

Medial Elbow Microinstability in Tennis: A Narrative Review and Proposed Tennis-Specific Load – Adaptation Continuum

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Background: Medial elbow microinstability in tennis remains insufficiently characterized, and current understanding is largely derived from throwing athletes. The biomechanical evolution of modern tennis and cumulative tennis-specific loading may require a broader framework for understanding medial elbow dysfunction.

Purpose: To synthesize the available evidence regarding medial elbow overload and microinstability in tennis players and propose a hypothesis-generating, tennis-specific load–adaptation continuum to guide future clinical and biomechanical research.

Methods: A focused narrative review was conducted using PubMed, Scopus, and Web of Science. Studies addressing tennis biomechanics, medial collateral ligament pathology, medial elbow instability, diagnostic evaluation, rehabilitation, and surgical management were considered. Tennis-specific evidence was prioritized, whereas evidence from throwing and other overhead athletes was included when relevant to medial elbow anatomy, biomechanics, diagnosis, or treatment. Findings were synthesized qualitatively because of the heterogeneity and limited tennis-specific literature. No formal risk-of-bias assessment was performed.

Results: Thirty-two studies were included in the qualitative synthesis. The literature describes the biomechanical evolution of modern tennis, including greater topspin production, increased trunk rotational contribution, widespread use of semi-Western and Western grips, and demanding serving mechanics. However, direct evidence linking these technical characteristics to increased medial elbow loading or ulnar collateral ligament injury in tennis players remains limited. Available evidence suggests that medial elbow symptoms may reflect interactions among repetitive valgus-torsional loading, kinetic-chain function, dynamic stabilizer capacity, and capsuloligamentous adaptation. Based on this synthesis, we developed a five-stage conceptual load–adaptation continuum. This framework represents an expert interpretation of heterogeneous evidence and has not been prospectively or biomechanically validated.

Conclusion: This review adds an integrated, tennis-specific conceptual framework connecting biomechanics, clinical assessment, rehabilitation, and surgical considerations, rather than treating these domains independently.

What this study adds: The proposed continuum may support earlier recognition of functional medial elbow dysfunction, inform individualized clinical assessment and rehabilitation strategies, and guide future tennis-specific research, while clarifying the distinction between direct tennis evidence and extrapolated findings from overhead athletes.

Potential impacts on research, practice, or policy: This may improve transparency in future clinical reasoning. Prospective biomechanical and clinical studies are required to validate its stages, diagnostic relevance, and potential management implications.

Study Design: Narrative review and conceptual framework

Level of Evidence: Level V

Keywords: Tennis; Medial elbow microinstability; Ulnar collateral ligament; Tennis biomechanics; Rehabilitation.

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Introduction

Medial elbow pain associated with ulnar collateral ligament (UCL) dysfunction and subtle valgus instability represents an

increasingly recognized source of disability in overhead athletes [1][2]. Although medial elbow instability has been extensively investigated in throwing athletes, its mechanisms, clinical presentation, and progression remain less clearly defined in tennis players [3]. In baseball pitchers,

repetitive high-magnitude valgus loading has traditionally been associated with progressive structural compromise of the UCL, and ligament injury is commonly interpreted as a discrete structural lesion [1][4][5][6]. Tennis, however, presents a different biomechanical environment. Over recent decades, the sport has evolved toward greater playing intensity, increased topspin production, widespread adoption of semi-Western and Western grips, greater trunk rotational contribution, and changes in stroke and serve mechanics [7][8][9][10]. These developments may influence loading patterns throughout the kinetic chain and upper extremity. Unlike baseball pitching, which consists of relatively brief episodes of high-magnitude valgus loading, tennis exposes the elbow to repetitive submaximal valgus and torsional stresses generated through serves, groundstrokes, and prolonged training and competition volumes [11]. Efficient load distribution also depends on coordinated proximal-to-distal energy transfer involving the lower extremities, trunk, scapula, shoulder, forearm, and wrist. Consequently, medial elbow dysfunction in tennis may result from the interaction between cumulative mechanical exposure, kinetic-chain efficiency, dynamic stabilizer capacity, and progressive capsuloligamentous adaptation rather than from an isolated episode of ligament failure. In this context, medial elbow microinstability may represent a functional and potentially progressive condition in which repetitive loading increasingly exceeds the adaptive and stabilizing capacity of the medial elbow. For the purpose of this review, medial elbow microinstability is considered a symptomatic functional state in which repetitive valgus and torsional loading exceeds the capacity of the medial elbow to maintain effective dynamic and static load sharing, resulting in subtle functional instability without necessarily implying complete structural failure of the ulnar collateral ligament. Within the proposed continuum, capsuloligamentous adaptation refers to early tissue adaptation or increased laxity that may remain clinically asymptomatic, whereas symptomatic microinstability refers to the emergence of pain, functional impairment, or load-related instability associated with altered medial elbow behavior. Structural insufficiency represents a more advanced state in which clinically relevant ligamentous failure can be demonstrated by structural and/or dynamic assessment. These distinctions are conceptual rather than based on validated tennis-specific diagnostic thresholds, and the available literature does not currently establish definitive clinical or imaging criteria separating these stages.

