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Biomechanics, pathomechanics, diagnosis, treatment, and return to play criterias of lateral ankle sprains: an evidence-based clinical guideline

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ABSTRACT

Lateral ankle sprain (LAS), which is common in recreational and professional athletes, recurs and becomes chronic if left untreated. Since the number of mechanoreceptors it contains is high, LAS should not be considered only as a musculoskeletal disorder. A detailed clinical evaluation is recommended. Clinically, the patient presents with limitation of dorsiflexion range of motion (ROM), muscle atrophy and unequal strength between the muscles, deterioration in walking, running and jumping, and proprioceptive losses. The patient should be approached from a biopsychosocial perspective both in the evaluation and treatment phases. The treatment of the patient is carried out in 3 phases and the transition between phases should be based on mentioned criterias. In the acute phase of rehabilitation, emphasis should be placed on eliminating pain and edema, and preventing loss of ROM and muscle strength. In the subacute and chronic stages, emphasis should be placed on increasing the proprioceptive sense and muscle strength, improving the biomechanics of daily life and sports-specific movements such as walking, running, jumping. If the patient is an athlete, return to sports should be planned after the criterias met.

Keywords: Lateral ankle sprain, rehabilitation, foot, ankle

INTRODUCTION

Stabilization of the talocrural joint is provided by the passive subsystem, the active subsystem, and the neural subsystem. While the active substructures responsible for stabilization are intrinsic and extrinsic foot muscles, the neural subsystem is musculotendinous receptors, local and global ligamentous receptors, and plantar cutaneous receptors.[1,2] The passive subsystem consists of the bone structures that make up the arches, the plantar fascia and the ligaments.[1] Medial to the ankle joint is the deltoid ligament, while lateral to the ankle joint is the anteriortalofibular ligament (ATFL), calcaneofibular ligament (CFL) and posteriortalofibular ligament (PTFL). Injury to one or all of these ligaments is called a lateral ankle sprain (LAS). LAS is also known as inversion or over-supinated injuries.[2] LAS is not only a musculoskeletal injury but also a neurophysiological injury due to the high number of mechanoreceptors contained in the ligaments.[3,4] LAS is one of the most common musculoskeletal injuries among both recreational and professional athletes. LAS accounts for 15-20% of injuries in the musculoskeletal system. LAS is seen in 50% of sports involving movements such as jumping, running, and cutting. While ATFL is injured in isolation in 65-73% of these injuries, ATFL and CFL are injured together in 25% of these injuries.[5]

FUNCTIONAL ANATOMY AND BIOMECHANICS OF THE FOOT-ANKLE COMPLEX

Ankle joint is composed of three joints: the talocrural joint, the subtalar joint and the distal tibiofibular joint.[6] Talocrural joint; the dome of the talus is formed by the union of the medial malleolus, lateral malleolus, and the lower surface of the tibia.[6] Due to its shape during walking, the talocrural joint is responsible for transferring the load by external/internal rotation of the tibia and fibula and pronation/supination of the rearfoot. The axis of rotation of the talocrural joint passes through the medial and lateral malleolus.[6] The axis of rotation passes slightly behind the fibula as it passes slightly in front of the tibia in the frontal plane. While the dorsiflexion/plantar flexion movement, which is the main movement of the talocrural joint, occurs in the sagittal plane, a small movement occurs in this joint in the transverse and frontal planes.[7] Passive stabilization of the talocrural joint is provided by the joint capsule, ATFL, CFL, PTFL, and deltoid ligament.[2]

The ATFL is located on the dorsolateral aspect of the foot. It courses from the lateral malleolus anteriorly and medially to the talus at an angle of approximately 45 degrees in the frontal plane. ATFL averages 7.2 mm wide and 24.8 mm long.[8] In vitro kinematic studies have shown that ATFL

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prevents anterior displacement of the talus from the tenon and excessive inversion and internal rotation of the talus on the tibia.[9] The tension in the ATFL increases as the ankle moves from dorsiflexion to plantar flexion. Studies on the ATFL, PTFL, CFL, and deltoid ligament have shown that the ATFL has the least tensile and maximum load-bearing capacity. This explains why the ATFL is most often injured.[10]

