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A case report: Triangular interval syndrome in an elite swimmer – A diagnostic pitfall of painless weakness

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Abstract

Triangular interval syndrome (TIS) is a rare cause of radial nerve entrapment, typically described in throwing athletes or military personnel. We present a case of TIS in an elite swimmer, uniquely characterized by painless weakness and a normal initial neurological examination, challenging conventional diagnostic pathways. A 17-year-old male presented with a 2-month history of isolated performance decline, manifesting as a loss of power during the pull-through phase. He denied pain but reported mild posterior shoulder discomfort post-training. Notably, his initial neurological exam was normal, leading to diagnostic delay. Diagnosis was confirmed via MRI showing lateral triceps edema and subsequent EMG demonstrating partial radial nerve denervation with reinnervation. The athlete's extreme training volume and identified glenohumeral internal rotation deficit (GIRD) were postulated as key biomechanical drivers. A multi-modal non-operative regimen including dry needling, neuromuscular electrical stimulation (NMES), and biomechanical correction resulted in complete resolution of symptoms and return to elite performance within two months. This case expands the etiological spectrum of TIS to include high-volume swimming. It underscores that TIS can present as an isolated motor syndrome without pain or initial electrodiagnostic abnormality. Clinicians should consider TIS in athletes with unexplained performance decline, utilizing advanced imaging and a high index of suspicion even in the face of normal preliminary investigations. Early, targeted intervention can yield excellent outcomes.

Keywords: Radial neuropathy, dry needling, swimming, shoulder

INTRODUCTION

Radial nerve entrapment represents a well-documented cause of upper extremity dysfunction, yet compression within the triangular interval remains a rare and frequently overlooked etiology. We present a diagnostically challenging case of triangular interval syndrome (TIS) in an elite swimmer that deviated from the classic presentation. The syndrome manifested primarily as isolated, painless weakness during the biomechanically specific pull-through phase of swimming, in the context of a normal initial neurological examination. This case aims to redefine the clinical phenotype of TIS and highlight the critical role of sport-specific biomechanical analysis in diagnosing obscure neuromuscular complaints.

CASE REPORT

This report details the case of a 17-year-old elite male swimmer from the Malaysian national program, who presented with a 2-month history of progressive performance decline, specifically a subjective loss of power and propulsion during the pull-through phase of freestyle and backstroke. The onset was insidious, characterized by an inability to achieve his personal best times despite intensified training. He explicitly denied frank pain, weakness in activities of daily living, or sensory disturbances. His only subjective complaint was intermittent, non-radiating discomfort localized to the posterior aspect of the right shoulder, which manifested post-training and resolved with rest.

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The athlete's regimen was notably rigorous, consisting of 4-6 hours of daily pool training. This volume was partitioned into multiple sessions with insufficient recovery, comprising high-intensity intervals of freestyle, backstroke, and breaststroke. This combination is critical, as freestyle and backstroke involve repetitive shoulder extension and adduction, directly loading the triangular interval, while breaststroke may contribute to overall muscular fatigue and GIRD. His schedule was further compounded by repeated competition cycles, creating a chronic state of overload without adequate physiological restitution.

Initial clinical examination, including basic neurological assessment (sensation, muscle strength in standard positions), was unremarkable, leading to initial dismissal. However, a subsequent evaluation revealed a significant glenohumeral internal rotation deficit (GIRD) of >20° on the affected side. Provocative testing, including resisted shoulder extension with the arm in adduction, reproduced his familiar sense of posterior shoulder heaviness and weakness. An initial nerve conduction study (NCS) was within normal limits (Table 1). A high-resolution MRI of the right humerus, however, revealed edema within the lateral head of the triceps brachii, highly suggestive of subacute denervation (Figure 1(a) and Figure 1(b). This finding, in the correct clinical context, is a pivotal diagnostic clue for TIS. Further examination on the MRI showed no space occupying lesion over the triangular interval area between teres major and humerus. (Figure 1(c) and Figure 1(d)) A follow-up electromyography (EMG) study confirmed the diagnosis, demonstrating partial denervation of the radial nerve with nascent potentials, indicating concurrent reinnervation (Table 2). His blood tests showed no signs of autoimmune disease or vitamin deficiency. (Table 3)

