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Clinical reasoning in patients with lower limb tendinopathy
A rehabilitation framework

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Κλινικός συλλογισμός σε ασθενείς με τενοντοπάθεια κάτω άκρου. Ένα μοντέλο αποκατάστασης

Περίληψη στο τέλος του άρθρου

Tendinopathy is an "umbrella" term that indicates a load-related, non-rupture injury of the tendon. The etiology of tendinopathy can be considered as multifactorial, and its identification and management have proved to be difficult. Numerous studies have concluded that in the majority of patients with tendinopathy, there is a failure of the healing process, and as a result, the limbs remain dysfunctional. The purpose of this study was to explore the scientific evidence related to the main theories, in order to provide a clinical framework for rehabilitation in patients with lower limb tendinopathy. The proposed model involves two different routes. The first route is based mainly on a structural concept, clearly related to the stage of pathology, biomechanics and function of the tendon, and the healing process. The second route focuses on the kinetic system as a whole, following the functional concept of a kinetic chain. The resulting proposed algorithm is based on both structural and functional features, according to current evidence, providing a clinical reasoning framework that aims to bridge the gap between research and clinical practice and to empower the patient-centered approach.

Key words

Clinical reasoning
Tendinopathy
Tendon pain

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1. INTRODUCTION

Overuse injury commonly occurs in loaded tendons. The term that is routinely used to describe this pathology is "tendinopathy". Tendinopathy is an "umbrella" term that indicates a load-related, non-rupture injury of the tendon.[1,2] The prevalence and incidence rates of lower limb tendinopathy have been reported as 11.83 and 10.52 per 1,000 persons, respectively, and it appears that lower limb tendinopathies are more prevalent among older patients.[3] Since the etiology of tendinopathy can be considered multifactorial, its identification and management have proved to be difficult. The main symptoms of lower limb tendinopathy are load-related pain localized in the tendon, and reduced functional capacity, particularly in energy storage activities such as running, jumping, etc.[4,5] There is no apparent correlation between pain and the pathophysiological changes or inflammatory response in the tendons.[4,6] In addition, in the majority of patients with tendinopathy, there is failure of the healing process and as a result, the limbs remain dysfunctional.[7,8] The relevant literature, however, provides promising data regarding the possible mechanisms and treatment options in patients with tendinopathy.[9]

The purpose of this study was to explore the scientific evidence related to the main theories, in order to provide a clinical framework for rehabilitation and to build an intervention algorithm for patients with lower limb tendinopathy.

2. BASIC PRINCIPLES AND TENDINOPATHY MODELS

Tendon pathology appears to have no clear relationship with inflammation, and therefore inflammation is not a key factor.[4,10,11] Although an increased concentration of cytokines has been observed, no typical inflammation process is detected.[1,10,12] Rather, it is considered to be a load-related degenerative pathology, in which pain is not related to the pathological state of the tendon (i.e., as presented in imaging studies).[4,11] The various risk factors can be separated into intrinsic and extrinsic. Extrinsic factors are load-related, such as the type of load (e.g., compression), the magnitude of the load, the duration of exercise, the rest time, the loading cycle, the environment, etc. Intrinsic factors are related to the personal characteristics, and include the muscle strength, elasticity, the athletic technique, etc.[11,13] Numerous theories have been put forward in an attempt

to interpret the pathological and recovery mechanisms of the tendon. Some of the most popular theories include the *mechanical theory*, the *vascular theory*, the *neural theory* and the *new continuum model of Cook*.[5,9,10]

According to the *mechanical theory*, repeated loading within the physiological stress range of the tendon causes tendon fatigue and, as a result, leads to tendon failure and microscopic degeneration. This theory introduces some valid points but fails to explain how chronic repetitive damage of the tendon accumulates over time, or why this damage would be degenerative rather than inflammatory.[10,12,14]

The *vascular theory* suggests that because metabolically active tissues require a vascular supply, supply disorder may cause degenerative damage. This theory, however, is unable to explain why some tendons do not present the same mechanism of pathology, or why a young healthy athletic population would be susceptible to vascular compromise.[10]

