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The Therapeutic Potential of Vitamin D in Tendinopathy: A Review of Current Evidence

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Abstract:

Introduction: Tendinopathy refers to pathological changes in tendons that cause pain, functional impairment, and limited physical activity. These changes often result from repetitive overloading without adequate recovery, leading to microtrauma, collagen disorganization, vascular alterations, and chronic inflammation. While corticosteroids offer short-term relief, they may weaken tendon structure, and regenerative therapies like PRP and stem cells remain inconclusive. Recently, vitamin D has gained attention for its potential role in tendon healing. Low vitamin D levels are linked to higher tendinopathy risk and delayed recovery. This review aims to summarize current evidence on the role of vitamin D in tendon regeneration and its potential therapeutic application.

Materials and methods: A review of selected literature in the PubMed, Scopus, Web of Science and Google Schoolar databases was conducted, using the following keywords: "Tendinopathy", "Vitamin D", "Tendon".

Conclusions: The potential role of vitamin D in tendon regeneration is a subject that offers novel perspectives on the treatment of tendinopathies. The therapeutic options currently available are limited in scope, frequently offering only symptomatic relief without addressing the underlying pathophysiology. The evidence from in vitro and in vivo experiments suggests that vitamin D can modulate inflammation, support collagen synthesis, and protect tenocytes from oxidative stress. Multidisciplinary management of tendinopathy should consider the assessment and correction of vitamin D deficiency, especially in older or physically active individuals. Further high-quality clinical studies are required to confirm these findings, determine optimal supplementation strategies, and evaluate long-term functional outcomes.

Keywords: "Tendinopathy", "Vitamin D", "Tendon"

AI: AI was utilized for two specific purposes in this research. First, text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process

Content

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1. Introduction

Tendinopathy is a term that covers a variety of pathological changes in tendons that lead to pain, impaired function and limitations in daily activity. As they transmit forces from the muscles to the skeletal system, tendons must be extremely resistant to high and repetitive loads, particularly during sporting activities where the force acting on the tendon can increase significantly. Excessive and repetitive loading is a major risk factor for microtrauma, which triggers abnormal biological processes(1).

Tendinopathy most often develops against a background of overload and inadequate recovery time. The result of these processes is the manifestation of complex, interconnected pathological mechanisms, including, but not limited to: disruption of collagen structure, oedema, vascularisation changes and chronic inflammatory responses(2). Despite the multifactorial aetiology of the disease, chronic inflammation is now recognised as a major contributing factor to its progression(3).

Traditional anti-inflammatory treatments, such as corticosteroid injections, provide short-term relief but can weaken the tendon structure. Regenerative therapies such as PRP, growth factors and stem cells have not yet demonstrated conclusive efficacy. However, increasing attention is being paid to vitamin D, which, in addition to its well-known effects on bone health, may also promote tendon regeneration. Studies indicate that vitamin D increases type I collagen synthesis and reduces the action of pro-inflammatory cytokines (IL-6, TNF-α and IL-1β), while also inhibiting extracellular matrix degradation. Deficiency in 25(OH)D is associated with an increased risk of tendinopathy and poorer healing. Supplementation may enhance tendon function and strength, particularly in older and physically active individuals(4–6).

The aim of this paper is to provide an overview of the current scientific evidence on the effect of vitamin D on tendon regeneration processes and to discuss its potential role in supporting the treatment of tendinopathies.

2. Epidemiology

Tendinopathies, which include both acute injuries and chronic degenerative tendon changes, account for more than 30% of all orthopaedic consultations. More than 30 million procedures related to tendon pathology are performed annually, with significant economic costs(7,8).

They are particularly common among athletes - as many as half of sports injuries are overload injuries and often involve tendons. Among the most commonly affected are the rotator

cone, Achilles tendon, patella tendon, thigh adductors or wrist. For instance, Achilles tendinopathy affects up to 30% of runners, and patellar tendon lesions are common in volleyball, handball and basketball(9).

In addition to physical activity, age, gender, occupational work (especially with repetitive movements) and the use of certain medications, including glucocorticosteroids, fluoroquinolones and statins, also contribute to the development of tendinopathy. Metabolic diseases such as gout, hypercholesterolaemia or diabetes can also lead to tendon degeneration. Being overweight and endocrine disorders, such as thyroid disease, are further significant risk factors(10).

3. Common Therapeutic Options

Treating tendinopathies with pharmacological agents is challenging due to the poor vascularisation of tendons, which limits the availability of drugs in the target tissue. Unlike in bone or muscle, drugs directly targeting tendon tissue have not yet been developed. Therefore, in clinical practice, analgesics, non-steroidal and steroidal anti-inflammatory drugs and various injectable methods are often used(11).

