Review

A NARRATIVE REVIEW ON TENNIS-RELATED UPPER LIMB PATHOLOGIES

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ABSTRACT

Tennis players are frequently affected by musculoskeletal injuries due to the repetitive nature of the sport, improper technique, and physical stress. The shoulder is one of the most commonly injured areas, particularly in non-professional players, where rotator cuff tendinopathy, impingement, and labral tears are prevalent. These injuries are often linked to faulty stroke mechanics, especially in overhead shots. The elbow is another critical area, with conditions such as tennis elbow (lateral epicondylitis) affecting non-professional players aged 30 to 50, primarily due to poor backhand technique and incorrect racket grip. This condition arises from microtrauma to the extensor carpi radialis brevis and extensor digitorum tendons. In contrast, epitrochleitis (medial epicondylitis) occurs more commonly in professional players and results from stress on the forearm flexors during forehand strokes and serves. Pathologies of the wrist and hand, including tenosynovitis of the extensor carpi ulnaris, De Quervain's disease, and triangular fibrocartilage complex (TFCC) injuries, are also prevalent, often caused by excessive wrist rotation, improper grip, and overuse of topspin strokes. De Quervain's disease affects the tendons at the radial styloid and is characterized by swelling and restricted movement of the thumb and wrist. TFCC injuries associated with wrist rotation are often caused by combined impacts and loading. Additionally, fractures of the hook of the hamate, though rare, are seen in players who relax their grip during powerful strokes. Early diagnosis, based on clinical tests such as Cozen's, Maudsley's, and Finkelstein's tests, as well as imaging modalities like ultrasound and MRI, is crucial. Conservative treatments, including rest, physical therapy, anti-inflammatory medications, and technique adjustments, are effective in most cases. Surgical intervention is rarely necessary but may be required for persistent or severe injuries. Preventative strategies, such as using proper equipment, and technique, and maintaining physical conditioning, are key to reducing the risk of common tennis injuries.

KEYWORDS: *tennis elbow, upper limb, tennis injuries, tendon, muscle*

INTRODUCTION

Traumatology of the locomotor system in tennis

In Italy, tennis has always been a widely practiced sport. According to estimates by CONI and ISTAT, it ranked second in 2022 in terms of the number of registered players, with growing participation, and reached the top position in regions such as Piedmont and Sicily (1). With over 1 million registered players and approximately 3 million recreational

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and casual players, tennis is one of the most popular sports in Italy, followed by more than 24 million sports enthusiasts. The number of senior players is also steadily increasing (2).

This growing interest in tennis underscores the importance of addressing the impact of tennis-related injuries in clinical practice for physicians and athletic trainers. The incidence of acute traumatic events in tennis is low, accounting for about 0.12% of all injuries. However, the incidence of conditions related to functional overuse is significantly higher (3).

For all age groups, the most common injuries are those related to tendons or muscles. In players over the age of 25, overuse injuries are the most prevalent. Among younger players, lower limb injuries are twice as common as upper limb and spinal injuries, with a high incidence of ankle sprains (4). Although the number of female players is increasing, there are no substantial gender-based differences in injury incidence, particularly in players over 25. For adolescent athletes, however, statistics indicate a 0.6% incidence per 1,000 hours of play among females, with a prevalence of patellar conditions and lower back pain. In contrast, males show an incidence of 1.7% per 1,000 hours, with a marked prevalence of contusions, abrasions, lacerations, ankle injuries, and lumbar spine pain (5).

Despite being an asymmetrical sport, tennis is a relatively comprehensive activity that features a variety of athletic movements that engage the entire musculoskeletal system (6). Tennis-related injuries include both acute traumatic injuries specific to the sport and more general injuries common to other sports.

Acute injuries caused by trauma that exceeds the mechanical resistance of anatomical structures are relatively rare. Tennis players are often exposed to forces they generate endogenously rather than exogenous forces, as there is no physical contact with opponents, and the game equipment poses minimal risk (7).

Chronic injuries, on the other hand, are linked to functional overuse and can be influenced by both predisposing and determining factors:

- predisposing factors can be exogenous, such as the playing surface, footwear, equipment, or environmental conditions, or endogenous, related to the athlete's congenital or acquired abnormalities, such as asymmetries or myotendinous imbalances;
- determining factors are represented by repeated and abnormal functional stresses due to specific athletic gestures (8).

Tennis involves rapid player movements on the court, including sudden changes of direction, sprints, jumps, stops, and dives, which exert considerable stress not only on the spine but also on the upper and lower limbs. Additionally, the specific movements of the dominant upper limb, particularly during serves and smashes (overhead movements), involve significant exertion. If not performed correctly with proper technique, these movements can cause injuries that compromise not only athletic performance but also daily life activities (9). Such injuries, especially those due to poor execution of movements, initially manifest as reactive inflammatory phenomena, followed by regressive and degenerative changes (2).

The following sections describe the most common trauma-based conditions affecting tennis players, with a focus on the upper limb. Emphasis will be placed on the causes, clinical manifestations, diagnostic possibilities, treatment methods, and, most importantly, prevention strategies.