The *neural theory* suggests that tendon pain is an indication of the sensitization of nociceptive neurotransmitters and, in the chronic state, a mechanism of central sensitization is responsible.[4,10,15] Although this theory explains some aspects of tendon pain, it is unable to explain the actual mechanism of the pathology and response to treatment. Tendinopathy appears to be a special form of chronic pathology, because it cannot be fully explained by peripheral or central pain mechanisms.[4]

The *continuum model,* based on homeostasis theory and time-phase behavioral presentation of pathology, demonstrates the different phases and outcomes in clinical practice. According to this model, tendinopathy constitutes a spectrum ranging from normal to pathological tendon, presenting three stages that differ in characteristics and treatment. The first stage is characterized as reactive tendinopathy, in which the tendon presents a reactive, painful, non-inflammatory, proliferative response. It is proposed that this response is caused by acute tensile or compressive overload. At this stage, the tendon would have the potential to revert to normal if the overload was sufficiently reduced or managed appropriately. The second stage is characterized as tendon disrepair. At this stage the damage is greater (structural damage). Long duration overloaded tendons can be considered to be at this stage, and reversibility of the pathology is still possible by load management and exercise. The last stage is characterized as degenerative tendinopathy, in which progressive matrix and cell changes have been incurred. At this stage there is limited or no capacity for reversibility of the pathological changes.[5] Although this model advances our knowledge regarding the tendon pathology and its management, it presents some limitations. Although it explains tendinopathy more comprehensively than other theories, this model is based on structural features and therefore does not clearly imply a direct relationship between structure, pain and dysfunction. In *Cook's* upgraded model,[9] the tendon is presented as producing painful, degenerative and healthy portions all at the same time. A second key feature of this model is that degenerative portions of the tendon are mechanically silent, which might result in overloading in the normal portion of the tendon. This could explain why chronically degenerative tendons present failure of the healing process. The reactive stage, therefore, could be present at the beginning, when the overloading occurred, and the tendon could suffer greater damage, as in the disrepair and degenerative stage. Because of this, the clinician must treat the condition early, in order to promote the rehabilitation process. In the disrepair stage, reversibility is still possible. To address these matters, there are two possible pathways; if the damage is not too massive, the tendon might have the potential to heal, and thus the treatment methods must focus on this aspect. If the damage is too severe and some portions have already sustained degenerative damage (in the new model this portion is considered to be mechanically silent), then the focus should be as functional as possible. Irrespective of the route (disrepair-degenerative or disrepair-normal), the principal goal is considered to be the functional outcome, which is possible, mainly by using progressive exercises.[9] In conclusion, this model focuses on pain and function as part of the same problem, but with different answers, while it departs from the structural features on which treatment was previously based.

3. DIAGNOSIS-ASSESSMENT

The differential diagnosis of tendinopathy in sports and exercise populations is based on a thorough history and physical assessment. The patient's history may be critical for the identification of tendon pathology, and localized pain and its relationship to load are considered to be the defining clinical features. Pain is also related to the intrinsic and extrinsic factors discussed above. In addition, pain may be diminished by repeated loading but there is always a worsening the next day, due to energy storage activities. Pain is rarely experienced in the resting state, but occurs immediately on loading and ceases when the load is removed.[4,11] During physical assessment, the point at which localized pain increases in parallel to the increase in load, can be considered to be tendon pain. This can be validated by recording tendon pain and load management capacity