Non-steroidal anti-inflammatory drugs (NSAIDs)

Despite widespread use, NSAIDs do not affect chronic tendinopathy without active inflammation. In addition, there is a possibility that they may exert a deleterious effect on regenerative processes. It is therefore recommended that their use be judicious(12).

Glucocorticosteroids (GCS)

Steroids, despite their popularity, have detrimental effects on tendon structure: they cause loss of fibre organisation, reduced cellular viability and weakened mechanical properties of the tissue(13). Although they can provide short-term pain relief (e.g. in the case of tennis elbow), their long-term use shows no advantage over other methods and is not pathophysiologically justified(14).

Hyaluronic acid

Hyaluronic acid has anti-inflammatory effects, promotes cell proliferation and collagen synthesis. Low-molecular-weight hyaluronic acid has found use in the treatment of, among

others, lateral epicondylitis and patellar tendinopathy, although scientific evidence is still limited(15,16).

Platelet-rich plasma

Platelet-rich plasma contains growth factors that stimulate tissue regeneration by activating chemotaxis and cellular proliferation. In vitro and animal studies have shown positive effects, but in human clinical trials the results are less consistent - mainly due to the variety of protocols and the limited number of randomised trials(17).

Prolotherapy

Prolotherapy involves repeatedly injecting solutions (e.g. glucose) into the area of the lesion. This causes a local inflammatory response and subsequently stimulates fibroblasts and collagen synthesis. Although it shows promising effects in various tendinopathies, the reliability of these results is limited by methodological shortcomings of the study(18).

Other injection methods

Other less common therapies are also available, such as glycerol trinitrate patches, 'dry needling' and ultrasound-guided injections of high-volume fluids. While some of these have been shown to be effective, they are neither widely available nor routinely used in clinical practice(19,20).

Traditional anti-inflammatory treatments, such as glucocorticosteroid injections, only provide short-term relief and may even weaken the tendon structure in the long term. In response to these limitations, regenerative therapies such as platelet-rich plasma (PRP), growth factors and stem cells have been investigated. Unfortunately, research to date on these methods has produced inconsistent results and does not unequivocally support their effectiveness in repairing damaged tendons(4,5).

In recent years, there has been a growing interest in the lesser-known functions of vitamin D, beyond its well-established effects on calcium and bone metabolism. Research is increasingly highlighting its potential effects on muscle and soft tissues, including tendons. Laboratory experiments (both in vitro and in vivo) suggest that vitamin D may be able to:

- support type I collagen production,
- inhibit the degradation of the extracellular matrix by reducing the activity of metalloproteinases (MMPs),

- reduce the action of pro-inflammatory cytokines such as IL-6, TNF-α or IL-1β(21,22).

In addition, epidemiological data suggest that vitamin D deficiency may contribute to the development of tendinopathy and hinder the healing process. For instance, around 80% of patients with a rotator cuff injury have reduced 25(OH)D levels. A growing body of research also suggests that vitamin D supplementation can promote tendon regeneration, enhancing its function and strength, particularly in older and physically active individuals(6,23).

4. The role of Vitamin D in tendon disease

Vitamin D is a prohormone and an essential precursor of the active steroid calcitriol, which regulates calcium and phosphate metabolism(24). It is fat-soluble and plays an important role in developing bones and skeletal muscles, as well as promoting overall health. Vitamin D deficiency is associated with reduced bone mineral density, increased risk of fractures, osteopenia, osteoporosis and muscle weakness(25).

In addition to its effects on the skeleton and muscles, vitamin D also has anti-inflammatory properties, suggesting that it may play an important role in the development and treatment of tendinopathies(26). Its active form, 1,25-dihydroxyvitamin D_3 [$1,25(OH)_2D_3$], has been shown to inhibit the production of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumour necrosis factor alpha (TNF-α) and interferon gamma (IFN-γ), while increasing the levels of anti-inflammatory cytokines, e.g. interleukin-10 (IL-10), as shown in studies on immune cells infected with Mycobacterium tuberculosis(27).

Vitamin D also regulates reactive oxygen species (ROS) levels, cyclooxygenase activity and signalling pathways involving the transcription factor NF-κB, thereby enhancing its anti-inflammatory effects(28). It also increases cell proliferation and improves parameters related to tendon repair by activating the ERK and p38 signalling pathways. It has a protective effect on damaged tenocytes (tendon cells), suggesting its potential use as a therapeutic agent for treating tendinopathies(29). In this regard, vitamin D appears to be a promising therapeutic agent for supporting tendon regeneration, due to its beneficial effects on the immune response and stimulation of cell growth and repair.