Traumatology of the upper limb in tennis

All sports are influenced by external factors that alter the dynamics of joint forces. Surfaces and shoes play a crucial role in determining directional changes, biomechanical responses to sprints, and landing after jumps. In tennis, the racket is the medium for transmitting forces between the body and the ball. The impact between the racket and the ball generates translational, rotational, and elastic forces in the upper limb, creating a specific type of stress in this part of the body.

Over the past forty years, tennis rackets have undergone a significant evolution in manufacturing, radically changing the way the game is played. The transition from wood to graphite enabled the creation of more efficient rackets, which are lighter, more maneuverable, and allow for increased shot speed, thereby fostering a style of play that emphasizes players' physical power. However, this has also led to greater stress on the upper limb joints (6).

New materials have also enabled an increase in racket size, making the sport more accessible to less experienced athletes who are consequently less skilled in their movements. The structure of modern rackets is characterized by greater stiffness compared to the wooden ones previously used; this ensures better performance in terms of ball rebound speed but also increases the vibrations transmitted to the upper limb (7).

In fact, the vibration frequency has shifted from 90 Hz with wooden rackets to 166–200 Hz with modern ones. The forces transmitted from the ball to the body depend not only on the materials but are also closely linked to stroke technique.

Within the racket head's ellipse, three specific "sweet spots" can be identified, each with distinct mechanical properties (8) (Fig. 1):

1. the center of percussion, which, when struck, neutralizes rotational and translational forces, reducing the stress transmitted to the grip;
2. the point of maximum rebound velocity, which imparts the highest ball speed;
3. the point of the first harmonic oscillation, which minimizes vibration transmission to the grip.

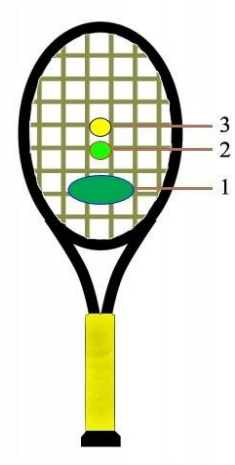

Fig. 1. *The sweet spots of the racket.*

An experienced player, thanks to their acquired sensitivity, can identify the racket area closest to these three points and use it for the majority of their strokes. Conversely, inexperienced players tend to strike random points farther from the sweet spots, generating significantly more stress on the upper limb, up to three times as much. Studies have shown that in terms of vibrations absorbed by the athlete, experienced players absorb approximately 80%, while beginners absorb about 93%. A high grip level on the handle provides greater comfort and security during strokes but also increases the transmission of vibrations. It is, therefore, preferable to choose lower grip levels, especially since these do not influence the ball's rebound speed or shot velocity. It has been observed that experienced players exert maximum grip force on the handle moments before the ball contacts the racket, then slightly reduce it during the strike. This is likely an unconscious protective mechanism against vibrations. However, this adjustment does not occur in beginners (8).

Repetitive rotational, translational, and vibratory forces create mechanical stress on the player's upper limb, increasing the likelihood of developing certain pathologies. This risk is particularly high for inexperienced athletes who, due to incorrect technique, significantly amplify the forces involved.

THE SHOULDER

In 1976, Priest and Nagel introduced the term “Tennis Shoulder,” highlighting specific morphological and functional alterations in the shoulder caused by the repetitive and sudden demands placed on the dominant upper limb in tennis players (9). According to these authors, increased muscle mass and elongation of suspensory muscles lead to a lowering of the dominant shoulder, resulting in an apparent elongation of the limb. This induces a compensatory scoliotic posture of the spine, promoting the onset of subacromial impingement syndrome. Additionally, tennis movements can cause posterior capsular stiffness, resulting in a deficit in internal rotation and an increase in external rotation of the dominant arm compared to the contralateral side.

Scientific interest in the tennis player’s shoulder and the entire upper limb is justified by biomechanical studies showing the complex muscle activity involved during tennis-specific movements (10). The serve, similar to a throwing motion, can be divided into four phases: preparation, loading, acceleration, and deceleration. The entire execution takes approximately 1.56 seconds, with the acceleration phase being the fastest. In contrast, the loading phase recruits the most muscle activity, involving the deltoid, biceps brachii, triceps brachii, supraspinatus, infraspinatus, and latissimus dorsi (11). During the loading phase, the shoulder experiences the highest muscular workload, with a compression force equivalent to approximately 80% of body weight, ensuring humeral centering. In the acceleration phase, primarily driven

by the pectoralis major, latissimus dorsi, and subscapularis, the angular velocity of the arm can exceed 2500°/sec, comparable to a washing machine during a spin cycle (10).

These characteristics make shoulder injuries the most common upper limb injuries in tennis players, from young athletes to all levels of competition (12). Such rapid and repeated forces, especially without a well-structured training program, lead to structural and functional alterations in the musculoskeletal and articular structures, including subacromial impingement syndrome, rotator cuff tears, glenoid labrum lesions, acromioclavicular microtrauma, long head of biceps tendon injuries, and shoulder instability.

Subacromial impingement syndrome

Impingement refers to the increased friction of soft tissues resulting from a discrepancy between the space and the structures it contains (12). In this case, the space "défilé" is defined superiorly by the acromion and coracoacromial ligament, and inferiorly by the humeral head and glenoid; the contents are the rotator cuff and subdeltoid bursa, which slide between these structures. Increased friction occurs when the space narrows or the contents expand. Subacromial impingement syndrome is rarely observed in individuals under 40; however, repetitive overhead movements, such as those involved in tennis, predispose athletes to its development (13).