However, a tennis-specific framework integrating the biomechanical mechanisms, clinical presentation, diagnostic assessment, rehabilitation, and surgical management of this condition is currently lacking. Moreover, much of the available knowledge continues to be extrapolated from baseball pitchers and other overhead athletes, and its direct

applicability to tennis remains uncertain. Therefore, the purpose of this narrative review was to synthesize the available evidence regarding medial elbow loading, UCL dysfunction, and microinstability in tennis players and to propose a tennis-specific load–adaptation continuum. The proposed framework is intended to support hypothesis generation and guide future biomechanical and prospective clinical research.

Methods

A focused narrative review using a structured literature search and selection process was conducted to synthesize the current evidence regarding medial elbow loading, ulnar collateral ligament (UCL) pathology, and medial elbow microinstability in tennis players, and to develop a tennis-specific conceptual load–adaptation continuum. The review was designed to integrate evidence from biomechanics, clinical assessment, imaging, rehabilitation, and surgical management into a unified conceptual framework relevant to tennis. A literature search was performed using the PubMed, Scopus, and Web of Science databases. The search strategy combined terms related to medial elbow pathology with tennis biomechanics and clinical management, including: (“ulnar collateral ligament” OR “medial collateral ligament” OR “medial elbow instability” OR “microinstability”) AND (“tennis” OR “tennis serve” OR “tennis biomechanics” OR “forehand” OR “kinetic chain”) AND (“rehabilitation” OR “return to play” OR “ligament repair” OR “internal brace” OR “ligament reconstruction” OR “elbow arthroscopy”). Priority was given to contemporary studies addressing tennis biomechanics, medial elbow loading, diagnostic evaluation, rehabilitation, and surgical treatment. When tennis-specific evidence was unavailable, relevant studies involving baseball pitchers and other overhead athletes were considered if their findings contributed to the understanding of medial elbow biomechanics, ligament function, diagnostic strategies, or treatment principles potentially applicable to tennis players. Eligible publications included biomechanical investigations, imaging studies, clinical studies, rehabilitation studies, systematic reviews, narrative reviews, and surgical case series relevant to medial elbow overload or instability in overhead athletes. The final literature search was performed in late July 2026. No publication-date restriction was applied. Conference abstracts, editorials, isolated case reports, pediatric studies, non-English publications, and studies unrelated to repetitive overhead loading were excluded. Study identification, screening, and data extraction were performed by the first author. This single-reviewer process may have introduced selection and data-extraction bias; however, the selection of the included evidence and the conceptual interpretation of the literature were subsequently discussed with the senior authors until consensus was reached. Given the heterogeneity of the available literature and the

conceptual objective of the review, findings were synthesized qualitatively rather than quantitatively. Evidence was organized into four principal domains:

1. biomechanical determinants of medial elbow loading;
2. interaction between the kinetic chain and dynamic stabilizers;
3. diagnostic evaluation of medial elbow microinstability;
4. conservative and surgical management.

The search and selection process was intended to provide a transparent and focused identification of relevant literature rather than to constitute a systematic review or formal evidence synthesis. Accordingly, no formal risk-of-bias assessment or quantitative synthesis was performed. Because the available literature was heterogeneous and included a substantial proportion of evidence derived from non-tennis overhead athletes, the evidence supporting the proposed continuum was categorized according to whether findings were directly derived from tennis populations or extrapolated from other overhead sports (**Table 1**). This categorization was intended to improve transparency

regarding the evidentiary basis of the proposed framework and does not represent a formal assessment of methodological quality or level of evidence. The available evidence was subsequently integrated into a proposed tennis-specific load–adaptation continuum describing the potential progression from functional overload to structural insufficiency. The proposed continuum should be interpreted as a conceptual, hypothesis-generating framework intended to stimulate future biomechanical and prospective clinical investigation rather than as a validated biological or clinical model.