The management strategy was multi-modal and targeted the identified pathomechanics. It included a series of dry needling sessions targeting hypertonic muscles bordering the triangular interval specifically the teres major and infraspinatus coupled with manual therapy to address the GIRD. The coaching staff was consulted to modify his training volume and critically analyze his stroke biomechanics to reduce excessive shoulder adduction during recovery. The therapeutic exercise regimen focused on early triceps activation using neuromuscular electrical stimulation (NMES) to counteract atrophy, followed by progressive resistance training for the triceps and periscapular stabilizers. Following this targeted 2-month rehabilitation program, the athlete's symptoms completely resolved, and he successfully returned to his pre-injury level of performance.

DISCUSSION

This case provides several novel insights into the diagnosis and management of Triangular Interval Syndrome. First, it identifies high-volume swimming as a new etiological mechanism for TIS, expanding beyond the classic descriptions in overhead throwers. The patient's primary complaint of weakness during the pull-through phase of arm adduction and extension is a pathognomonic clue, directly implicating the teres major and triceps as key anatomical players. This specific functional deficit arises from the radial nerve's vulnerability within the fibro-osseous triangular interval, where it is susceptible to dynamic compression and fascial entrapment, particularly during the combined shoulder extension and adduction motion of the pull-through.[1] The swimmer's regimen, particularly the millions of repetitive shoulder extension-adduction cycles in freestyle and backstroke, acted as a chronic, dynamic stressor to the radial nerve within the triangular interval. The presence of GIRD is a crucial biomechanical finding that likely contributed to the pathogenesis. GIRD alters glenohumeral kinematics, potentially

Table 1: Initial nerve conduction study showed normal conduction

	Onset latency (ms)	Peak latency (ms)	Amplitude (microv)	segment	Distance (cm)	Velocity (m/s)
Right Radial (Superficial)	2.06	2.69	24.6	Forearm-wrist	10	48
Left Radial (Superficial)	1.54	2.27	27.7	Forearm-wrist	10	65

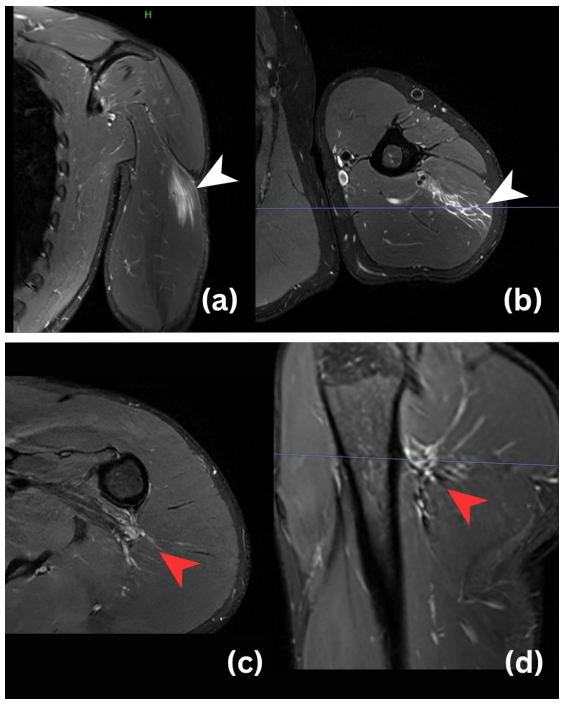

Figure 1. MRI of left humerus. (a) Coronal view of MRI humerus showing lateral triceps oedema (white arrow), corresponded to the axial view of MRI (b). (c) Axial view and (d) sagittal view showed no space occupying lesion or compression over radial nerve at triangular interval at left shoulder (red arrow)

increasing the tension and friction on the radial nerve as it courses through the triangular interval during the swimming stroke.