The CFL runs downward and posteriorly from the lateral malleolus and attaches to the lateral calcaneus at an angle of approximately 133 degrees from the long axis of the fibula.[8] CFL restricts excessive supination of both the talocrural and subtalar joints. In vitro experiments have shown that CFL restricts excessive inversion and internal rotation of the rearfoot and is most tense when the ankle is in dorsiflexion.[9] The CFL is the second most frequently injured ligament within the lateral talocrural ligaments.[9]

The PTFL runs posteriorly from the lateral malleolus and terminates in the posterolateral portion of the talus. The PTFL has wide endings on both the talus and the fibula. It limits both inversion and internal rotation of the talocrural joint during walking. PTFL is the least injured of the lateral ankle ligaments.[10]

The distal tibiofibular joint is formed between the distal ends of the tibia and fibula. This joint, in the form of syndesmosis, has limited range of motion between the two bones.[13] However, the limited gliding movement in this joint is important for the normal mechanics of the entire ankle complex. In the LAS, the normal position of the distal tibiofibular joint is impaired and therefore the ankle dorsiflexion movement is restricted.[11,12] Therefore, posterior and superior mobilizations to the fibula bone in the early and middle period of rehabilitation allow patients to relieve their symptoms.[13]

By contracting the muscles, they create stiffness in the muscle-tendon units and thus contribute to the dynamic stabilization of the joints. Muscles are responsible for releasing movement by concentric contraction, and by eccentric contraction, they make an important contribution to dynamic stabilization.[14,15] The peroneus longus and brevis muscles contract eccentrically to support the lateral ankle ligaments during supination of the rearfoot. In addition, the tibialis anterior, extensor digitorum longus, extensor digitorum brevis and peroneus tertius muscles on the front of the leg contract eccentrically during supination of the hind foot and support the lateral ligaments. These muscles provide support by slowing the ankle plantar flexion movement, especially when the bare foot is in supination.[14,15]

INJURY MECHANISM

LAS most commonly occur due to excessive supination of the rearfoot about an externally rotated tibia and fibula soon after initial contact of the bare foot during gait or landing from a jump.[16] Excessive inversion and internal rotation of the bare foot, coupled with external rotation of tibia and fibula, results in strain to the lateral ankle ligaments. If the strain in any of the ligaments exceeds the tensile strength of the tissues, ligamentous damage occurs. Increased plantar flexion at initial contact appears to increase the likelihood of suffering a lateral ankle sprain.[16] The ATFL is the first ligament to be damaged during a lateral ankle sprain, followed most often by the CFL. PTFL injury appears to be least common.[16]

After LAS, damage to the ligaments and sensory disturbances in the central nervous system occur.[17] Subsequently, changes in ligament structure and inhibition of spinal reflexes appear. The result is altered joint loading and altered normal movement patterns. Thus, the risk of osteoarthritis in the ankle increases.[17]

CLASSIFICATION OF LATERAL ANKLE SPRAIN

LAS can be classified according to the severity and duration of the injury. Traditionally, LAS are diagnosed by a grading system. Grade I LAS are the most common type of sprains, and patients with grade I LAS typically present with mild pain and swelling over the ATFL with no joint instability.[18] Patients with grade II LAS present with greater disability, moderate pain, and swelling over both the ATFL and the CFL. Furthermore, grade II LAS typically involve complete rupture of the ATFL and stretching of the CFL fibers. The most severe sprains are classified as grade III, but these are relatively uncommon in athletes and result in extreme disability. This type of injury involves damage to the ATFL, CFL, and PTFL.18 According to the duration of the injury, it is called acute ankle instability for 0-6 weeks, subacute for 6-12 weeks, and chronic ankle instability for 12 weeks and above.[5]

