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Application of Rehabilitation Interventions in Triangular Fibrocartilage Complex Injury

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Abstract. The triangular fibrocartilage complex (TFCC) is an important soft tissue stabilizing structure on the lateral side of the wrist, and its injury is one of the most common causes of lateral wrist pain. The mechanisms of TFCC injury include acute trauma, degenerative microtrauma, and penetrating injuries, and the clinical manifestations include lateral wrist pain, decreased grip strength, and limited forearm rotation, etc. The Palmer classification divides the lesions into traumatic type I and degenerative type II. Due to the uneven blood supply of the TFCC, the central fibrocartilage disc has no blood supply and a poor ability of spontaneous repair, and timely intervention is crucial for prognosis. For some cases of partial thickness tear, degenerative lesion, and chronic symptomatic cases, conservative rehabilitation is the first-line treatment, which mainly includes physical factor therapy, therapeutic exercise, and orthopedic braces, targeting pain and inflammation regulation, dynamic neuromuscular stabilization, and passive mechanical protection. The subtypes of injury, tissue blood supply, severity of symptoms, and functional requirements jointly determine the optimal intervention combination. This article reviews these three rehabilitation modes, compares the clinical effects, and points out the shortcomings in current conservative treatment.

Keywords: TFCC injury, triangular fibrocartilage complex, wrist rehabilitation, conservative management, therapeutic exercise

1. Introduction

The triangular fibrocartilage complex (TFCC) is an integrated soft-tissue assembly on the ulnar aspect of the wrist, comprising the fibrocartilaginous disc, radioulnar ligaments, ulnocarpal ligaments and adjacent tendon sheaths. As the primary static stabiliser of the distal radioulnar joint (DRUJ), it transmits axial load from the hand to the forearm and permits the pronation–supination motion required by everyday gripping, lifting and rotational tasks. TFCC injury is one of the commonest causes of ulnar-sided wrist pain, and its presentation spans several distinct clinical pathways. Young, active patients and athletes typically sustain acute injury after a fall on an outstretched hand, whereas older adults and manual labourers more often develop degenerative lesions through repetitive microtrauma [1]. Penetrating wrist trauma represents a further, less common but clinically distinct mechanism: Charoenpol and Tawonsawatruk [2] described a double

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TFCC lesion following penetrating injury, illustrating that TFCC damage can coexist with other soft-tissue injuries and demands careful assessment before rehabilitation begins.

The Palmer classification remains the dominant clinical framework, separating traumatic Type I lesions from degenerative Type II lesions. A key biological constraint is the uneven blood supply across the complex: the peripheral rim is vascularised and capable of endogenous healing, while the central fibrocartilaginous disc is largely avascular, so full-thickness central tears have little spontaneous repair potential [3]. Patients typically report ulnar-sided pain, reduced grip strength, restricted forearm rotation and tenderness on weight-bearing or twisting movements. Without timely management, acute injury can progress to chronic wrist dysfunction that impedes return to work and sport. Across this spectrum, structured rehabilitation is a central determinant of functional outcome; even the evidence base for post-repair pathways confirms that the design, timing and progression of rehabilitation directly shape clinical and functional recovery [4]. For the majority of patients—those with partial-thickness tears, degenerative lesions or chronic symptomatic cases—conservative rehabilitation is the first-line approach, and three modalities form its backbone: physical agent therapy, therapeutic exercise and orthosis bracing. This paper reviews these three rehabilitation modalities, compares their appropriate clinical roles through the available literature, and identifies current gaps in conservative TFCC care.

Conservative rehabilitation for TFCC injury encompasses three core therapeutic categories, each with a distinct biological and mechanical target. Physical agent therapy primarily modulates pain and inflammation; therapeutic exercise builds dynamic neuromuscular stability; and orthosis bracing provides passive mechanical protection. No single modality is optimal for all patients or all recovery stages. Differences in injury subtype, tissue vascularity, symptom acuity, patient functional demands and treatment phase heavily influence which intervention, or which combination of interventions, will deliver optimal results.

2. Physical agent therapy

Physical agent therapy is most often used during the early symptomatic phase, when patients present with swelling, local tenderness and pain provoked by active or passive wrist movement. These non-invasive modalities modulate secondary inflammatory and nociceptive processes rather than directly repairing damaged fibrocartilage.

2.1. Therapeutic ultrasound

Therapeutic ultrasound is among the most widely used physical modalities in wrist rehabilitation. It produces both thermal and non-thermal effects intended to improve local perfusion and accelerate resolution of inflammatory exudate around peri-articular soft tissues. Narrative reviews of non-surgical TFCC management note that appropriately dosed ultrasound can provide symptomatic pain relief in acute TFCC-related irritation [3, 5]. Its biological ceiling is nonetheless clear. Because the central disc is avascular, ultrasound energy cannot stimulate fibrocartilage regeneration in central perforations; its value is confined to reducing secondary inflammation and pain rather than restoring structural integrity. Available supporting evidence comes largely from observational studies and case series, with high-quality controlled trials remaining scarce [1]. In routine practice, ultrasound is generally delivered within the first two to four weeks as one component of a multimodal programme.

