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Vascular adaptations and hemodynamic consequences of Thoracic Outlet Compression in overhead athletes – a comprehensive review

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Abstract

Introduction: Thoracic outlet syndrome (TOS) encompasses a range of disorders caused by compression of the neurovascular bundle at the thoracic outlet. Overhead athletes are at elevated risk due to repetitive overhead motion, muscle hypertrophy, and sport-specific biomechanics. Vascular compression, particularly of the subclavian vein and artery, induces distinct hemodynamic changes that may impair athletic performance and predispose to complications such as effort thrombosis, stenosis, and distal embolization [1–3]. This review paper aims to to synthesize current evidence regarding vascular adaptations, hemodynamic consequences, diagnostic strategies, and management approaches for thoracic outlet compression in overhead athletes.

Materials and methods: Literature from 2015–2025 was systematically searched through PubMed, Scopus, and Google Scholar using the terms: “thoracic outlet syndrome,” “overhead athletes,” “Paget-Schroetter syndrome,” “effort thrombosis,” “vascular compression,” and “rehabilitation.” Priority was given to systematic reviews, clinical trials, and high-quality case studies [4–6].

Summary: Vascular TOS in athletes manifests as venous thrombosis or stenosis due to repetitive microtrauma and hypertrophy of the scalene and pectoralis minor muscles [7–9]. Dynamic ultrasonography, MR angiography, and advanced 4D flow imaging enhance detection of flow disturbances during provocative maneuvers. Management includes conservative rehabilitation and surgical decompression, with individualized return-to-play protocols optimizing vascular recovery and functional outcomes [10–14].

Conclusions: Thoracic outlet compression represents an underrecognized but clinically significant cause of vascular dysfunction in overhead athletes. Early recognition, tailored management, and preventive training interventions are critical for preserving vascular health and athletic performance [15,16].

Keywords: Thoracic outlet syndrome, vascular compression, overhead athletes, effort thrombosis, subclavian vein, rehabilitation, hemodynamics

Introduction

Thoracic outlet syndrome (TOS) encompasses disorders caused by compression of the brachial plexus and subclavian vessels at the thoracic outlet [1]. Three main subtypes are recognized: neurogenic, venous, and arterial [2]. Neurogenic TOS is the most prevalent in the general population, but vascular forms—venous and arterial—pose serious concerns in athletes because of the risk for effort thrombosis, aneurysm formation, and distal embolization [3,4].

Repetitive overhead motion in sports such as baseball, volleyball, swimming, javelin throwing, and tennis produces high mechanical stress on the thoracic outlet, exacerbating structural narrowing [5,6]. Muscle hypertrophy of the scalene, subclavius, and pectoralis minor,

combined with sport-specific posture adaptations, reduces the costoclavicular space and can alter vascular flow [7–9]. Consequently, subclavian vein or artery compression leads to abnormal hemodynamics, impaired venous return, endothelial injury, and increased thrombosis risk [10,11].

This review examines vascular and hemodynamic adaptations in overhead athletes with TOS, emphasizing current evidence on diagnosis, management, and rehabilitation.

Epidemiology and classification of Thoracic Outlet Compression in athletes

TOS has an estimated prevalence of 3–80 cases per 100,000 individuals, with variation depending on diagnostic criteria and population studied [12]. In athletes, true prevalence is difficult to establish, as subclinical or transient compressions frequently go undetected [13]. However, vascular TOS—including venous (VTOS) and arterial (ATOS) forms—is increasingly recognized among high-performance overhead athletes [14,15].

VTOS accounts for roughly 3–5% of all TOS cases but is responsible for most acute presentations in athletes, often manifesting as Paget-Schroetter syndrome, or effort thrombosis, characterized by acute subclavian vein thrombosis after strenuous activity [16,17]. Arterial TOS represents less than 1% of TOS cases but may result in aneurysm formation, distal embolization, or digital ischemia [18].

Both anatomic variations—such as cervical ribs, fibrous bands, or hypertrophic scalene muscles—and functional narrowing due to repeated overhead motion increase compression risk [19,20]. These factors, combined with sport-specific kinematic patterns, predispose athletes to endothelial stress, turbulent flow, and vascular remodeling [21].

Pathophysiology and mechanisms of vascular compression

The thoracic outlet is bordered by the scalene muscles, first rib, and clavicle. Repetitive overhead activity produces microtrauma to these structures, causing inflammation, fibrosis, and progressive narrowing of the costoclavicular and subpectoral spaces [22,23]. Venous compression results in turbulent flow, endothelial injury, and thrombus formation—a process analogous to classical effort thrombosis described historically by Paget and Schrötter [24,25].

In arterial TOS, compression of the subclavian artery can lead to intimal hyperplasia, aneurysm formation, or distal embolization [26]. Chronic hemodynamic stress promotes vascular wall remodeling, oxidative stress, and endothelial dysfunction [27]. Doppler studies in overhead athletes have shown that shoulder abduction can reduce subclavian venous diameter by up to 60%, transiently decreasing flow and increasing venous pressure [28,29]. These dynamic changes explain exercise-induced swelling, cyanosis, and fatigue during overhead activity.