Friction forces cause pathophysiological changes in the subacromial space, classified into three evolutionary stages by Neer (1972; 1983):

1. **Stage 1**: edema and microhemorrhages in the subacromial bursa and rotator cuff tendons, generally found in athletes under 25 years old;
2. **Stage 2**: development of fibrosis and scarring in the subacromial bursa, tendinitis predominantly affecting the supraspinatus and long head of the biceps, typically observed in athletes over 25 years old;
3. **Stage 3**: bone changes and tendon degeneration, potentially progressing to tendon rupture, frequently seen in individuals over 40 years old (Neer, 1983).

Shoulder impingement is classified into two main categories: internal and external. Internal impingement involves the rotator cuff structures and the glenohumeral joint, often due to skeletal abnormalities, extreme repetitive movements, or instability that alters biomechanics and causes excessive contact even at moderate joint angles (14). External impingement arises from excessive friction between the rotator cuff and the inferior margin of the acromion or the coracoacromial ligament, often due to skeletal deformities or inadequate centering of the humeral head on the glenoid (15).

In the early stages, subacromial impingement presents as pain that appears after sports activity, often at night, and disappears with rest. In more advanced stages, pain becomes persistent, worsening with movement and at night. Diagnosis is aided by clinical tests such as Neer, Hawkins, and Yocum tests, which are based on compressing the cuff between the humeral trochite and the acromial roof (12). Radiological imaging, such as standard radiographs, is essential for detecting osseous abnormalities, calcifications, or subacromial space narrowing (16).

Conservative treatment involves functional rest, analgesics, anti-inflammatory medication, and personalized rehabilitation protocols focusing on restoring normal shoulder biomechanics and strengthening the internal and external rotators (17). Approximately 60% of cases improve with conservative management. For cases unresponsive to conservative treatment or presenting with advanced-stage pathology, surgical intervention is recommended in about 30% of cases (18).

Internal impingement

Internal impingement occurs when extreme shoulder movements, characteristic of specific overhead sports gestures, result in repeated and unnatural contact between the internal surface of the rotator cuff and the glenoid joint. This condition affects athletes under 40 years old engaged in overhead sports and is characterized by vague but progressively worsening symptoms. Athletes often report a gradual loss of speed and control during athletic movements, described in the 1980s as "dead arm syndrome", typically associated with glenoid labral lesions (9, 10).

The condition gained attention through arthroscopic studies by Walch (1992) and cadaveric studies by Jobe (1993), which highlighted the interaction between the rotator cuff and the articular margin of the glenohumeral joint. During hyperabduction and external rotation, the articular margin of the cuff and posterior capsule are compressed against the joint, potentially pinched between the humerus and glenoid, causing mechanical injury to the labrum. Similarly, hyperadduction and internal rotation produce a comparable mechanism involving the anterior portion of the cuff, leading to posterosuperior impingement (PSI) in the former case and anterosuperior impingement (ASI) in the latter.

Imaging and cadaveric studies have shown that contact between the rotator cuff and the joint in these positions is physiological; however, the repetitive nature of overhead athletic movements, as seen in tennis, baseball, or volleyball,

leads to overstress on the cuff, capsule, and glenoid labrum, eventually resulting in a pathological condition (11-21). Recurrent mechanical insults induce a posterior capsular contracture, initially involving the inferior glenohumeral ligament and posteroinferior capsule, clinically manifesting as a deficit in internal rotation (glenohumeral internal rotation deficit, GIRD) (14). This shifts the articular contact point posteriorly, allowing a greater range of external rotation, increasing stress on the long head of the biceps tendon, and predisposing the shoulder to SLAP lesions (22).

The classical presentation includes partial rotator cuff tears on the articular side of the supraspinatus and glenoid labrum tears in the posterior or posterosuperior region. The rarer anterosuperior internal impingement is based on similar mechanical concepts but involves reversed movements, affecting the anterosuperior structures of the cuff and capsule. In adduction and internal rotation, the subscapularis, pulley, and long head of the biceps tendon come into contact with the glenohumeral joint, leading to partial cuff tears in the anterior region and potential subluxation of the biceps tendon due to pulley damage (2).

Symptoms are typically diffuse, with posterior shoulder pain localized near the joint line. Pain may also be felt anteriorly in the coracoid region and may be associated with symptoms of concurrent lesions, such as cuff, labral, or biceps tendon injuries. Clinical examination often reveals a discrepancy of at least 30°-40° between internal or external rotation compared to the non-dominant arm, depending on whether the impingement is primarily anterior or posterior. Specific tests for associated lesions may be positive, but subacromial impingement tests are usually negative (12). Scapulothoracic dyskinesia, often present in this pathology, should also be investigated; it manifests as an asymmetry relative to the contralateral scapula and increased prominence of the scapular inferior margin (13).

Conservative treatment involves an initial phase of absolute rest from overhead activities, potentially combined with anti-inflammatory medications. In the second phase, muscle strength, flexibility, neuromuscular proprioceptive control, and scapulothoracic biomechanics should be restored and reinforced. Arthroscopic surgical intervention is considered only after the failure of conservative treatment (14). Return to sport is permitted only after complete resolution of pain and satisfactory muscle recovery (15).