Results

Study Selection

The database search yielded 247 records from PubMed, Scopus, and Web of Science. After duplicate removal and screening, 78 articles were selected for full-text review. Of these, 32 studies satisfied the eligibility criteria and were included in the qualitative synthesis (**Figure 1**).

A total of 247 records were identified through database searching. After duplicate removal, title/abstract screening, and full-text assessment, 32 studies met the inclusion criteria and were included in the qualitative synthesis.

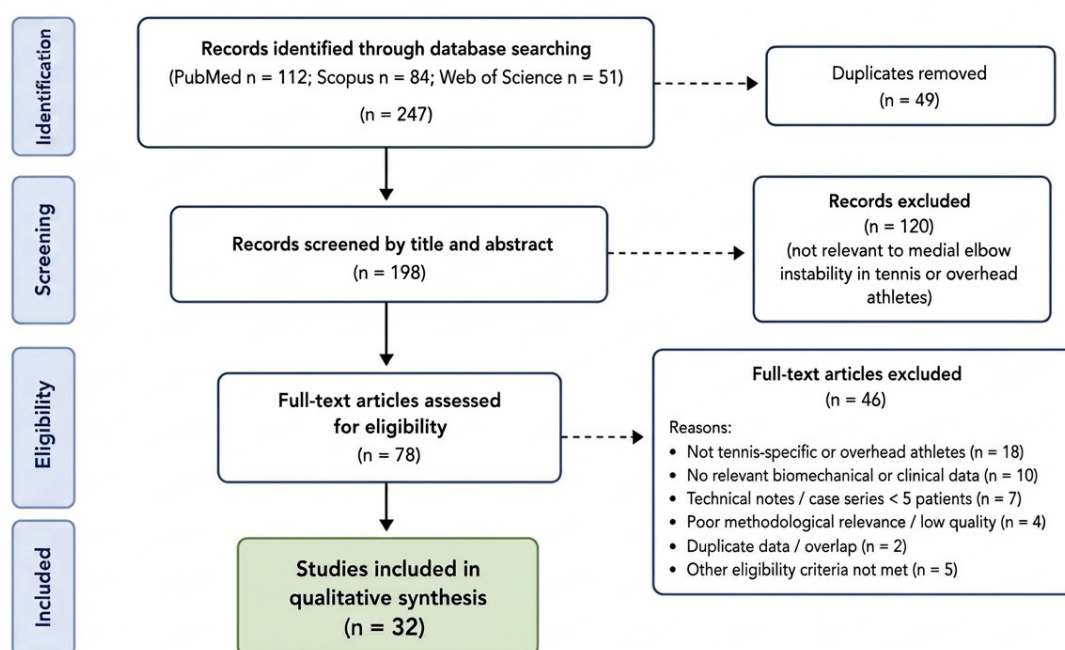


Figure 1. Flow diagram of study selection.

Component of proposed model	Tennis-specific evidence	Evidence extrapolated from overhead/throwing athletes	Nature of evidence
Repetitive valgus and torsional loading	Tennis-specific [7,8,9,10,12,13,14,15,16]	Extrapolated [1,2,3,11,17]	Direct tennis biomechanical evidence; relationship with medial elbow injury remains incompletely established
Flexor-pronator dynamic stabilization	Tennis-specific [7,8,9,10,12,13,15]	Extrapolated [3,18]	Evidence extrapolated
Ligamentous adaptation / elongation	– (No clearly established direct tennis-specific evidence)	Extrapolated [2,3,4,6,11,17]	Evidence extrapolated
Symptomatic medial elbow microinstability	Tennis-specific [11]	Extrapolated [2,3,4,17]	Evidence extrapolated
Structural UCL insufficiency	– (Limited direct tennis-specific evidence)	Extrapolated [1,2,3,6,11,17,19,20,21,22]	Evidence extrapolated
Possible secondary adaptive stiffness	– (No clearly established tennis-specific evidence)	Extrapolated [23–26]	Evidence extrapolated

*Referenced citations denote source studies in corresponding literature.

Table 1. Tennis-specific versus extrapolated evidence supporting the proposed medial elbow load–adaptation continuum.