Effect of vitamin D on the rotator cone – in vitro studies

The effect of vitamin D on tendon regeneration, particularly within the rotator cuff, has been the subject of several experimental studies. In a 2014 laboratory study, Angeline et al. investigated the impact of vitamin D deficiency on the healing process at the tendon-bone attachment site following rotator cuff reconstruction in rats. The animals were placed on a

vitamin D-deficient diet and deprived of UV light exposure before undergoing surgical reconstruction of the supraspinatus tendon attachment using the tunnel method. While no differences in bone mineral density were observed after four weeks, biomechanical studies revealed significantly lower tissue strength in the vitamin D-deficient group after just two weeks. Histological analysis confirmed poorer collagen fibre organisation and less intense bone formation in the deficient group. These results suggest that low vitamin D levels may negatively impact the initial stages of tendon-to-bone healing by weakening the bone structure and impairing muscle function(6).

A study by Maman et al. assessed the effects of vitamin D and other hormonal substances on cells derived from the rat supraspinatus tendon. They analysed cell proliferation and the expression of oestrogen receptors (ERα and ERβ), vitamin D receptor (VDR), scleraxis (SCX) and type I collagen (COL-1). Vitamin D was found to increase tendon cell proliferation via ERα and VDR, but not ERβ. Interestingly, the proliferative effect was not correlated with an increase in SCX or COL-1 expression - on the contrary, their levels were reduced(30).

In a recent 2023 study, Kim et al. analysed samples of the supraspinatus muscle, shoulder muscle, and supraspinatus tendon taken during arthroscopy from patients with different levels of vitamin D. They found that patients with vitamin D deficiency had markedly elevated levels of pro-inflammatory cytokines (IL-1β and IL-6) in their rotator cuff muscles. However, no significant differences in gene expression were observed in the tendon itself (31).

Effect of vitamin D on the rotator cone - in vivo studies

In recent years, numerous studies have evaluated the role of vitamin D in the development of rotator cuff injuries and surgical outcomes. Two studies have examined hypovitaminosis D as a potential risk factor for such injuries. A retrospective analysis by Lee et al. involving 176 patients undergoing arthroscopic reconstruction of a full-thickness lesion found that as many as 44.3% were vitamin D deficient (<20 ng/mL) and only 29% had normal levels (>30 ng/mL). The study also noted that low vitamin D levels were more prevalent among younger patients and individuals employed in indoor occupations, suggesting the potential significance of lifestyle factors in the aetiopathogenesis of the condition(32). By contrast, a study by Liu et al. found that low vitamin D levels were an independent risk factor in patients with a concomitant diagnosis of osteoporosis and rotator cuff injury, suggesting a possible prognostic role(33).

Subsequent studies have assessed the effect of vitamin D levels on surgical outcomes for rotator cuff injuries. In a study by Ryu et al. 88% of patients were found to be deficient in

vitamin D prior to surgery. However, no significant correlations were identified between vitamin D levels and lesion size, muscle fat infiltration, or functional outcomes following surgery(34). Similarly, Degen et al. found no significant differences in recurrence rates or functional outcomes between patients with deficient and normal vitamin D levels following arthroscopic repair(35).

By contrast, Harada et al. conducted a retrospective study involving nearly 1,900 patients and demonstrated that vitamin D deficiency was associated with an increased likelihood of experiencing postoperative shoulder stiffness that required surgical revision(36). Similar observations were made by Cancienne et al., who documented a higher reoperation rate in patients with low vitamin D levels. While the differences were statistically significant, they were relatively small and subject to overlapping confidence intervals, which limited the clinical relevance of these results(37).

A more recent study by Chen et al., which involved 89 patients, found that those with vitamin D deficiency were more likely to experience tendon re-tears (26.7% vs. 9.1%) and experienced greater pain and fat infiltration of the supraspinatus muscle during the first three months after surgery. Notably, however, there was no correlation between vitamin D levels and the extent of damage, symptom duration or functional outcomes at later follow-ups(38).

In conclusion, the results of in vivo studies are inconsistent. While low vitamin D levels may be associated with poorer early postoperative outcomes and a higher risk of complications, not all studies support the idea that they impact distant structural and functional outcomes. Further prospective studies are needed to establish the clinical relevance of vitamin D in treating rotator cuff injuries.

Effect of vitamin D on tendon healing - in vitro studies

In a 2019 study, Min et al. investigated the impact of vitamin D on damaged human tenocytes (tendon cells). In the study, the cells were damaged using dexamethasone (Dex) under laboratory conditions, which resulted in the inhibition of their growth and a decrease in the expression of characteristic tendon markers, such as mohawk (MKX), scleraxis (SCX), tenomodulin (TNMD), tenascin C (TNC), and types I and III collagen (COL-1 and COL-3). Following the administration of vitamin D, gradual restoration of cell proliferation in a dose-dependent manner, as well as increased TNMD and COL-1 expression, was observed. These protective mechanisms were attributed to the antioxidant effects of vitamin D, which reduce reactive oxygen species (ROS) levels, and its effects on ERK and p38 signalling pathways. Additionally, the presence of vitamin D receptors (VDR) and the enzyme 1α-hydroxylase in

tendon cells suggests that they are capable of actively responding to vitamin D, in a manner similar to bone and muscle cells(39).