Rotator cuff injuries

The rotator cuff is a musculotendinous structure responsible for centering the humeral head in the glenoid cavity during shoulder movements. It consists of four muscles: the supraspinatus, infraspinatus, subscapularis, and teres minor, whose tendons almost entirely encase the humeral head, forming a sort of "cuff" (1-3).

Rotator cuff tears may result from acute trauma or progressive tendon degeneration, eventually leading to rupture. An acute injury may occur due to a sudden movement involving abduction and external rotation (such as a poorly executed smash or backhand) or a fall on the shoulder, elbow, or extended limb. This trauma is typically followed by sharp, intense pain accompanied by a tearing sensation, significant functional impairment initially, and gradually reduced mobility. It primarily limits abduction and external rotation, particularly when resistance is applied (4, 5).

Degenerative tears are commonly observed in athletes over 50, typically affecting an anatomically hypovascular region of the supraspinatus tendon, located about 1 cm from its insertion on the humerus (6-8). Passive abduction evokes pain between 70° and 110° due to the injured area passing under the acromiocoracoid arch. Specific tests reveal pain during targeted muscle activation, and a strength deficit may also be present (9, 10). Imaging studies such as ultrasound, MRI, and arthrography are valuable for diagnosis (2, 11).

Complete tears are often preceded by tendinous degeneration, which can be classified using Zlatkin's MRI-based grading system (12, 13):

- **stage 0**: normal morphology and signal intensity;
- **stage 1**: increased signal intensity without thinning, irregularity, or discontinuity;
- **stage 2**: increased signal intensity with tendon thinning or irregularity;
- **stage 3**: supraspinatus tendon rupture.

Once confirmed, tears are categorized using various schemes considering location, partial or full-thickness nature, shape, size, affected tendon(s), muscle trophism, the state of the long head of the biceps, and associated ligamentous injuries (14-16).

Initial treatment is conservative, involving functional rest, cryotherapy, and possibly anti-inflammatory medications. After the acute phase, functional and motor recovery programs begin (17-19). Surgical intervention targets the cause of the rupture: acute traumatic tears in young or athletic individuals warrant immediate repair. In contrast, degenerative tears often stem from subacromial impingement, necessitating a subacromial decompression procedure based on symptoms and functional limitations (20-22). Notably, as Codman proposed in 1934 and Fukuda confirmed histologically in 1994, cuff tears are progressive and do not heal spontaneously; only symptoms may regress (23-25).

Glenoid labrum injuries

The Glenoid labrum is a fibrocartilaginous structure encircling the articular surface of the glenoid cavity, aiding in the centering of the humeral head within the glenoid concavity. A labral tear decreases shoulder stability, causes pain (due to nociceptive fibers within the labrum and at the insertion of the long head of the biceps tendon), and may produce mechanical sensations such as clicks, intra-articular noises, or functional limitations (26, 27).

Sports like tennis and other overhead disciplines impose repeated stress on the labrum, structurally weakening it and predisposing it to injury (28, 29). Depending on the location, these injuries are classified into different categories: considering the glenoid from a lateral perspective, it can be visualized as a clock. Tears between 9 and 3 (the superior margin) are classified as SLAP lesions involving the superior anterior and posterior labrum. Tears in the inferior portion, from 3 to 9, are called Bankart lesions (30, 31).

SLAP lesions are most frequently observed in tennis players. The acronym SLAP was introduced in 1985 by Andrews and later developed by Snyder in the 1990s; these injuries are closely related to anatomy. The long head of the biceps tendon inserts at the superior glenoid margin, where part of its fibers connect to the apical region of the labrum. Violent trauma or repetitive actions may lead to tears in this area due to reflexive transmission of biceps contractions to the superior labrum. During the deceleration phase (after hitting the ball during a serve or volley), the biceps undergo eccentric contraction to prevent elbow hyperextension, transmitting force to the labrum. Additionally, the labrum's superior portion may be torn during abrupt abduction and external rotation in the loading phase, during which the biceps tendon is pulled posteriorly (32-36).

Diagnostic clinical tests simulate the traumatic mechanism and reproduce pain (37):

- **modified Dynamic Labral Shear test (mDLS)**: elbow at 90°, shoulder abducted beyond 120° and maximally externally rotated. The examiner abducts the arm in the horizontal plane, exerting shear force on the joint, then adducts the arm from 120° to 60°. The test is positive if pain is elicited, particularly in the posterior region;
- **O'Brien test**: elbow extended, shoulder elevated anteriorly to 90° and adducted, with the arm fully internally rotated. The examiner applies downward pressure while the patient resists. The test is positive if pain or intra-articular noises are noted. It may also be positive for acromioclavicular joint issues;
- **Biceps Load test II**: the patient is supine with the arm abducted to 120°, externally rotated, elbow flexed at 90°, and forearm supinated. The patient flexes the elbow against the examiner's resistance. The test is positive if elbow flexion exacerbates pain;
- **Passive Distraction test (PDT)**: the patient is supine with the shoulder at the table's edge, the arm elevated to 150°, the elbow extended, and the forearm supinated. The examiner pronates the forearm. The test is positive if pain occurs in the anterior or posterior shoulder region.