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2.2. Extracorporeal shock wave therapy

Extracorporeal shock wave therapy (ESWT) delivers focused mechanical impulses to target tissues and is considered mainly for persistent chronic ulnar-sided wrist pain attributed to TFCC pathology [5]. Mechanical stimulation can alter peripheral nociceptor sensitivity and improve circulation within the vascularised peripheral rim, which distinguishes its theoretical rationale from that of ultrasound. Despite encouraging symptomatic reports in chronic refractory cases, there is no widely accepted standardised energy-dosage protocol specifically for TFCC lesions, and improperly high-energy settings risk secondary soft-tissue irritation. For this reason, clinical reviews position ESWT as an adjunct symptomatic tool rather than a standalone primary intervention [3]. Large-scale controlled trials clarifying its effect across Palmer subtypes are still lacking.

2.3. Comparative analysis of physical agent modalities

Therapeutic ultrasound and ESWT fit different clinical contexts, and neither is universally superior. Ultrasound is better suited to acute presentations dominated by inflammatory oedema, whereas ESWT is more appropriate once chronic fibrotic change and nociceptive sensitisation predominate [5]. Head-to-head randomised trials comparing the two within TFCC cohorts are absent from the current literature [1]. A shared limitation is that pain relief from either modality tends to be transient when it is not paired with exercise or bracing. Physical agents should therefore be embedded in multimodal rehabilitation rather than used as monotherapy.

3. Therapeutic exercise

Therapeutic exercise is the foundation of mid- to late-stage TFCC rehabilitation, introduced once acute swelling and sharp movement-provoked pain have sufficiently subsided. Its core principle is not to repair torn fibrocartilage directly, but to enhance neuromuscular control and build dynamic joint stability that compensates for the compromised static TFCC.

3.1. Isometric and dart-throwing motion training

Isometric muscle work is the safe starting point. Patients hold static wrist–forearm postures without large joint displacement, keeping DRUJ shear forces low and avoiding tensile loading on injured fibres. This activates the wrist flexor, extensor, pronator and supinator groups to build baseline strength without aggravating the lesion [6]. As tolerance improves, clinicians introduce dart-throwing motion training: a combined wrist flexion–extension with slight radial deviation that deliberately avoids high-stress ulnar deviation. The pattern engages multiple forearm muscles simultaneously and provides a functional bridge between static holds and daily activity [6]. Barlow similarly described a non-surgical exercise pathway for TFCC tears that begins with low-load isometric contraction and progresses incrementally, emphasising that progression must be matched to the individual patient rather than a fixed timetable [7].

3.2. Proprioceptive re-education and functional drills

Proprioceptive re-education is an essential additional component. It includes wrist joint-position-sense practice, perturbation training and upper-limb balance tasks aimed at restoring afferent feedback from wrist ligaments and surrounding soft tissues [6]. Systematic review evidence indicates that multi-component exercise programmes combining strength work with proprioceptive

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training yield better composite functional outcomes than strength-only protocols [1]. Scoping evidence on TFCC rehabilitation strategies likewise confirms that structured, progressively loaded programmes—combining controlled activation, functional movement and sensorimotor training—are associated with superior clinical and functional outcomes [4]. For athletic populations, later phases incorporate sport-specific progressive drills that reproduce the loading patterns and movement demands of the patient's activity, supporting a safe return to function.

3.3. Comparative analysis of exercise protocols

Each exercise component serves a distinct phase, and no single one achieves full recovery alone. Isometric work enables safe early activation but cannot build dynamic functional strength; dart-throwing drills provide a useful intermediate step but are not a terminal programme; proprioceptive training optimises neural feedback and may reduce re-injury risk but cannot replace resistance-based strength building [6]. Premature progression to heavy resistance or forceful ulnar deviation risks recurrent mechanical irritation and can convert an acute lesion into a chronic one. Relative to physical agents, exercise addresses the underlying mechanical stability deficit rather than only suppressing pain, and although physical agents may relieve pain more quickly, structured exercise produces more durable functional gains essential for return to work and sport [1].

4. Orthosis bracing

Orthosis and splinting provide passive mechanical protection to damaged TFCC tissue during the healing window. By constraining harmful movement patterns, bracing creates a stable biomechanical environment, though the optimal strategy differs by injury type and stage.