Hemodynamic consequences in overhead athletes

Hemodynamic alterations in TOS result from multifactorial interactions between mechanical compression, vascular remodeling, and endothelial dysfunction. Venous obstruction elevates local venous pressures, impairs muscle oxygenation, and accelerates fatigue during activity [30]. Recurrent episodes of venous stasis may lead to collateral vessel formation, chronic venous insufficiency, and increased thrombotic risk [31]. Severe cases may progress to axillary or subclavian thrombosis, occasionally resulting in pulmonary embolism [32].

Arterial compression, though less frequent, can compromise limb perfusion during peak exertion, causing transient ischemia and limiting athletic performance [33]. Chronic compression can also contribute to intimal damage, predisposing to aneurysmal changes and distal embolization [34].

Advanced imaging studies, including 4D flow MRI, have demonstrated reductions in flow velocity, abnormal shear stress, and altered nitric oxide-mediated endothelial signaling in compressed vessels [35,36]. These alterations may lead to maladaptive vascular remodeling and contribute to long-term functional impairment if untreated.

Diagnostic approaches

Diagnosis of vascular TOS is complicated by symptom overlap with other musculoskeletal and neurogenic conditions [37]. A combination of clinical assessment, provocative maneuvers, and imaging modalities is essential for accurate diagnosis [38].

Clinical evaluation: Symptoms include limb swelling, heaviness, cyanosis, paresthesia, or exertional fatigue. In venous TOS, acute post-exercise swelling and pain suggest effort thrombosis [39].

Imaging: Dynamic ultrasonography with Doppler is considered first-line, providing real-time assessment of vessel patency during arm movements. MR and CT angiography allow detailed anatomic evaluation and identification of stenoses, thromboses, or aneurysms [40].

Functional and hemodynamic tests: Venous plethysmography, near-infrared spectroscopy (NIRS), and laser Doppler flowmetry quantify perfusion and flow alterations during provocative positions, supporting early detection of subclinical compression.

Management Strategies

Management depends on TOS subtype, severity, and athlete-specific goals.

Conservative treatment: Focuses on posture optimization, scapular stabilization, and stretching of the scalene and pectoralis minor muscles [1,2]. Physiotherapy aims to restore the costoclavicular space, correct scapular dyskinesis, and improve shoulder kinematics [3,4]. Adjunctive interventions may include anti-inflammatory therapy, gradual conditioning, and load modification to reduce recurrent compression.

Pharmacologic and endovascular strategies: Anticoagulation remains the mainstay for venous thrombosis. Catheter-directed thrombolysis followed by surgical decompression yields favorable outcomes in athletes [5–7]. Endovascular stenting is considered in select cases of residual stenosis, though long-term patency remains a concern.

Surgical decompression: Techniques include first-rib resection, scalenectomy, and pectoralis minor tenotomy. Early surgical intervention in athletes presenting with effort thrombosis facilitates faster functional recovery and return to sport [8–10]. Postoperative rehabilitation emphasizes gradual range-of-motion exercises and vascular reconditioning.

Rehabilitation and return to play

Post-decompression rehabilitation is multidisciplinary, involving physiotherapists, vascular surgeons, and sports physicians [11]. Core objectives include restoring normal shoulder mechanics and vascular dynamics, preventing scar-related re-compression, and facilitating gradual return to overhead activities [12]. Rehabilitation programs integrating scapular control, cervical mobility, breathing mechanics, and neuromuscular retraining have been shown to reduce recurrence risk and optimize functional recovery [13,14]. Return-to-play timelines are generally 3–6 months following successful treatment, contingent on individualized progression and monitoring of vascular integrity [15].

Vascular adaptations and training implications

Chronic compression results in compensatory collateral venous formation, intimal thickening, and subclavian wall remodeling [16,17]. Endothelial dysfunction and reduced nitric oxide bioavailability contribute to maladaptive vascular responses [18,19]. Experimental and clinical data suggest that supplementation with L-citrulline or L-arginine may enhance nitric oxide production, potentially improving endothelial function and perfusion during high-intensity activity, though controlled studies in athletes remain limited [20]. Preventive strategies should incorporate load management, postural optimization, and sport-specific stretching protocols [21–23]. Ergonomic analysis of throwing or swimming techniques may further reduce subclavian compression risk.

Future directions

Prospective studies are needed to standardize diagnostic criteria and quantify the true prevalence of vascular TOS in overhead athletes. Advanced imaging modalities, including dynamic 4D MRI and Doppler techniques, may clarify the relationship between altered vascular flow and functional performance [24,25]. Further investigation into pharmacologic and nutraceutical interventions that enhance endothelial resilience could provide novel

preventive and therapeutic strategies. Additionally, integrating biomechanical assessment with individualized training programs may mitigate the risk of recurrent vascular compression and optimize long-term athletic outcomes [26,27].

Conclusions

Vascular compression at the thoracic outlet is an underrecognized but clinically significant issue in overhead athletes. It produces hemodynamic alterations that impair performance and may lead to thrombosis or ischemic complications.

Multimodal diagnosis, individualized management, and multidisciplinary rehabilitation are essential for safe return to sport. Preventive training modifications, early recognition, and tailored interventions remain fundamental for maintaining vascular health and long-term athletic performance [28–30].

Disclosure

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