Imaging studies, including MRI or contrast-enhanced CT, are about 90% sensitive and specific, but the gold standard for diagnosis and treatment remains arthroscopic surgery (38, 39).

Tendinopathies of the Biceps Brachii

The long head of the biceps brachii (LHB) is an intra-articular tendon that passes through the bicipital groove of the humerus and inserts on the supraglenoid tubercle of the scapula. Over the years, its function has been significantly reconsidered: it was previously thought to play an important role in actively stabilizing the glenohumeral joint by compressing the humeral head against the glenoid, thereby preventing upward and anterior displacement. However, studies, including cadaveric analyses, have shown that tenotomy of the LHB does not cause upward displacement of the humeral head, suggesting that the LHB likely serves as a passive stabilizer of the joint (9, 10).

LHB pathologies can be categorized into three types: inflammatory, degenerative, and instability. All three commonly present with diffuse shoulder pain. Since the LHB sheath communicates with the joint space and is connected to the rotator cuff, inflammatory changes in these structures often affect the LHB, which may exhibit early symptomatic signals (4). Tendinous degenerative changes can be secondary to subacromial impingement or primary in origin, presenting as tendon thickening, surface irregularities, and scar-like lesions (Neer, as cited in (15). Contrary to earlier beliefs linking degeneration with aging, cadaveric studies have found a low prevalence of tendinopathy in older anatomical specimens, while overhead athletes demonstrate higher rates of LHB tendinopathy due to anterior subluxation of the humeral head during the loading phase of a stroke (5).

Instability of the LHB arises from laxity or rupture of the pulley system that holds the tendon within the bicipital groove. This can lead to medial displacement during contraction, especially in abducted and externally rotated positions.

Although rare in isolation, LHB instability is more frequently associated with rotator cuff injuries and is often observed in overhead athletes (3).

Clinically, pain is exacerbated by both external and internal rotation and by palpation of the bicipital groove. The Yergason and Speed tests are sensitive but lack specificity, while instability may be detected through a palpable click during intra-to-extra rotation with the shoulder abducted to 90° (12). Imaging modalities such as radiography help exclude other conditions, while ultrasound and MRI provide more detailed assessments of the tendon.

Treatment typically begins with conservative management, focusing on functional rest, pain control, and maintaining passive shoulder mobility. Gradual progression includes strengthening the rotator cuff and periscapular muscles, ultimately advancing to sport-specific exercises (14). Surgical intervention, such as arthroscopic debridement, decompression, or tenotomy, may be required if symptoms persist despite conservative treatment (24).

Acromioclavicular joint micro traumatic arthropathy

This degenerative condition affects the acromioclavicular joint and results from repeated microtrauma that damages cartilage and subchondral bone. The acromioclavicular joint, along with the sternoclavicular joint, connects the arm to the trunk and serves as a fulcrum for scapular movement during deceleration in serves and acceleration in backhand strokes (6, 7) (Fig. 2).

Fig. 2. *Backhand stroke.*

Symptoms include activity-related pain, initially sporadic but becoming more frequent over time, tenderness upon palpation of the joint, and pain during forced horizontal abduction with posterior shoulder displacement and internal rotation of the arm. Radiographic findings may reveal bone changes, such as subchondral cysts, typically on the acromial side, without joint space narrowing (4).

Conservative treatment is preferred, but in some cases, resection of the lateral clavicle's end may be necessary surgically (17).

Shoulder instability

Shoulder instability is classified based on pathogenic mechanisms and anatomopathological criteria into three categories: TUBS (Traumatic Unilateral Bankart Surgery), AMBRI (Atraumatic Multidirectional Bilateral Rehabilitation Inferior capsular shift), and AIOS (Acquired Instability Overstressed Surgery).

The first group includes acute traumas that cause damage to the labrum, capsuloligamentous complex, and possibly the bone. The second group includes instabilities without evident traumatic events, likely associated with the constitutional laxity of stabilizing structures. The third group (AIOS) involves microinstabilities resulting from repetitive microtraumas associated with overhead sports, leading to supraequatorial labral lesions, damage to the long head of the biceps (LHB) insertion, and rotator cuff injuries (1, 27).

An unstable shoulder requires assessment of anterior-posterior laxity compared to the contralateral shoulder, sulcus sign for longitudinal laxity, and anterior and posterior apprehension tests to evoke dislocating movements, triggering involuntary protective muscle contractions (21) (Fig. 3, 4). Signs and symptoms of secondary lesions must also

be evaluated. A standard radiographic exam is essential, complemented by MR arthrography or CT arthrography where necessary (22).

Treatment involves at least six months of conservative management before considering surgery. Arthroscopic intervention is preferred, involving multiple capsular plications, SLAP repair, debridement, and repair of any rotator cuff injuries (25).