CHRONIC ANKLE INSTABILITY

Chronic ankle instability (CAI) is defined as a continuous ankle sprain and feeling of emptiness 1 year after the ankle injury. CAI is divided into functional and mechanical.[19] If anatomical changes occur in the first ankle injury, mechanical instability develops in the ankle. Mechanical instability with untreated injury leads to other changes. These changes are pathological laxity, deterioration in arthrokinematic movements, and degenerative joint damage. These changes can occur in isolation or in combination with each other.[18]

Injury to the lateral ligaments of the ankle results in adverse changes to the neuromuscular system that provides dynamic support to the ankle. Freeman et al.[20] first described the concept of functional instability in 1965. They attributed impaired balance in individuals with lateral ankle sprains to damaged articular mechanoreceptors in the lateral ankle ligaments, which resulted in proprioceptive deficits. The contribution of impaired proprioception, while important, does not fully explain why ankle-ligament injury predisposes athletes to functional ankle instability. The pathoetiologic model is not com plete without including impaired neuromuscular control, thus resulting in inadequacies of the dynamic defense mechanism protecting against hypersupination of the rearfoot.[4]

CLINICAL EVALUATION

Usually following 3 tests are used in physical examination:

- Anterior drawer test
- Anterolateral drawer test
- Reverse anterolateral drawer test

Anterior Drawer Test

Musculoskeletal care providers routinely examine talocrural joint integrity with the anterior drawer test (ADT) to identify the severity of anterior talocrural joint laxity in the acute setting, to advance clinical progression during rehabilitation, or to assign subjects to a research group on the basis of those joint examinations. The ADT presumably can be used to detect the presence of injuries to the ATFL. The clinician performs the test by manually applying an anteriorly directed force at the calcaneus or a backward push on the tibia and attempting to discern pathological talocrural joint laxity from normal physiological laxity.[21] van Dijk et al. evaluated 160 inversion ankle injuries and reported a sensitivity of 71% and specificity of 33% in the diagnosis of ankle ligament tears when the physical exam was based on a cluster of signs and symptoms, such as the formation of a hematoma, location of pain, palpation, and the results of the ADT, with arthrography used as the gold standard.[22] In the ADT, when the ATFL is injured, the medial deltoid ligament can remain intact, which can lead to false negative results.[23]

Anterolateral Drawer Test

The test is performed in a sitting position with the calf hanging over the edge of the examination bed. While the tester stabilizes the tibia with one hand and pulls the calcaneus forward with the other hand while the ankle is plantar flexed, he tries to feel the forward displacement of the talus with his thumb. More specifically, the index and middle fingers are pressed firmly against the posterior aspect of the heel to provide the force directed forward. The palm supports the sole of the foot to provide 10°–15° plantar flexion and tighten the lateral ligaments. The thumb is placed on the relatively flat plane of the lateral surface of the anterior talar dome and 1 cm proximal to the tip of the anterior surface of the lateral malleolus. Evaluation is done as in ADT.[24]

Reverse Anterelateral Drawer Test

The examination is perform, while the patient was lying in the bed with the knee flexed and the angle of the knee adjusted to facilitate plantar flexion. The heel is completely pressed on the bed by the examiner with one hand after adjusting the ankle to a 10°–15° degree plantar flexion and unconstrained internal rotation. The index and middle fingers is place along the relatively smooth plane of the lateral aspect of the anterior talar dome and the anterior aspect of the lateral malleolus 1 cm proximal to its tip. The other hand hold the distal tibia, and the base of the palm pushed against the tibia to induce a posteriorly oriented displacement of the tibia with a force parallel with that of the articular surface arch of the talus. This test is more sensitive than ADT and ADLT.[25]

CLINICAL SYMPTOMS

Inflammation symptoms such as pain, edema, increased temperature, redness and loss of function occur after acute ankle injury. In CAI, the symptoms are divided into 4 main headings: i) limitation of movement, ii) loss of strength, iii) loss of balance, and iv) functional limitation.[26]