The cornerstone of successful management in this case was a targeted biomechanical intervention. Repetitive overhead motion, common in athletes and laborers, imposes significant strain on the posterior shoulder girdle. In TIS, hypertrophic or hypertonic teres major and long head of triceps muscles can narrow the triangular interval, creating a compartment-like syndrome.[2] Our rehabilitation protocol focused on modifying these aggravating activities, coupled with scapular stabilization and neuromuscular

Table 2: Electrodiagnostic evidence if left isolated partial radial nerve motor neuropathy with evidence of chronic reinnervation changes

		Spontaneous			MUAP			Recruitment pattern	Interference pattern
Muscle	nerve	IA	Fib	PSW	AMP	duration	PPP		
First dorsal interosseous	Ulnar	N	None	None	N	N	N	N	N
Extensor indicis propius	Radial	1+	None	None	N	N	2+	N	N
Pronator teres	median	N	None	None	N	N	N	N	N
Deltoid	Axillary	N	None	None	N	N	N	N	N
Biceps brachii	Musculocutaneous	N	None	None	N	N	N	N	N
Triceps	Radial	N	None	None	N	N	N	N	N
Cervical paraspinalis	spinal	N	None	None	N	N	N	N	N

Table 3: Autoimmune screening and vitamin level showed normal examination

Autoimmune screening			
	Anti Double stranded DNA (IU/ml)	negative	
	Anti Nuclear Antibody	negative	
	Extractable nuclear antigen	negative	
	C3 (mg/dl)	93.2	79 – 152
	C4 (mg/dl)	16.8	16 – 38
	C reactive protein (mg/dl)	< 0.5	< 0.5
Vitamin			
	Vitamin B12 (pmol/L)	661	136-652
	Folate (nmol/L)	24	7 – 46.4

re-education to restore optimal glenohumeral rhythm. By addressing the aberrant movement patterns that perpetuate nerve irritation, we aimed to mitigate the primary compressive forces, a principle supported in the management of other peripheral nerve entrapments.

A critical component of our intervention was the application of dry needling directly into the teres major muscle. We postulate that the profound weakness was not solely due to axonal disruption but also a significant neuropraxic component exacerbated by myofascial restriction and hypertonicity leading to compression of the radial nerve at triangular interval. Dry needling is thought to elicit a local twitch response, reducing muscle tension, improving local blood flow, and decreasing the release of inflammatory mediators, thereby decompressing the neurovascular structures within the interval.[3] The immediate and sustained improvement in our patient's pain and functional strength following dry needling sessions strongly suggests its role as a potent adjunct for releasing the primary mechanical compression in TIS.

Following the reduction of primary compression via dry needling and biomechanical correction, our focus shifted to nerve regeneration and neuromuscular recovery. Early activation of the compromised triceps muscle was achieved using Neuromuscular Electrical Stimulation (NMES). By providing an external stimulus for muscle contraction, NMES helps prevent atrophy, maintains muscle contractility, and promotes synaptic efficiency at the neuromuscular junction during the critical early phase of reinnervation.[4]

This case has several limitations. It is a single report, and the outcomes may not be generalizable to all TIS presentations. Furthermore, the synergistic effects of the multi-modal intervention make it difficult to isolate the individual contribution of each modality. Future prospective studies with larger cohorts and objective electrodiagnostic follow-up are warranted to validate this treatment algorithm.

TIS is a rare but clinically significant cause of radial nerve dysfunction. A high index of suspicion, triggered by specific functional weaknesses like the pull-through deficit, is crucial for early diagnosis. A multi-modal management approach addressing the underlying biomechanics with activity modification, releasing myofascial compression via dry needling, and promoting nerve recovery through early NMES and neuromodulation can yield excellent functional outcomes, potentially obviating the need for surgical intervention.

DISCLOSURE

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Conflict of interest: None

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