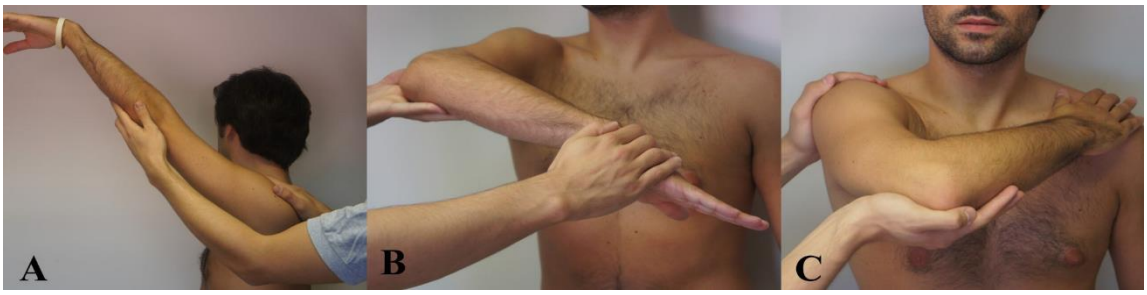

Fig. 3. ***A****): Neer test: the examiner performs passive arm elevation while maintaining scapular depression;* ***B****): Hawkins test: the shoulder and elbow are flexed at 90°, followed by internal rotation leveraging the elbow and dorsal wrist;* ***C****): the patient places the hand of the affected arm on the opposite shoulder, followed by passive elbow elevation and resistance testing.*

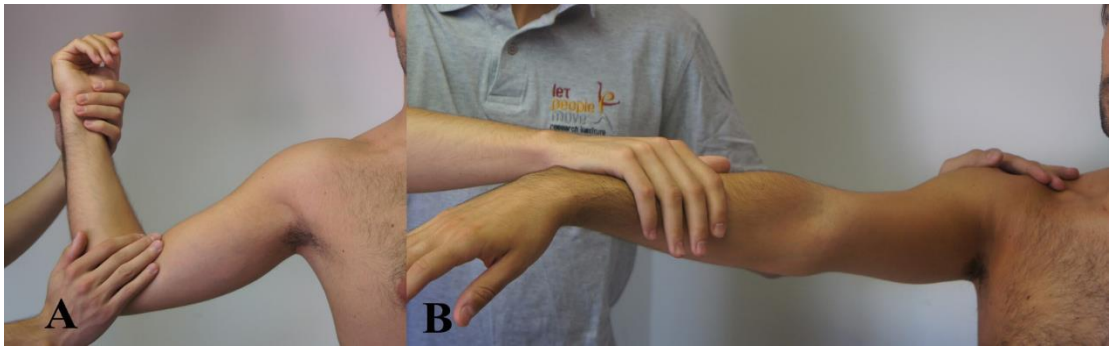

Fig. 3. ***A****): Modified Dynamic Labral Shear test (mDLS);* ***B****): O'Brien's test.*

THE ELBOW

The elbow is among the most affected body regions in microtraumatic pathologies of tennis players, to the extent that the term "tennis elbow" was coined to describe the insertional pathology of tendons on the radial condyle (31, 26).

"Tennis Elbow": epicondylitis in non-professional tennis players

This condition primarily affects tennis players aged 30 to 50, with an incidence of 50% among non-professional players who play more than three times a week (27, 2). It is mainly caused by technical errors, insufficient physical preparation, poor coordination, or an inadequate grip on the racket (30, 3).

The primary movements that favor epicondylitis are characteristic of non-professional players, particularly during incorrect backhand strokes. While professional or experienced players combine proper elbow flexion and trunk rotation with wrist hyperextension to mitigate impact force during the backhand, inexperienced players prematurely rotate their trunk, lead with their elbow, and flex their wrist (on average 13°) during impact (28;7). This results in eccentric contraction of the epicondylar muscles before the stroke and subsequent concentric contraction, causing excessive strain and tension on tendinous structures and muscle bellies (5).

A heavy racket, an inadequately short grip, deflated or heavy balls, excessive string tension, and playing on fast surfaces exacerbate the condition, contributing to the pathology's development (6, 8).

The pathophysiology is linked to the overuse of microlesions of the extensor carpi radialis brevis and sometimes the common extensor digitorum tendon. Chronic inflammation leads to the formation of granulation tissue and adhesions, resulting in painful symptoms when the tendons are under tension (26, 31).

Clinical examination reveals localized tension and pain on palpation of the humeral condyle or during certain diagnostic movements, such as:

- **Cozen's test**: pain during resisted extension of the wrist and fingers with the elbow flexed;

- **Maudsley's test**: pain during resisted extension of the middle finger (Fig. 5);
- **Mills' maneuver**: pain during forced pronation with the wrist flexed and the elbow extended;
- **Solveborn's test**: pain while lifting a chair by its backrest with the elbow extended, forearm pronated, and wrist dorsiflexed.

Radiographic imaging may reveal small calcified zones associated with chronic inflammation or osteophyte formation on the epicondyle (29). In rare cases, fractures may also occur. Ultrasound can demonstrate degenerative tendon thickening, vascular proliferation, and mucoid degeneration (38).

Pain occurs during sports activities and rarely persists at night (1). Before establishing a definitive diagnosis, it is necessary to exclude articular pathologies of the elbow and radicular pathologies of the radial nerve (32). Treatment is primarily conservative, including rest, ice, deep massage, physical therapy with TENS, ultrasound, and orthoses such as dynamic braces (31). After the acute phase, sport-specific training sessions, including stretching of the flexor and extensor muscles, are essential (2).