Range of Motion

Loss of range of motion (ROM) in LAS may occur due to arthrokinematic and osteokinematic reasons. ROM is the both a cause and a consequence of the LAS. It has been reported that patients with ankle dorsiflexion limitation of motion are more likely to have LAS.[26] In daily life, 10 degrees of passive ankle dorsiflexion movement is required for walking and going down stairs, and 20-30 degrees of active ankle dorsiflexion movement is required for running and sprinting.[27] People with dorsiflexion limitation of motion change their biomechanics to compensate for the loss of ROM. Thus, they become more vulnerable to injury. Due to pain and inflammation after acute LAS, patients can only do 7-8° dorsiflexion movements.[28] After inversion injury, the ATFL applies an inferior, anterior directional traction force to the fibula. This leads to biomechanical changes in the proximal and distal tibiofibular joints, resulting in loss of dorsiflexion range of motion.[29] At the same time, in chronic LAS, ankle dorsiflexion ROM decreases due to limitations in posterior talus sliding movements.[30,31]

Strength

Both concentric and eccentric strength losses are seen in CAI.[32] The ability of the skeletal muscle surrounding the ankle to provide dynamic support during joint decelerations and perturbations is important for normal ankle function and injury prevention.[32] Following an acute LAS, isometric eversion strength is reduced when compared bilaterally for up to 3 weeks following injury.[33,34] However, inversion strength deficiencies have also been described in the first few weeks following a LAS, and therefore both inverter and evertor muscle strength may be equally important in the early stages of LAS rehabilitation.[35] During early recovery, strength deficiencies can be predicted to be associated with previous deficiencies present before the initial injury, pain-related muscle inhibition and swelling from the acute inflammatory process, or acute stretching of the surrounding musculature. However, the long-term consequences of a LAS, including decreased physical activity, decreased ankle ROM, and altered motion patterns, potentially result in recurrent strength deficits in a subset of developing patients.[36] For this reason, atrophy that may occur can be prevented by isometric muscle strengthening in the acute period of the injury. Concentric, eccentric and pilometric strengthening should be emphasized in the middle and late periods.[18]

Postural Control

Individuals with LAS injury have decreased neuromuscular control and proprioception sense compared to healthy controls.[18] As the reason for the decrease in postural control; Inhibition of spinal reflexes due to disruption of sensory pathways in the central nervous system can be demonstrated.[37] Although there are differences in the physiological definitions of neuromuscular control and proprioception, both can be improved with balance exercises. After LAS, the static balance of the patients can be evaluated by standing on one leg, and the dynamic balance can be evaluated with the Star Excursion Balance Test.[38] In the literature, it is stated that balance exercises reduce the incidence of ankle sprain.[39] In rehabilitation after LAS, balance exercises should have an important place in improving neuromuscular control and proprioception sense. Balance exercises should be progressed with different methods such as from static to dynamic, from simple to difficult, from single task to double task, from fixed floor to moving floor.[37-39]

Functional Activity

Loss of functional activity in CAI occurs during walking, running, jumping and cutting movements. In the acute and chronic period after LAS, an increasing inversion pattern is observed in the ankle before the first contact in walking.[40] In addition, patients experience a decrease in plantar flexion ROM and increase in knee flexion ROM. The reason for this is that they feel safe by putting their ankle in the closed position.[41] Thus, especially in the acute phase of the injury, patients walk with shorter stride length, shorter single leg support time and reduced maximum power.[42] In the literature, it has been shown that gait training given to patients after LAS injury improves the gait of patients.[43] There are changes in the biomechanics of jumping and landing in patients after LAS.[36] Acute LAS patients decrease the flexor moment on the injured side and increase the extensor moment on the healthy side while jumping. This creates an asymmetry between the two sides. The reason for this is that they slow down the body and exhibit a protective mechanism. Similarly, protective reactions continue in the descent position and patients use increased hip flexion to reduce ground reaction.[44] In chronic LAS, patients use increased inversion and knee flexion while descending extra.[45]

BIOPSYCHOSOCIAL MODEL OF LATERAL ANKLE SPRAIN

If the LAS injury is not treated, CAI develops. Detailed biopsychosocial model has been stated by Hertel et al.[46] Primary tissue injury occurs after LAS. There is an effect of biopsychosocial factors in both recovery and chronicity of LAS. The biopsychosocial model of LAS includes pathomechanical disorders, sensory-perception disorders, and motor-behavioral disorders, and there are personal and environmental factors that affect them. While personal factors affecting the course of injury after LAS are demographic information, past medical history, physical attitude and psychological state, environmental factors are physical activity, work-home demands, social support and access to health care.[46] Evaluation and treatment of patients after chronic LAS within the framework of a biopsychosocial model may allow patients to fully recover.[47] Table 1 shows the biopsychosocial model of LAS.

