their careers, highlighting the clinical significance of tendinopathy in sports medicine.

Rafael Nadal (Tennis):

Rafael Nadal has been widely reported to suffer from chronic **patellar tendinopathy (jumper's knee)**. The repetitive jumping, sprinting, and abrupt directional changes associated with professional tennis place considerable stress on the patellar tendon. His case demonstrates how chronic tendon overload can affect athletic performance and require long-term rehabilitation strategies.

David Beckham (Football):

David Beckham experienced significant **Achilles tendon-related problems** during his professional career. Achilles tendinopathy is common among football players because of repetitive running, acceleration, and kicking activities that place excessive strain on the Achilles tendon.

Kobe Bryant (Basketball):

Kobe Bryant suffered from chronic Achilles tendon degeneration before sustaining a complete Achilles tendon rupture in 2013. His case highlights the potential progression of untreated or persistent tendinopathy to tendon rupture, particularly in aging athletes exposed to repetitive loading.

Usain Bolt (Track and Field):

Usain Bolt experienced recurrent Achilles tendon issues throughout his sprinting career. High-speed sprinting generates substantial forces across the Achilles tendon, making sprinters particularly susceptible to tendon overload and degeneration.

Maria Sharapova (Tennis):

Maria Sharapova faced recurrent shoulder problems associated with **rotator cuff tendinopathy**. Repetitive overhead serving and powerful strokes can contribute to tendon degeneration and pain in professional tennis players.

Tiger Woods (Golf):

Tiger Woods has experienced multiple musculoskeletal injuries, including tendon-related conditions affecting performance. Repetitive rotational movements and prolonged training can contribute to chronic tendon stress and overuse injuries.

Cristiano Ronaldo (Football):

Despite maintaining exceptional physical conditioning, Cristiano Ronaldo has occasionally managed patellar and tendon-related overuse issues during his career. His case emphasizes the importance of preventive rehabilitation, recovery strategies, and load management in elite sports.

CONCLUSION:

Tendinopathy is a common and multifactorial musculoskeletal disorder characterized by pain, impaired function, and progressive tendon degeneration. Although traditionally considered an inflammatory condition, current evidence suggests that tendinopathy results from a complex interaction of mechanical overloading, cellular dysfunction, extracellular matrix degradation, oxidative stress, neurovascular ingrowth, and a failed healing response. The condition affects both athletic and non-athletic populations and represents a significant cause of disability, reduced physical performance, and healthcare burden worldwide.

Recent advances in molecular biology, imaging techniques, and tendon research have considerably improved the understanding of tendon pathology. Emerging evidence highlights the important roles of inflammatory cytokines, matrix metalloproteinases, growth factors, and mechanobiological pathways in the development and progression of tendinopathy. Furthermore, systemic factors such as aging, obesity, diabetes mellitus, and metabolic disorders have been increasingly recognized as important contributors to tendon degeneration.

Management of tendinopathy requires a comprehensive and individualized approach. Exercise-based rehabilitation, particularly eccentric loading and heavy slow resistance training, remains the cornerstone of treatment. Additional modalities including physiotherapy, extracorporeal shock-wave therapy (ESWT), platelet-rich plasma (PRP), and other regenerative therapies have shown promising outcomes in selected patients. Pharmacological interventions mainly provide symptomatic relief, whereas surgical treatment is reserved for chronic and refractory cases that fail conservative management.

Despite significant progress, no universally accepted gold-standard treatment exists, and treatment outcomes continue to vary among individuals. Future research should focus on identifying precise molecular targets, optimizing regenerative therapies, and developing personalized treatment strategies. Advances in stem cell therapy, gene therapy, tissue engineering, and biologic interventions may revolutionize the management of tendinopathy and improve long-term tendon healing and functional recovery.

In conclusion, a deeper understanding of tendon biology and the integration of evidence-based therapeutic approaches are essential for improving patient outcomes and reducing the growing global burden of tendinopathy..

REFERENCE

1. Abate M, Silbernagel KG, Siljeholm C, Di Iorio A, De Amicis D, Salini V, Werner S, Paganelli R. Pathogenesis of tendinopathies: inflammation or degeneration? *Arthritis Research and Therapy*. 2009;11(3):235.
2. Alfredson H, Lorentzon R. Chronic tendon pain: no signs of chemical inflammation but high concentrations of the neurotransmitter glutamate: implications for treatment. *Current Drug Targets*. 2002;3(1):43-54.
3. Alfredson H, Ohberg L, Forsgren S. Is vasculo-