Surgical treatment is rarely necessary and is indicated only after failure of a conservative program lasting at least six months. Surgical techniques involve releasing the extensor carpi radialis brevis from the lateral condyle and removing degenerated tissue. This procedure can also be performed arthroscopically. Return to sport typically occurs 4–6 months post-surgery (26, 29).

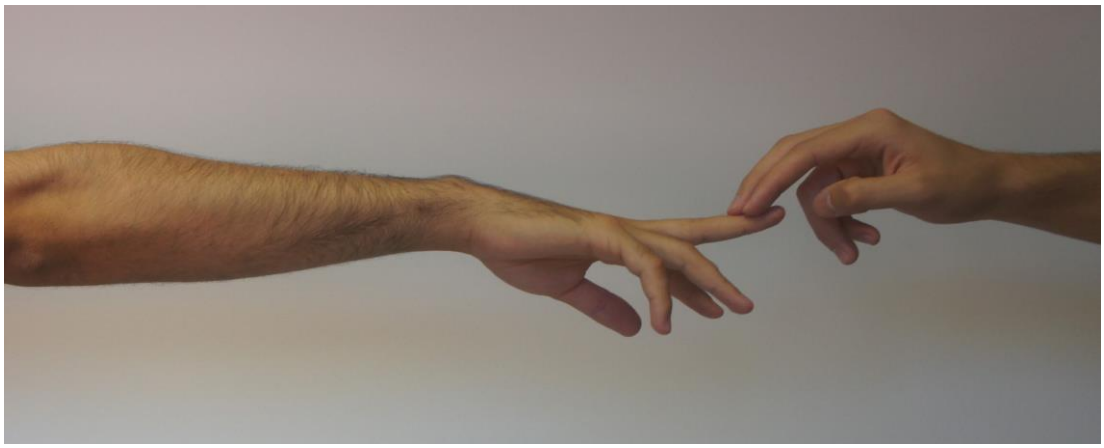

Fig. 5. *Maudsley's test: pain during resisted extension of the middle finger.*

Epitrochleitis: tennis elbow in professional players

Also referred to as "golfer's elbow" or "professional tennis elbow," this condition is associated with forehand strokes rather than backhands, even when executed with proper technique. It is caused by microtrauma to the finger flexor tendons and forearm pronators, which are heavily strained during serves, smashes, and forehand strokes (4, 5).

Typical degenerative anatomopathological alterations are observed in the pronator teres, flexor carpi radialis, and sometimes the palmaris longus, flexor digitorum superficialis, and flexor carpi ulnaris (27, 32).

Differential diagnosis is performed through clinical, radiographic, and electromyographic examinations to distinguish it from articular lesions such as the medial collateral ligament of the elbow, ulnar nerve entrapment, and medial elbow instability (30, 29).

Clinically, pain is associated with palpation of the medial elbow, specifically in the trochlear region, and exacerbated by resisted wrist flexion or forearm pronation (26, 32). Radiography may reveal small calcified zones associated with chronic inflammation, osteophyte formation in the humeral trochlea, or fractures (6, 29).

Treatment is similar to that for epicondylitis and includes rest, ice, orthoses, correction of equipment, and proper training with stretching of the flexor muscles (3, 5). Surgical treatment is rare and should only be considered after the failure of at least three months of conservative therapy. However, arthroscopic treatment is not recommended due to the proximity of the ulnar nerve and medial collateral ligament (27).

THE WRIST AND HAND

Wrist and hand pathologies in tennis are common, as these areas are the link between the body, where force is generated, and the racket, through which this force is transmitted to the ball. Similar to other anatomical regions, the main conditions involve overuse, microtrauma, and movement during strokes, although direct and acute injuries are also

possible. These can be caused by excessive wrist rotations during off-center ball impacts on the racket, as well as subsequent counter-shocks (2, 5).

Frequent stress on the wrist and hand is often exacerbated by the overuse of topspin strokes, excessive wrist movement, incorrect grip, and unsuitable string tension (6, 7). Among the most common pathologies related to the functional overload of tendon and muscle structures are tenosynovitis and tenovaginitis, which are chronic, painful inflammations of the sheaths that cover the tendons. These issues are more prevalent in players over 30, with a higher occurrence in women (1).

Tenosynovitis of the extensor carpi ulnaris

The most common wrist issue among tennis players affects the ulnar side and is often attributable to tenosynovitis of the extensor carpi ulnaris (ECU) muscle. It is characterized by the gradual onset of pain during play, especially in top-spin-based games, due to the rapid wrist pronation required for effective strokes. Pain symptoms tend to worsen progressively but improve with rest (33). Swelling in the posteromedial region may sometimes appear, with palpation exacerbating the pain. The clinical examination revealed that resisted ulnar extension of the hand elicits pain. Ultrasound imaging can confirm tenosynovitis, while X-rays rule out stress fractures (38).

Treatment is primarily conservative, involving rest, ice, non-steroidal anti-inflammatory drugs (NSAIDs), stretching, iontophoresis, and ultrasound therapy for the extensor carpi ulnaris (ECU). In some cases, surgical intervention, such as tenolysis or the removal of degenerated tissue, is necessary. In severe cases, tendon instability may require reconstruction of the sixth extensor compartment (34, 35).