Table 1. Biopsychosocial model of LAS

Subgroups of the related impairments		
Pathomechanical	**Sensory-Perceptual**	**Motor-Behavioral**
Pathologic laxity	Pain	Altered reflexes
Arthrokinematic restrictions	Diminished somasosensation	Nöromuskuler inhibition
Osteokinematic restrictions	Perceived instability	Muscle weakness
Secondary tissue injury	Kinesiophobia	Balance deficit
Tissue adaptations	Lower self-reported funciton	Altered movements patterns
	Reduced quality of life	Reduced physical activity

REHABILITATION

LAS injuries are treated in 3 phases: acute, subacute and chronic.

Acute Phase

During this period, which covers the first 4 weeks, the patient has pain, edema, and limitation of movement. This period is important to prevent the disease from becoming chronic and to reduce functional losses. Our aim in this phase is to restore the ankle joint and soft tissue, to prevent muscle imbalance, to increase proprioceptive sensation and to provide normal ROM (Table 2).[18]

Therefore, POLICE (Protect, Optimal Loading, Ice, Compression, Elevation)[48] application should be started immediately after injury. Ice application can be applied to the area where the lateral ligaments are located for 12-15 minutes either as a fixed or as an ice massage every 2 hours. In Grade II and III injuries, brace, rigid taping and kinesiotaping can be used for immobilization if necessary.[18,49]

Due to the deterioration of the biomechanics of the distal tibiofibular joint and talocrural joint, limitations in ankle dorsiflexion movement may develop. Therefore, the anterior-posterior mobilization of the talocrural joint and the upward and posterior mobilization of the distal talofibular joint will increase the ankle dorsiflexion movement. This reason, mobilization exercises for ATFL injuries are important in the restoration of ROM. Green et al.[29] reported that the anterior and posterior mobilization of the talus increases painless dorsiflexion movement in the acute period ATFL sprain. In subacute and chronic period, it has been reported that there is a discernable treatment effect on ankle ROM and pain with early mobilization with movement.[13,50,51] When edema and pain disappear, Phase 2 is started.[52]

Table 2. Rehabilitation techniques in the acute period
POLICE
Active dorsiflexion-plantar flexion movements of the ankle
4-way isometric muscle strengthening to the muscles around the ankle
4-way isotonic strengthening of the muscles around the hip area
TENS, NMES
In-water proprioceptive applications
Posterior mobilization of the talocrural joint
Distal talofibular joint mobilization with movement
Cardiovascular training with stationary bike
Lumbopelvic region exercises

TENS: Transcutaneous electrical nerve stimulation, NMES: neuromuscular electrical stimulation

Subacute Phase

This period, when the patient has no or minimal pain and edema, covers 3-6 weeks. The subacute phase begins when the patient begins to tolerate weight bearing. Neuromuscular training and balance exercises are emphasized for the provision of impaired proprioceptive sense, muscle hypertrophy and proprioceptive sense. Mobilizations are continued to increase the ankle dorsiflexion ROM. Balance exercises should be given progression such as double-leg-single-leg, fixed surface-moving surface, eyes open-closed. Wobble board exercises and stretching exercises for the gastrosoleus muscle are performed on the incline board. Initially, the focus should be on dorsiflexion and plantar flexion. When the ligament healing is completed, first active movement and then resistant movements are started in inversion and eversion movements.