Subsequent research was conducted by Kim et al., who developed a novel vitamin D delivery system based on hyaluronic acid (HA) hydrogel and Tween 80 (T80) surfactant. The formulation, designated as Vit D@Gel/T80, combined the anti-inflammatory properties of hyaluronic acid (HA) with the biological activity of vitamin D. In vitro, this formulation protected human tenocytes from TNF-α-induced damage by inhibiting the NF-κB pathway, phosphorylation of ERK and p38 proteins, and down-regulating inflammatory cytokines (COX-2, IL-6). Furthermore, the expression of TNC and COL-3 was reduced, indicating regenerative properties. In the course of the present study, rats with collagenase-induced tendinopathy were treated with an injection of Vit D@Gel/T80. After a period of four weeks, it was evident that the rats had recovered the morphological structure of the tendons, as evidenced by a smooth, shiny appearance that was similar to healthy tissue. Concurrently, there was a decrease in the expression of genes associated with inflammation (TNF-α, COX-2, NF-κB) and apoptosis (BAK, BID), and an increase in the expression of genes typical of healthy tendons (SCX, TNMD, COL-1A1)(40).

In conclusion, the in vitro results suggest that vitamin D protects tenocytes from oxidative stress and inflammatory damage while promoting tendon regeneration at molecular and morphological levels. These findings could lay the groundwork for further research into using vitamin D to treat tendinopathies.

Effect of vitamin D on tendinopathies - in vivo studies

Clinical studies suggest that vitamin D deficiency may be associated with some forms of tendinopathy. Cavalli et al. conducted a study in which they evaluated markers of calcium phosphate metabolism, including vitamin D levels, in 30 patients with calcific rotator cuff tendinitis. These patients underwent ultrasound-guided percutaneous two-needle therapy. Twenty-eight of the patients had low vitamin D levels and high (or borderline) levels of calcitriol — the active form of vitamin D — and parathyroid hormone (PTH), which may indicate secondary hyperparathyroidism associated with vitamin D deficiency. The authors hypothesized that transient hyperparathyroidism may promote the formation of heterotopic calcification within tendons and that vitamin D supplementation, by regulating PTH levels and having a direct effect on connective tissue cells, may limit or prevent the development of this pathology(41(p28)).

By contrast, Yaka et al. conducted a retrospective study to evaluate the potential link between vitamin D levels and lateral epicondylitis of the humerus (tennis elbow). The study compared patients diagnosed with the condition (the 'study group') with patients without symptoms (the 'control group'). Vitamin D levels were significantly lower in the tendinopathy group ($p < 0.001$). As many as 77.5 per cent of patients with epicondylitis were deficient in vitamin D, compared to 12.5 per cent with normal levels. By comparison, deficiency was present in 43.9% of patients in the control group, while normal levels were recorded in 25.8%. The authors suggested that low vitamin D levels may be a risk factor for developing this type of tendinopathy(42).

In conclusion, in vivo studies suggest a potential link between vitamin D deficiency and an increased risk of developing certain tendinopathies, such as calcifying rotator cuff arthritis or tennis elbow. While the exact mechanisms are not fully understood, vitamin D's role as a risk factor and potential target for preventive interventions appears promising and merits further investigation.

5. Summary and Conclusion

Tendinopathy is a complex condition whose development is influenced by biomechanical and molecular factors, such as chronic inflammation and impaired regenerative processes. Traditional therapies, such as non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroid injections, only provide short-term relief and may interfere with the body's natural tendon repair mechanisms. Although regenerative therapies (PRP and stem cells) show promise, their efficacy has yet to be conclusively proven.

In recent years, the role of vitamin D in the regeneration of soft tissues, including tendons, has received increasing attention. Numerous in vitro and in vivo studies indicate that vitamin D can modulate the inflammatory response, promote collagen synthesis, and protect tendon cells from oxidative stress and degradation. The presence of vitamin D receptors in tenocytes, along with its effect on signaling pathways involved in tissue repair, supports the therapeutic potential of vitamin D. Furthermore, clinical data suggest that vitamin D deficiency may increase the risk of developing tendinopathy and be associated with poorer surgical outcomes.

Although the results to date are promising, they are still somewhat inconsistent. Further well-designed, randomized, prospective studies are needed to clearly assess the efficacy of vitamin D supplementation in treating tendinopathies, determine optimal doses, and identify patient groups who could benefit most from such therapy. Nevertheless, vitamin D appears to

be a safe, accessible and inexpensive means of promoting tendon regeneration, particularly in cases of tendon deficiency.

Disclossure

Supplementary Materials

Not applicable.

Author's contribution

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