Tenosynovitis of the finger extensors and the long extensor of the thumb

Tenosynovitis of the finger extensors and the long extensor of the thumb is less common among tennis players than among instructors. These conditions are associated with the repetitive use of "wrist shots," typically slower and more frequent balls used in teaching beginners (5). Pain is localized to the dorsal mid-third of the forearm for finger extensors and to the anatomical snuffbox for the long extensor of the thumb. Pain is triggered by local palpation and resisted extension of the fingers and thumb. Diagnosis is primarily clinical and anamnesis-based, supported by ultrasound and X-rays to exclude fractures (38).

Conservative treatment includes rest, ice, NSAIDs, and, if necessary, immobilization using a splint. Physical therapy, including iontophoresis and ultrasound, may also be employed, followed by proper physiokinesitherapy after the acute pain phase subsides (34).

De Quervain's disease

De Quervain’s disease is one of the most frequent tendon problems among tennis players. It is caused by irritation of the extensor pollicis brevis and abductor pollicis longus tendons as they pass through the osteotendinous pulley of the radial styloid (5). Chronic inflammation of the tendon sheath leads to swelling that hampers tendon gliding, creating a vicious cycle of increased irritation.

The condition arises from microtrauma and repetitive movements of thumb flexion and extension. Pain, typically radial-side wrist pain, has a progressive onset and may radiate to the forearm. Swelling at the thumb-wrist junction and firm-elastic formations may be evident. Movements of the thumb and wrist are limited by pain, and an audible clicking sensation may occur with thumb extension. Finkelstein's test is consistently positive (2, 33) (Fig. 6).

Fig. 6. *Finkelstein’s test: severe tenderness and pain on the radial aspect of the wrist is caused when the thumb is flexed into the palm and the wrist is ulnar deviated.*

Initial treatment is conservative, involving rest, ice, NSAIDs, and thumb splints. Infiltration therapy may be added. Conservative management resolves symptoms in 80% of cases, restoring functionality without residual effects. Surgical pulley release may be required for persistent or recurrent symptoms. Return to sport after surgery usually occurs around 8 weeks (35).

Triangular fibrocartilage complex (TFCC) injury

The TFCC comprises structures between the ulnar head and the ulnar carpus, including:

- fibrocartilage proper, with limited reparative ability due to poor vascularization;
- radioulnar ligaments, stabilizing the distal forearm during pronation and supination;
- ulnocarpal ligaments, reinforcing the anterior carpus;
- ECU tendon sheath, strengthening the dorsal carpus and absorbing approximately 20% of impact forces transmitted from the carpus to the forearm (36).

Tennis's combination of wrist impacts and rotation predisposes players to TFCC lesions. Clinically, pain occurs at the ulnar styloid, worsening with ulnocarpal loading. X-rays exclude carpal fractures, while MRI helps confirm the diagnosis (38). Conservative management involves immobilization, rest, and NSAIDs, but surgical repair, often arthroscopic, may be needed for persistent symptoms or central fibrocartilage injuries (37).

Fracture of the hook of the hamate

Fractures of the hamate hook, though rare, are typical tennis injuries. They may result from a relaxed racket grip during powerful responses or from centrifugal force overpowering the grip. These fractures are classified as stress or acute injuries based on patient history (39).

Pain, which renders racket use nearly impossible, is exacerbated by resisted abduction/adduction of the fifth finger and palpation of the ulnar carpal side. Standard X-rays often miss this injury, requiring specialized carpal tunnel projections or CT scans for confirmation. Initial treatment includes immobilization with a cast including the fifth finger, but high rates of pseudoarthrosis necessitate surgical removal of the fragment in many cases (39).

DISCUSSION

Upper limb injuries in tennis are predominantly caused by repetitive and high-intensity movements, particularly those involving the shoulder, elbow, wrist, and hand. Overhead strokes, such as serves and smashes, impose substantial mechanical stress on the rotator cuff, biceps tendon, and glenoid labrum, frequently leading to impingement syndromes, tendinopathies, and joint instability. Additionally, chronic conditions such as lateral and medial epicondylitis are prevalent, often attributed to excessive load, inadequate technique, and improper equipment.

Technological advancements in racket design have significantly influenced the biomechanics of tennis. While modern rackets enhance power and maneuverability, they also increase vibratory forces transmitted to the upper limb, thereby exacerbating the risk of overuse injuries. Other contributing factors include training intensity, playing surfaces, and individual anatomical predispositions, all of which may modulate injury susceptibility.

A comprehensive approach to injury management emphasizes early diagnosis, conservative treatment, and preventive strategies. Biomechanical optimization, structured strengthening programs, and adequate recovery periods are fundamental in mitigating injury risk. In cases where conservative management fails, surgical intervention - ranging from arthroscopic procedures to decompressive techniques - may be required to restore function and alleviate symptoms.

CONCLUSIONS

Chronic overuse injuries represent a significant concern in tennis, surpassing the incidence of acute traumatic events. Understanding the biomechanical and physiological demands of the sport is crucial for developing effective prevention and management strategies. Proper technique, optimized equipment, and structured training regimens can significantly reduce the incidence of injuries. While conservative treatment remains the primary therapeutic approach, surgical intervention may be necessary in refractory cases. Future research should focus on refining injury prevention strategies, optimizing rehabilitation protocols, and evaluating the long-term musculoskeletal impact of repetitive tennis-related movements.

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