Elastic bandage is recommended for foot exercises. After single-planned movements, exercises should also be performed in the diagonal plane (inversion/eversion). During this period, clinicians should perform exercises aimed at sitting and correct walking biomechanics. When full ROM without pain is achieved, phase 3 is initiated when the muscle strength reaches 80-90% of the contralateral side (Table 3).[52]

Table 3. Rehabilitation techniques in the subacute period
Ankle resistant dorsiflexion-plantar flexion motion
Active eversion-inversion motion of the ankle
Resisted eversion-inversion movement of the ankle [If there is no pain in active movement, VAS<3 in resistance exercise (concentric first / then eccentric)]
4-way isotonic strengthening of the muscles around the hip area
NMES
Proprioceptive applications (Must be progressive)
Mobilization techniques for the talocrural and distal talofibular joint
Heel-raise
Cardiovascular training with stationary bike
Walking training
Balance exercises (must be progressed) Core stabilization exercises
NMES: Neuromuscular electrical stimulation

Chronic Phase

During this period when the patient's symptoms are not relieved, muscle strengthening, increasing proprioceptive, coordination, correction of walking and running biomechanics, and advanced exercises are included. It is continued until the 3rd month. After the 3rd month, sports-specific movements are started (Table 4).

Table 4. Rehabilitation techniques in the chronic period
Advanced proprioceptive exercises
Advanced balance exercises
Exercises for resistant (concentric/eccentric) lower extremity muscles
Plyometric exercises
Improvement of walking, running, jumping, cutting biomechanics
Aerobic exercises
Sport-specific movements
Core stabilization exercises

CURRENT CRITERIAS FOR RETURN TO PLAY AFTER LAS

Due to the high re-injury rate after rehabilitation, the time to return to sports and recreational activities should be well planned. There are no common criterias for returning to sports after LAS in the literature. Although there are different opinions for the criteria for returning to sports after LAS, the criteria in Table 4 as a result of the literature review can be used to decide on returning to sports. However, further studies are needed.

CONCLUSION

LAS, which is common in recreational and professional athletes, recurs and becomes chronic if left untreated. Since the number of mechanoreceptors it contains is high, LAS should not be considered only as a musculoskeletal disorder. A detailed clinical evaluation is recommended. Clinically, the patient presents with limitation of dorsiflexion ROM, muscle atrophy and unequal strength between the muscles, deterioration in walking, running and jumping, and proprioceptive losses. The patient should be approached from a biopsychosocial perspective both in the evaluation and treatment phases. The treatment of the patient is carried out in 3 phases and the transition between phases should be based on mentioned criterias. In the acute phase of rehabilitation, emphasis should be placed on eliminating pain and edema, and preventing loss of ROM and muscle strength. In the subacute and chronic stages, emphasis should be placed on increasing the proprioceptive sense and muscle strength, improving the biomechanics of daily life and sports-specific movements such as walking, running, jumping. If the patient is an athlete, return to sports should be planned after the criterias met.

ETHICAL DECLARATIONS

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Parameter	Evaluation criteria	Criterion
Pain/edema	VAS NPRS Foot and Ankle Index	Free of pain and swelling
ROM	Goniometric Weight bearing lunge test	Full ROM
Strength	Handheld dynamometry Standing jump	A return to normal strength 90% of uninjured limb strength
Static balance	Single-leg balance test Balance error system	Ability to balance without pain Maintain single-limb stance for 10 s
Dynamic balance	SEBT Y balance test	Compare with the uninjured limb 80% of uninjured limb reach Balance test with little to no pain
Running	Run program Running technique	Little to no pain while running Run at max speed without pain
Sports-specific movements	Lateral hopping Vertical hopping Shuttle run Sport-specific test Jumping and cutting Agility T test	80% of the uninjured leg amount Consistent T-test times between 8.9 and 13.5 s Speed and quality movement during sport-specific test
Patient-based reports	Foot and ankle ability measure Lower-extremity function scale Lower limb task questionnaire Injury-psychological readiness to return sport scale	Athlete feels confident The athlete is ready to return

VAS: Visual analog scale, NPRS: Numeric pain rating scale, SEBT: Star excursion balance test

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