Biomechanical and Functional Determinants of Medial Elbow Loading

Modern tennis is characterized by substantial biomechanical evolution compared with traditional stroke mechanics. Contemporary play increasingly relies on open-stance

forehands, greater topspin production, increased trunk rotation, and widespread use of semi-Western and Western grips [7][10]. These technical adaptations have modified upper-limb loading patterns during both groundstrokes and serving activities [12][13][14] (Figure 2A).

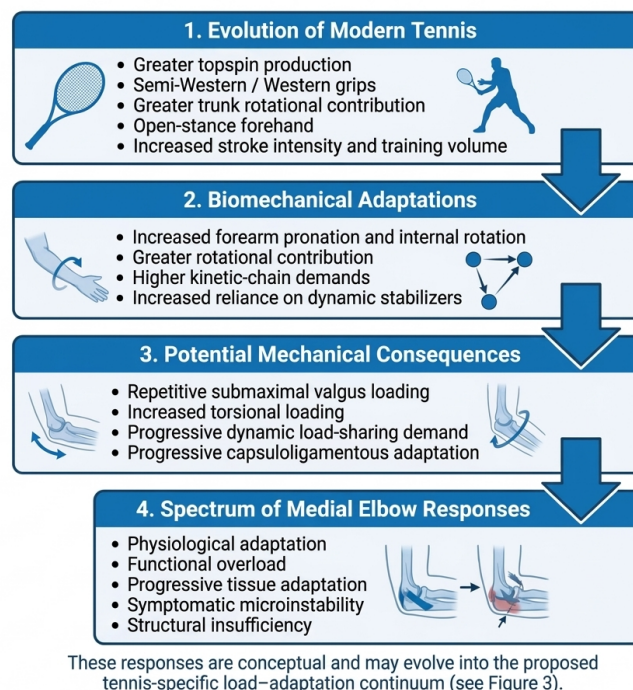


Figure 2A. Proposed conceptual model describing the biomechanical evolution of modern tennis and the potential mechanisms contributing to medial elbow overload. The proposed sequence represents a hypothesis-generating synthesis of the currently available literature and should not be interpreted as a validated causal pathway. Direct tennis-specific evidence remains limited.

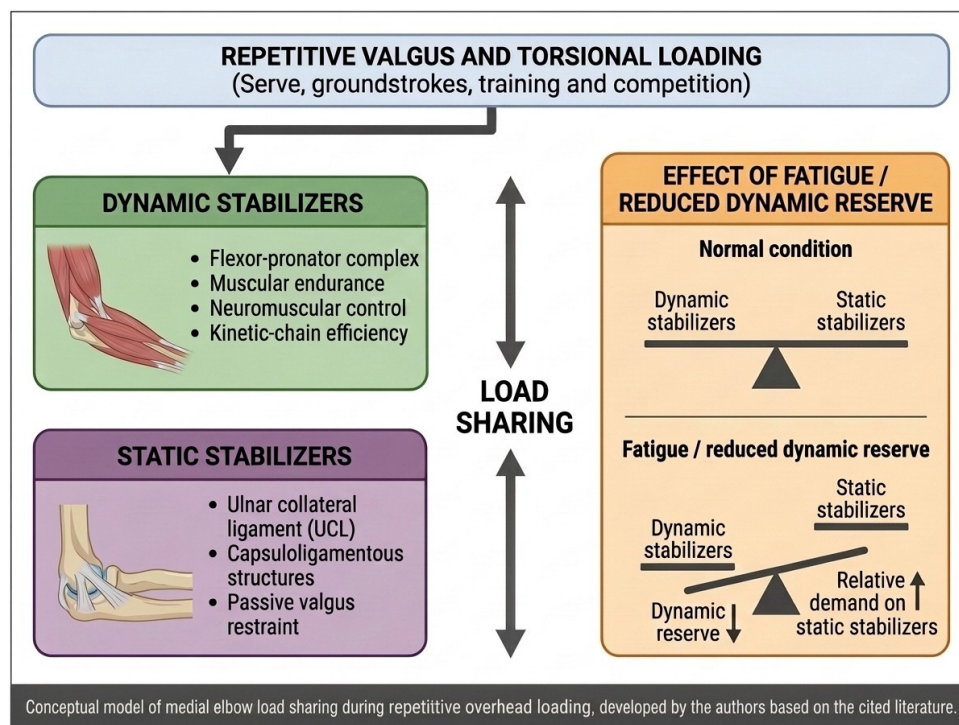


Figure 2B. Conceptual model of medial elbow load sharing during repetitive overhead loading. Repetitive valgus and torsional loads are distributed between dynamic stabilizers, particularly the flexor–pronator complex, and static medial elbow structures, including the ulnar collateral ligament and capsuloligamentous tissues. Reduced dynamic reserve due to fatigue or impaired kinetic-chain function may increase the relative demand placed on static stabilizers. The model is schematic and conceptual and does not represent measured patient data or a validated biomechanical relationship.