in response to a progressive loading stimulus.[16–18] Stretching tests may have some clinical utility in combination with other tests.[19,20] Palpation appears to have low specificity for diagnosing tendinopathy.[21] The clinician should evaluate muscle strength and elasticity, range of joint motion and motor control deficits of the kinetic chain. Irrespective of the degree of tendon disorganization, atrophy and or reduced strength of the quadriceps, gluteus maximus, calf and core muscles are often observed in lower limb tendinopathies.[22,23] Hamstring, calf and quadriceps flexibility may be diminished in lower limb tendinopathies.[13] Functional tests, such as jumping, squatting, walking and running may detect deficits in strength or motor control.[22–28] Foot posture and static balance also need to be assessed, since these are indicative of muscle imbalance and impaired motor control strategies, and may result from different pathologies.[29,30] Questionnaires such as the Victorian Institute of Sport Assessment (VISA) can be used to assess the severity of symptoms at diagnosis, and to monitor the outcome of intervention.[31–33] Last but not least, the clinician should use daily pain provocation tests in order to monitor the patient's condition, and should assess the pain by the 24 hour response after each rehabilitation session or any energy storage activity. The pain level should always remain low, less than 5/10 on the Visual Analogue Scale (VAS).[16–18]

Since there is poor correlation between abnormal tendon structure and pain, imaging plays a minimal role in the diagnosis and prognosis of tendinopathy, although it can support the differential diagnosis, confirming tendon rupture or peri-tendon pathology.[6,34]

4. A CLINICAL FRAMEWORK FOR THE MANAGEMENT OF PATIENTS WITH LOWER LIMB TENDINOPATHY

The proposed framework is a continuum model based on current evidence, and it involves two different routes (fig. 1). The first route is based mainly on the structural concept, and is thus clearly related to the pathological stage, biomechanics and function of the tendon and the healing process.[35,36]

The second route focuses on the kinetic system as a whole, following the functional concept of the kinetic chain. The movement system is basically composed of muscular, articular and neural components. To address these components, this route is divided into three stages: management of biomechanical factors, muscle imbalance rehabilitation and motor control-sensorimotor rehabilitation. It is crucial that these routes create an integrated model, based on the findings of the assessment and the goals of the patient.

4.1. The tissue adaption route

The tissue adaption route is one of the two routes of the proposed algorithm for pain management and func-