4.1. Immobilisation for acute traumatic cases

Wrist orthoses, including Muenster-style splints, restrict excessive forearm pronation–supination while preserving limited wrist flexion–extension, thereby reducing tensile and shear stress on the TFCC and stabilising DRUJ kinematics [8]. Cadaveric biomechanical work by Shin et al. demonstrated that a well-fitted external wrist band brace can reduce abnormal DRUJ translation under loading conditions, providing mechanical support for the clinical use of orthoses [9]. For acute traumatic Palmer Type I peripheral tears, short-term immobilisation supports healing within the vascular peripheral rim. Clinical evidence indicates that appropriately applied wrist bracing improves weight-bearing tolerance in patients with TFCC lesions, but prolonged fixation carries risks of disuse weakness and joint stiffness [8]. Clinicians must therefore closely monitor immobilisation duration in acute cases.

4.2. Intermittent bracing for degenerative lesions

Bracing strategy must be adjusted for degenerative Palmer Type II lesions. Sustained rigid immobilisation is discouraged because joint disuse worsens the pre-existing weakness and stiffness characteristic of degenerative wrist pathology. Intermittent protective bracing is preferred: patients wear the orthosis during high-load labour or sport to constrain harmful movement, and remove it during daily activity to preserve joint mobility [3]. The complications literature on wrist ligamentous and TFCC injuries cautions that over-immobilisation and excessive reliance on passive support are among the avoidable complications of management, alongside delayed progression to active

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rehabilitation [10]. Orthoses provide only passive mechanical support and cannot substitute for therapeutic exercise in the long term.

4.3. Comparative analysis of bracing strategies

Bracing approaches have distinct merits depending on injury subtype, and no single protocol fits all presentations. Short-term rigid splinting delivers immediate mechanical protection that neither physical agents nor exercise can replicate, making it irreplaceable in the early healing phase of unstable acute peripheral tears [8]. Yet bracing is purely passive: if patients rely only on splinting and neglect exercise, symptom relief tends to disappear once the orthosis is removed. For degenerative lesions, intermittent bracing better preserves joint mobility than prolonged rigid immobilisation. Within a full clinical workflow, orthosis bracing functions best as a time-limited protective bridge that allows safe progression into exercise-based rehabilitation [9].

Beyond these three pillars, manual therapy techniques such as soft-tissue mobilisation, DRUJ joint mobilisation and myofascial release are occasionally used as adjuncts. Although many patients report transient symptom relief, the high-quality controlled evidence supporting independent long-term benefit remains limited, and manual therapy should be regarded as an optional adjunct rather than a primary intervention [1].

5. Limitation

Despite expanding real-world clinical experience, multiple notable gaps persist within existing TFCC rehabilitation practice.

First, most rehabilitation studies do not stratify protocols by Palmer injury subtype. Traumatic peripheral tears, avascular central perforations and degenerative lesions have very different healing capacities, yet uniform programmes are frequently applied across subgroups. A protocol suited to a vascular peripheral tear may cause unnecessary stiffness in a degenerative lesion, while overly rapid exercise progression in a central avascular tear can perpetuate chronic irritation.

Second, there is a lack of standardised, objective quantitative criteria to guide rehabilitation progression. In practice, advancement is still largely driven by patients' subjective pain reports; biomechanical testing, standardised force measures and functional performance benchmarks are rarely formalised as decision thresholds. This inconsistency leaves some patients delaying necessary progress because of expected soreness, while others push through harmful mechanical overload.

Third, few trials provide long-term follow-up. Most report short-term pain endpoints in the weeks after treatment, and rarely track persistent DRUJ instability or recurrent ulnar-sided pain after patients return to full work or sport. Scoping reviews of TFCC rehabilitation similarly note substantial heterogeneity in outcome measures and follow-up duration, which limits direct comparison across programmes [4].

6. Conclusion

In conclusion, TFCC injury is a leading cause of disabling ulnar-sided wrist impairment. Multimodal rehabilitation—physical agent therapy, therapeutic exercise and orthosis bracing—plays an irreplaceable role across partial-thickness tears, degenerative lesions and chronic symptomatic cases. Physical agents manage early pain and inflammation but cannot repair fibrocartilage and must not be used alone. Progressive, individualised exercise builds the dynamic neuromuscular stability that compensates for damaged static structures, and delivers more durable functional gains than

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physical agents alone. Appropriately timed bracing supplies critical mechanical protection during healing, while over-immobilisation and over-reliance on passive support must be avoided. Manual therapy remains a cautious adjunct given limited high-level evidence. Future research should directly address the three identified gaps: studies stratified by Palmer subtype to match protocols to healing biology; objective, quantifiable progression criteria integrating biomechanical and functional benchmarks; and extended long-term follow-up with standardised outcome measures to evaluate sustained results and complication rates. These directions will refine evidence-based, patient-centred rehabilitation pathways for the diverse patients affected by TFCC injury.

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