Semi-Western and Western grips have been associated with greater forearm pronation and altered racquet trajectories compared with traditional Eastern-grip mechanics [8][13][14]. Modern forehand techniques have additionally been characterized by increased rotational contribution from the trunk and kinetic chain [10][13][15][16]. The tennis serve has been identified as one of the most demanding overhead motions in sport [17][18][19]. Efficient proximal-to-distal sequencing of the kinetic chain has consistently been reported as an important determinant of both performance and load distribution [12][20][21]. Trunk rotation timing, lower-limb force generation, and energy-transfer efficiency contribute substantially to upper-extremity loading patterns during the serve [12][20][21]. Both dynamic and static stabilizers contribute to medial elbow stability. Dynamic stabilization is primarily provided by the flexor-pronator musculature, whereas static stabilization depends predominantly on the anterior bundle of the ulnar collateral ligament and the medial capsulo-ligamentous structures [5][17][18]. Experimental and clinical investigations have reported that muscular fatigue may influence dynamic stabilization capacity during repetitive loading conditions [22][23] (**Figure 2B**). Several studies additionally described the interaction between muscular and ligamentous structures as a load-sharing mechanism contributing to valgus stability during overhead athletic activities [5][17][22][24]. Fatigue-

related alterations in serve mechanics have also been reported. Repetitive serving tasks may modify kinematics and kinetic-chain sequencing, with associated changes in upper-limb loading patterns [12][21][25][26].

Diagnostic evaluation

Clinical studies reported that athletes with medial elbow pathology commonly present with medial elbow pain during serving activities, high-demand forehand mechanics, or repetitive overhead loading [5][22][27]. Additional reported symptoms included decreased performance, reduced stroke control, and activity-related discomfort [22][27]. Physical examination findings described in the literature included tenderness along the medial elbow, pain during valgus stress testing, positive moving valgus stress tests, and findings suggestive of flexor-pronator dysfunction. Assessment of kinetic-chain function, scapular control, and trunk mechanics was also reported as part of the clinical evaluation of overhead athletes [24]. Magnetic resonance imaging was the most frequently reported imaging modality for assessment of structural abnormalities involving the ulnar collateral ligament and surrounding soft tissues [5][27]. Reported findings included ligament thickening, signal alterations, partial-thickness tears, and associated changes involving the flexor-pronator mass and adjacent capsulo-ligamentous structures [2][16]. Magnetic resonance arthrography and CT

arthrography were additionally described as useful techniques in selected cases, particularly when subtle partial-thickness injuries, undersurface ligament lesions, or capsulo-ligamentous abnormalities were suspected [5][27]. Dynamic ultrasound evaluation was additionally described as a useful adjunct for assessment of medial joint opening during valgus stress testing and for evaluation of functional instability patterns [19]. Several studies highlighted its ability to provide real-time assessment of medial elbow behavior under loading conditions and to complement static imaging findings [19]. Dynamic imaging approaches may provide additional information regarding functional joint behavior when conventional static imaging findings are inconclusive.