- neural ingrowth the cause of pain in chronic Achilles tendinosis? *Knee Surgery, Sports Traumatology, Arthroscopy*. 2003;11(5):334-338.
4. Alfredson H, Ohberg L. Neovascularisation in chronic painful patellar tendinosis. *Knee Surgery, Sports Traumatology, Arthroscopy*. 2005;13(2):74-80.
5. Alfredson H. The chronic painful Achilles and patellar tendon. *Scandinavian Journal of Medicine and Science in Sports*. 2005;15(4):252-259.
6. Archambault JM, Jelinsky SA, Lake SP, Hill AA, Glaser DL, Soslowsky LJ. Rat supraspinatus tendon expresses cartilage markers with overuse. *Journal of Orthopaedic Research*. 2007;25(5):617-624.
7. Arnoczky SP, Lavagnino M, Egerbacher M. The mechanobiological etiopathogenesis of tendinopathy. *International Journal of Experimental Pathology*. 2007;88(4):217-226.
8. Astrom M, Rausing A. Chronic Achilles tendinopathy: a survey of surgical and histopathologic findings. *Clinical Orthopaedics and Related Research*. 1995;316:151-164.
9. Cook JL, Feller JA, Bonar SF, Khan KM. Abnormal tenocyte morphology in asymptomatic athletes. *Journal of Orthopaedic Research*. 2004;22(2):334-338.
10. Cook JL, Rio E, Purdam CR, Docking SI. Revisiting the continuum model of tendon pathology. *British Journal of Sports Medicine*. 2016;50(19):1187-1191.
11. De Jonge S, Van den Berg C, De Vos RJ. Incidence of midportion Achilles tendinopathy in the general population. *British Journal of Sports Medicine*. 2011;45(13):1026-1028.
12. Fu SC, Rolf C, Cheuk YC, Lui PP, Chan KM. Deciphering the pathogenesis of tendinopathy: a three-stage process. *Sports Medicine, Arthroscopy, Rehabilitation, Therapy and Technology*. 2010;2:30.
13. Gaida JE, Ashe MC, Bass SL, Cook JL. Is adiposity an under-recognized risk factor for tendinopathy? *Arthritis Care and Research*. 2009;61(6):840-849.
14. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. *Sports Medicine*. 1999;27(6):393-408.
15. Maffulli N, Khan KM, Puddu G. Overuse tendon conditions: time to change a confusing terminology. *Arthroscopy*. 1998;14(8):840-843.
16. Maffulli N, Ewen SW, Waterston SW, Reaper J, Barrass V. Tenocytes from ruptured and tendinopathic Achilles tendons produce greater quantities of type III collagen. *American Journal of Sports Medicine*. 2000;28(4):499-505.
17. McShane JM, Nazarian LN, Harwood MI. Sonographically guided percutaneous needle tenotomy for treatment of common extensor tendinosis. *Journal of Ultrasound in Medicine*. 2006;25(10):1281-1289.
18. Millar NL, Murrell GAC, McInnes IB. Inflammatory mechanisms in tendinopathy. *Nature Reviews Rheumatology*. 2017;13(2):110-122.
19. Tang C, Chen Y, Huang J, Zhao K, Chen X, Yin Z. The roles of inflammatory mediators and immunocytes in tendinopathy. *Journal of Orthopaedic Translation*. 2018;14:23-33.
20. Littlewood C, May S, Walters S. Epidemiology of rotator cuff tendinopathy: a systematic review. *Shoulder and Elbow*. 2013;5(4):256-265.
21. Docking SI, Cook J. Pathological tendons maintain sufficient aligned fibrillar structure on ultrasound tissue characterization. *Journal of Science and Medicine in Sport*. 2016;19(11):919-923.
22. Docking SI, Ooi CC, Connell D. Tendinopathy: Is imaging telling us the entire story? *Journal of Orthopaedic and Sports Physical Therapy*. 2015;45(11):842-852.
23. Rio E, Kidgell D, Purdam C, Gaida J, Moseley GL, Pearce AJ, Cook J. Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. *British Journal of Sports Medicine*. 2015;49(19):1277-1283.
24. Silbernagel KG, Hanlon S, Sprague A. Current clinical concepts: conservative management of Achilles tendinopathy. *Journal of Athletic Training*. 2020;55(5):438-447.
25. Scott A, Squier K, Alfredson H. ICON 2019: International Scientific Tendinopathy Symposium Consensus. *British Journal of Sports Medicine*. 2020;54(20):1183-1185.
26. Dakin SG, Martinez FO, Yapp C. Inflammation activation and resolution in human tendon disease. *Science Translational Medicine*. 2015;7(311):311ra173.
27. Dean BJF, Dakin SG, Millar NL, Carr AJ. Review of tendon pathology and future therapeutic directions. *Sports Medicine*. 2023;53(4):711-728.
28. Millar NL, Silbernagel KG, Thorborg K. Tendinopathy. *Nature Reviews Disease Primers*. 2021;7(1):1.
29. Khan KM, Rio E. Advances in tendinopathy management and rehabilitation. *Lancet Rheumatology*. 2024;6(2):e120-e132.
30. Cook JL, Docking SI. Tendon pathology: understanding the continuum model. *British Journal of Sports Medicine*. 2021;55(21):1189-1190.