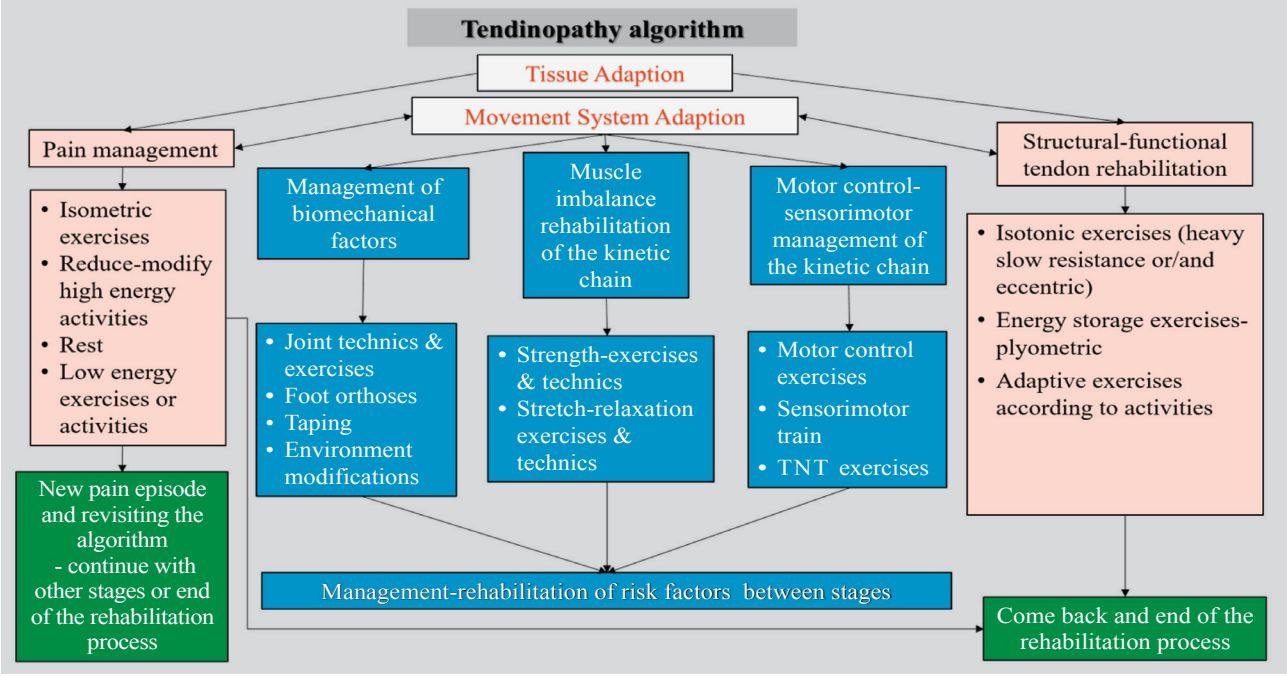

Figure 1. An intervention algorithm for patients with lower limb tendinopathy (does not fall under copyright).

tional rehabilitation of the tendon (fig. 1). Addressing the symptoms is usually the first step in treatment. According to *Cook's* model, patients with tendon pain are allocated to the reactive stage. In this algorithm, management of pain is usually the first step, and if the problem is a reactive response caused by overloading with no tissue damage or degeneration, then this may be the end of the algorithm. Load modification is the key concept during this stage. The patient should avoid energy storage exercises, and he(she) may need to modify related activities. Activities such as walking or cycling may be beneficial for the patient's general physical condition, but the severity of pain needs to be kept at a low level even during high energy activities, and the pain should not get worse during the 24 hours following any activity.[16–18,37–39] According to recent evidence, isometric contraction appears to be promising for immediate pain reduction. A lower duration of contraction may be beneficial, but the patient needs to make an effort ("heavy" contractions) without worsening his(her) symptoms.[40–42] These exercises should be repeated several times daily, especially in the early stages of treatment.

Strengthening exercises appear to have a therapeutic effect on tendon morphology and function.[2,14,36,43] Progressive exercises, such as isotonic strengthening, can be introduced into the rehabilitation program when pain is minimal (3 or less on the Numeric Rating Scale [NRS]). Heavy slow resistance exercises appear to be as effective as eccentric exercises, so the principal key is load-related factors such as type, magnitude, etc.[44–46] According to current evidence, the eccentric factor is not a key feature, and it is proposed to include isotonic exercises in the rehabilitation program. Finally, isometric exercises can be used if pain is still present during the rest day or when pain is aggravated. If the patient is an athlete using movements involving lower limb energy storage or impact loading, then plyometric exercises and exercises that simulate energy storage activities of everyday living should be the key component. The criteria for starting this type of exercises are: Sufficient muscle strength, minimal or no pain during energy storage activities, and return of symptoms to baseline in the following 24 hours. The choice of exercises will depend on individual sport, and thus, the selection and also the parameters of energy storage programs may vary widely.[16–18,47]

Ending the rehabilitation program and return to participation in sport is recommended when full training is tolerated without provocation of symptoms (24-hour response on a load test) and there is successful response to the clinical tests.[48,49]

4.2. Movement system adaption route

The movement system adaption route is the second route of the proposed algorithm, and it focuses on management of the kinetic system as a whole, along with management of possibly related intrinsic and extrinsic factors (fig. 1). Rehabilitation according to the movement system route can start from the beginning or during different phases of rehabilitation, but is based on the findings during assessment and the understanding of how these findings are related to the patient's pathology. Basically, the therapist aims to improve the patient's movement during various activities. It is well known that pain has an effect on movement patterns but it is not clear whether the changes precede or follow the pain.[26,50] Flexibility, strengthening and motor control issues contribute to both sensory and motor systems.[22,26] The treatment according to this route is aimed at three categories of deficits: biomechanical, muscular and sensorimotor. Biomechanical deficits may be joint stiffness or poor joint alignment. Environmental or equipment management can be included in this category because of the relation with ergonomic principles. Muscle deficits may be muscle tightness or weakness that could alter the movement pattern, and reduced capacity of the muscles may lead to pathology or injury.[26,51] Sensorimotor deficits may consist of poor motor control, proprioception or technique of the patient during different activities.[26,52–54]