Treatment strategies

Based on principles extrapolated from overhead athletes and broader sports rehabilitation literature, conservative management may represent a reasonable first-line approach for tennis players presenting with medial elbow overload, functional microinstability, or partial UCL involvement without advanced structural insufficiency. Rather than focusing exclusively on the ligament, treatment should address the multiple factors contributing to medial elbow loading, including cumulative training exposure, stroke mechanics, kinetic-chain function, and dynamic stabilizer performance. Rehabilitation may initially aim to reduce symptom-provoking mechanical loads while maintaining general physical conditioning. As symptoms improve, progressive loading of the flexor-pronator musculature may be introduced to restore dynamic valgus restraint and improve load sharing between the dynamic and static stabilizers of the medial elbow. Rehabilitation should also target deficits throughout the kinetic chain, including lower-limb force generation, trunk rotational control, scapular function, shoulder strength, forearm endurance, and neuromuscular coordination, as alterations in these components may increase mechanical demand on the medial elbow during repetitive tennis strokes. Sport-specific progression may therefore follow a gradual increase in mechanical demand rather than relying solely on time-based criteria. Progression may include sequential reintroduction of groundstrokes, serving drills, ball velocity, spin production, stroke volume, and match-specific activities according to symptom response and functional capacity. Although return-to-play protocols have been extensively described for baseball pitchers, comparable evidence for tennis players remains limited, highlighting the need for sport-specific rehabilitation strategies [28]. In the absence of validated tennis-specific criteria, return-to-play decisions may integrate clinical examination, restoration of strength and endurance of the flexor-pronator complex, recovery of kinetic-chain efficiency, tolerance to progressive sport-specific loading, and the athlete's ability to repeatedly perform high-demand strokes, particularly the serve, without symptom

recurrence. Validated tennis-specific return-to-play criteria are currently unavailable. Surgical treatment may be considered in athletes with persistent symptoms despite appropriate rehabilitation, clinically relevant valgus instability, progressive structural ligament insufficiency, or inability to tolerate competitive tennis demands [29]. Available procedures include primary UCL repair, repair with internal brace augmentation, and ligament reconstruction [30]. Primary repair is generally reserved for selected proximal or distal tears with adequate tissue quality, whereas reconstruction remains the preferred option for chronic insufficiency, complete rupture, poor tissue quality, or non-repairable lesions [22][23]. Although published studies have reported favorable outcomes following both repair and reconstruction, most evidence derives from baseball pitchers and other overhead athletes [31][32]. Consequently, the optimal surgical indications, rehabilitation protocols, and return-to-play expectations for competitive tennis players remain to be established through prospective sport-specific investigations.

Discussion

The principal contribution of the present narrative review is the integration of the currently available evidence into a tennis-specific conceptual framework describing medial elbow microinstability as a progressive load-adaptation continuum. Although the biomechanics of tennis, ulnar collateral ligament (UCL) pathology, diagnostic approaches, rehabilitation strategies, and surgical management have each been investigated previously, these topics have generally been addressed independently and predominantly in throwing athletes. By integrating these domains, the present review proposes a tennis-specific conceptual framework for understanding medial elbow microinstability. Importantly, the proposed continuum should be regarded as a hypothesis-generating framework rather than a validated biological sequence or clinical classification. Current evidence suggests that medial elbow loading in tennis differs substantially from the traditional throwing-athlete paradigm. Whereas baseball pitching is characterized by brief episodes of high-magnitude valgus stress, tennis exposes the upper extremity to repetitive submaximal valgus and torsional loading generated through serves, groundstrokes, prolonged training sessions, and cumulative competitive exposure. In addition, the biomechanical evolution of modern tennis—including greater topspin production, widespread adoption of semi-Western and Western grips, increased trunk rotational contribution, and higher stroke intensity—has likely modified loading patterns throughout the kinetic chain and upper limb. Nevertheless, direct evidence linking these technical characteristics to medial elbow injury remains scarce, and much of the current understanding continues to rely on extrapolation from baseball pitchers and other overhead athletes. A second important concept emerging from the