31. Ackermann PW, Hart DA. General overview and future perspectives in tendinopathy. *Advances in Experimental Medicine and Biology*. 2016;920:167-181.
 32. Longo UG, Ronga M, Maffulli N. Achilles tendinopathy. *Sports Medicine and Arthroscopy Review*. 2018;26(1):16-30.
 33. Sharma P, Maffulli N. Biology of tendon injury and healing. *Healing and Regeneration*. 2005;13(1):11-19.
 34. Rees JD, Stride M, Scott A. Tendons—time to revisit inflammation. *British Journal of Sports Medicine*. 2014;48(21):1553-1557.
 35. Lui PPY. Tendinopathy: from pathogenesis to treatment. *Chinese Journal of Traumatology*. 2022;25(2):65-72.
 36. Andarawis-Puri N, Flatow EL, Soslowsky LJ. Tendon basic science: development, repair, regeneration, and healing. *Journal of Orthopaedic Research*. 2015;33(6):780-784.
 37. Xu Y, Murrell GAC. The basic science of tendinopathy. *Clinical Orthopaedics and Related Research*. 2008;466(7):1528-1538.
 38. Abate M, Schiavone C, Salini V. Management of limited joint mobility in diabetes and tendon disorders. *Current Diabetes Reviews*. 2013;9(1):74-84.
 39. Fredberg U, Stengaard-Pedersen K. Chronic tendinopathy tissue pathology and imaging. *Scandinavian Journal of Medicine and Science in Sports*. 2008;18(1):3-15.
 40. Magnussen RA, Dunn WR, Thomson AB. Nonoperative treatment of tendinopathy: a systematic review. *Clinical Journal of Sport Medicine*. 2009;19(1):54-64.
 41. Coombes BK, Bisset L, Vicenzino B. Efficacy and safety of corticosteroid injections in tendinopathy. *Lancet*. 2010;376(9754):1751-1767.
 42. Fitzpatrick J, Bulsara M, Zheng MH. The effectiveness of platelet-rich plasma in tendinopathy. *American Journal of Sports Medicine*. 2017;45(1):226-233.
 43. Kaux JF, Crielaard JM. Platelet-rich plasma application in tendon disorders. *Sports Medicine*. 2013;43(7):573-580.
 44. Mani-Babu S, Morrissey D, Waugh C, Screen H, Barton C. The effectiveness of extracorporeal shock wave therapy in lower limb tendinopathy. *American Journal of Sports Medicine*. 2015;43(3):752-761.
 45. Zhi X, Liu X, Han J. Stem cell-based therapies for tendon regeneration and repair. *Stem Cell Research and Therapy*. 2024;15(1):89.
 46. Chen X, Yin Z, Chen JL, Shen WL, Liu HH, Tang QM, et al. Scleraxis-overexpressing tendon stem cells for tendon repair. *Biomaterials*. 2014;35(29):8267-8277.
 47. Wang JHC. Mechanobiology of tendon regeneration. *Journal of Orthopaedic Research*. 2022;40(8):1681-1692.
- Web References**
48. World Health Organization. Musculoskeletal conditions [Internet]. Geneva: World Health Organization; 2022 [cited 2026 Jun 16]. Available from: <https://www.who.int/news-room/fact-sheets/detail/musculoskeletal-conditions>
 49. World Health Organization. Rehabilitation [Internet]. Geneva: World Health Organization; 2024 [cited 2026 Jun 16]. Available from: <https://www.who.int/news-room/fact-sheets/detail/rehabilitation>
 50. Cleveland Clinic. Tendinopathy: Symptoms, causes and treatment [Internet]. Cleveland (OH): Cleveland Clinic; 2024 [cited 2026 Jun 16]. Available from: <https://my.clevelandclinic.org/health/diseases/22289-tendinopathy>
 51. National Health Service. Tendinopathy [Internet]. London: NHS; 2024 [cited 2026 Jun 16]. Available from: <https://www.nhs.uk/conditions/tendonitis>
 52. Mayo Clinic. Tendinitis: Symptoms and causes [Internet]. Rochester (MN): Mayo Foundation for Medical Education and Research; 2024 [cited 2026 Jun 16]. Available from: <https://www.mayoclinic.org/diseases-conditions/tendinitis>
 53. American Academy of Orthopaedic Surgeons. Tendinitis and tendinosis [Internet]. Rosemont (IL): AAOS; 2023 [cited 2026 Jun 16]. Available from: <https://orthoinfo.aaos.org...>