Exercises of the whole kinetic chain are needed to improve the patient's functionality. Flexibility deficits may be a risk factor for tendinopathy or other pathologies. Strengthening and stretching exercises may be a part of the treatment plan, especially if there is a relation with poor functioning.[13,53] Manual therapy techniques, orthotics or even taping constitute valid options for addressing the patient's intrinsic risk factors.[55–59] Finally, motor control and learning strategies can be used in combination with exercises during the rehabilitation process. Rio and colleagues[52] suggest that external pace during contractions could be a good method for enhancing the biomechanical properties of the tendon, as well as neuroplasticity. Proprioception training and retraining of movement patterns such as lumbo-pelvic control, landing strategy or running patterns should be part of the treatment, and these practices need to be wisely used.[24,52–54,60]

5. CONCLUSIONS

The proposed algorithm is based on structural and functional features combined with current evidence to

provide a clinical reasoning framework that bridges the gap between research and clinical practice. There are a few limitations to use of this algorithm; although it contains features concerning the function and pathology of tendons, and also the movement system concept, it does not include other factors, such as cognitive-psychological features, central sensitization, nutrition, etc. The main concept on which this reasoning framework is based ensures the active contribution of the patient, empowering the patient-centered approach. Further research is needed on the possible mechanisms and other factors such as central sensitization, in order to provide a better understanding, with a focus on evaluation of improvement of function and satisfaction of the patient.

ΠΕΡΙΛΗΨΗ

Κλινικός συλλογισμός σε ασθενείς με τενοντοπάθεια κάτω άκρου. Ένα μοντέλο αποκατάστασης

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Ο όρος τενοντοπάθεια αποτελεί έναν όρο «ομπρέλα» που αφορά στην παθολογία του τένοντα και σχετίζεται άμεσα με τη φόρτισή του. Η αιτιολογία της τενοντοπάθειας μπορεί εύκολα να χαρακτηριστεί πολυπαραγοντική. Επομένως, τόσο η διάγνωση όσο και η αντιμετώπισή της μπορεί να αποδειχθούν αρκετά δύσκολες. Από την αρθρογραφία προκύπτει ότι ασθενείς με τενοντοπάθεια παρουσιάζουν δυσκολία στην αποκατάστασή τους καθώς παραμένουν λειτουργικά ελλιπείς. Σκοπός της ανασκόπησης είναι η μελέτη της σύγχρονης και σχετικής αρθρογραφίας, καθώς και η μελέτη μερικών βασικών θεωριών, με αποτέλεσμα τη δημιουργία ενός κλινικού μοντέλου αποκατάστασης για ασθενείς με τενοντοπάθεια κάτω άκρου. Το μοντέλο που προτείνεται έχει δύο κατευθύνσεις. Η πρώτη ακολουθεί μια πιο δομική κατεύθυνση βασισμένη στην εμβιομηχανική, στη λειτουργικότητα του τένοντα και στην επούλωσή του. Η δεύτερη κατεύθυνση έχει ως στόχο τη βελτίωση της λειτουργίας του κινητικού συστήματος μέσα από μια περισσότερο «ολιστική-λειτουργική» προσέγγιση της κινητικής αλυσίδας. Ο προτεινόμενος αλγόριθμος αποτελείται τόσο από δομικά όσο και από λειτουργικά χαρακτηριστικά σύμφωνα με τη σύγχρονη αρθρογραφία, με σκοπό τη δημιουργία ενός μοντέλου κλινικού συλλογισμού που στοχεύει στη γεφύρωση του κενού μεταξύ της έρευνας και της κλινικής πρακτικής, αλλά και στην ενίσχυση του ασθενο-κεντρικού προτύπου παρέμβασης.

Λέξεις ευρετηρίου: Επώδυνος τένοντας, Κλινικός συλλογισμός, Τενοντοπάθεια

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