available literature is the central role of the kinetic chain in modulating medial elbow loading. Efficient force generation and proximal-to-distal energy transfer allow mechanical loads to be distributed across multiple anatomical segments, thereby reducing stress on the medial elbow. Conversely, deficits involving the lower extremities, trunk, scapula, shoulder, or forearm musculature may increase reliance on the static stabilizers of the elbow. In particular, the flexor-pronator musculature appears to function as an important dynamic valgus restraint through a load-sharing mechanism with the medial capsuloligamentous complex. Progressive fatigue, impaired neuromuscular control, or reduced muscular endurance may therefore compromise dynamic stabilization and contribute to symptomatic overload despite the absence of advanced structural ligament injury. These biomechanical concepts have relevant clinical implications. Early stages of medial elbow dysfunction may manifest as pain during serving, reduced stroke control, decreased performance, or fatigue despite minimal abnormalities on conventional imaging. Consequently, evaluation of symptomatic tennis players should extend beyond structural assessment of the UCL and include analysis of kinetic-chain function, scapular control, trunk mechanics, dynamic stabilizer performance, and neuromuscular coordination. Likewise, imaging findings should be interpreted in conjunction with clinical examination and functional assessment, as structural abnormalities alone may not accurately reflect the athlete's capacity to tolerate repetitive sport-specific loading. Dynamic imaging modalities, including stress ultrasonography, magnetic resonance arthrography, and CT arthrography, may provide complementary information when conventional magnetic resonance imaging is inconclusive or when functional instability is suspected. Based on the available evidence, we propose a five-stage tennis-specific load-adaptation continuum consisting of functional overload, ligament elongation, symptomatic microinstability, structural insufficiency, and secondary adaptive stiffness (**Figure 3**). Rather than representing distinct pathological entities, these stages should be viewed as interconnected phases within a dynamic process in which repetitive loading progressively exceeds the adaptive capacity of the medial elbow. As the ability of the dynamic stabilizers to share mechanical loads declines, increasing demand may be transferred to the static capsuloligamentous structures, potentially resulting in symptomatic instability and structural compromise. Conversely, prolonged adaptation to chronic loading may culminate in secondary stiffness and altered joint mechanics [2]. Although biologically plausible and consistent with the available literature, this continuum remains conceptual and requires prospective validation before being incorporated into clinical decision-making. The fifth component, secondary adaptive stiffness, should be interpreted particularly cautiously. Unlike the earlier

components of the proposed continuum, its occurrence as a downstream consequence of medial elbow microinstability has not been directly established in tennis players. It is therefore presented as a possible adaptive response to chronic or recurrent loading rather than as an obligatory stage in the natural history of medial elbow microinstability. From a rehabilitation perspective, the proposed continuum supports an approach that extends beyond treatment of the ligament itself. Restoration of flexor-pronator strength and endurance, optimization of kinetic-chain function, correction of biomechanical deficits, progressive exposure to tennis-specific loading, and careful management of cumulative training demands may all contribute to reducing mechanical stress on the medial elbow. Similarly, return-to-play decisions should integrate symptom resolution, functional performance, dynamic stabilizer capacity, and sport-specific loading tolerance rather than relying exclusively on imaging findings. However, validated tennis-specific rehabilitation and return-to-play criteria remain unavailable, highlighting an important area for future research. Finally, several questions remain unanswered. Prospective longitudinal studies are required to determine whether the proposed continuum accurately reflects the natural history of medial elbow dysfunction in tennis players. Future biomechanical investigations should quantify valgus and torsional loading during contemporary stroke mechanics, evaluate the contribution of the kinetic chain and dynamic stabilizers to medial elbow load sharing, and establish objective diagnostic criteria and evidence-based rehabilitation protocols. Improved understanding of these mechanisms may ultimately facilitate earlier recognition of medial elbow dysfunction, more individualized treatment strategies, and optimized return-to-play decision-making in competitive tennis athletes.

Limitations

The present review has several limitations. First, the literature specifically addressing medial elbow pathology in tennis remains limited, and many biomechanical, diagnostic, rehabilitation, and surgical concepts are extrapolated from baseball pitchers and other overhead athletes. Consequently, the direct applicability of these findings to tennis players remains uncertain. Second, this study was intentionally designed as a focused narrative review aimed at developing a conceptual framework rather than a systematic review or meta-analysis. Accordingly, no formal methodological quality assessment or risk-of-bias analysis was performed, and the proposed continuum should be interpreted as an expert synthesis of heterogeneous evidence rather than a quantitatively validated model. Third, the included literature comprised heterogeneous study designs, including biomechanical investigations, imaging studies, rehabilitation research, and surgical series, precluding quantitative

comparison of findings. Finally, the proposed load-adaptation continuum remains hypothetical and has not undergone prospective clinical or biomechanical validation. Future longitudinal studies combining biomechanical analysis, dynamic imaging, and clinical outcomes will be necessary to determine whether the proposed stages accurately reflect the progression of medial elbow pathology in tennis players.

Conclusion

Current evidence suggests that medial elbow pathology in tennis may involve biomechanical characteristics that are not fully represented by the traditional throwing-athlete paradigm. Although tennis-specific evidence remains limited, the available literature supports the hypothesis that repetitive

loading, kinetic-chain function, dynamic stabilizer performance, and capsuloligamentous adaptation collectively contribute to medial elbow overload. The present narrative review integrates these concepts into a proposed tennis-specific load-adaptation continuum describing medial elbow microinstability as a progressive conceptual model. Rather than representing a validated biological sequence or clinical pathway, this framework is intended to generate hypotheses, facilitate future biomechanical and clinical investigation, and encourage a broader functional interpretation of medial elbow pathology in tennis players. Prospective tennis-specific studies are now required to validate the proposed continuum, define its diagnostic value, and determine its potential implications for rehabilitation, return-to-play decision-making, and surgical indication.

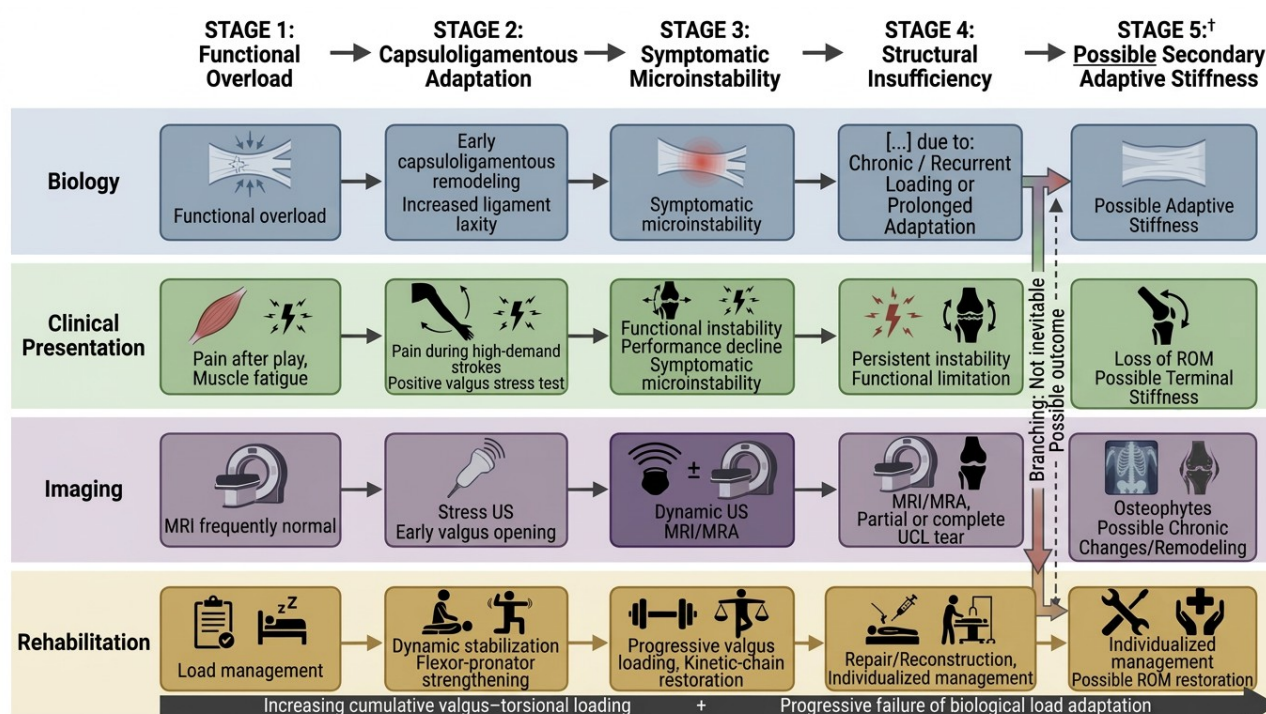


Figure 3. Proposed tennis-specific load-adaptation continuum of medial elbow microinstability. The model summarizes the hypothesized relationship among functional overload, ligamentous adaptation, symptomatic microinstability, and structural insufficiency by integrating biological changes, clinical manifestations, imaging findings, and management priorities. Possible secondary adaptive stiffness is presented as a hypothetical downstream consequence of chronic or recurrent loading rather than as an obligatory final stage. The continuum represents a conceptual framework intended to support clinical reasoning and generate future research hypotheses. Transitions between stages are not necessarily sequential or inevitable and have not yet been prospectively validated.

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Conflicts of interest

The authors declare that they have no conflicts of interest related to this work.

Author contributions

CC conceived the study, developed the original conceptual framework, performed the literature review, analyzed and synthesized the available evidence, and drafted the entire manuscript. DB, DDR, SC, and PA contributed to critical manuscript revision, scientific supervision, and final approval of the submitted version of the manuscript.

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