Overview of venous pathology related to repetitive vascular trauma in athletes

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ABSTRACT

Background: Athletes are generally young, high-functioning individuals. Pathology in this cohort is associated with a decrease in function and consequently has major implications on quality of life. Venous disorders can be attributed to a combination of vascular compression with a high burden of activity.

Objective: This article promotes increased awareness of these uncommon conditions specific to the athlete by summarizing pathophysiology, clinical features, investigation, and treatment protocols for use in clinical practice. Prognostic outcomes of these management regimens are also discussed, allowing for clinicians to counsel these high-functioning individuals appropriately. With the aim of providing an overview of sport-related venous pathology, a literature review was undertaken identifying articles that were independently reviewed by the authors.

Results: Lower limb venous thrombosis has been identified in young, high-functioning athletes attributed to both compression-related venous trauma, associated with repetitive movements resulting in intimal damage, and blunt trauma. The diagnosis and treatment follow the same protocols as for the general population. Of note, early ambulation is advocated, with an aim to return to premorbid (noncontact) function within 6 weeks. Athletes performing high-intensity repetitive upper limb movement, such as baseball players, are predisposed to upper limb deep venous thrombosis (DVT). Diagnosis follows the same protocols as for lower extremity DVT; however, the optimal treatment strategy remains debated. Current guidelines advocate the use of anticoagulation alone. A specific subset of primary upper limb DVT is effort thrombosis, where there is compression at the level of the thoracic outlet. Thrombolysis with first rib resection is indicated in the acute setting within 14 days. In cases of complete occlusion, surgical decompression with venous reconstruction may be required. Popliteal vein entrapment syndrome is also discussed. This entity has been identified as an overuse injury associated with popliteal vein compression. Duplex ultrasound examination is indicated as a first-line investigation, with conservative noninvasive options considered as an initial management strategy. Chronic venous insufficiency or persistent symptoms may require subsequent surgical decompression.

Conclusions: Key conditions including upper extremity and lower extremity venous thrombosis, venous aneurysms, Paget-Schroetter syndrome (effort thrombosis), and popliteal vein entrapment syndrome are discussed. Further studies evaluating long-term outcomes on morbidity for current treatment regimens in upper extremity DVT, effort thrombosis, venous thoracic outlet syndrome, and popliteal venous entrapment syndrome are required. (J Vasc Surg: Venous and Lym Dis 2019;7:756-62.)

Keywords: Athlete; Deep venous thrombosis; Popliteal vein entrapment syndrome; Venous aneurysm; Venous thoracic outlet syndrome

Venous disorders in the athlete form part of the spectrum of vascular pathology associated with high-stress, repetitive trauma. They are a rare but important cause of morbidity in this largely young and healthy population. Athletes are at particular risk owing to a combination of high-volume activity in the context of muscular hypertrophy.[1] Early recognition and treatment of these conditions is imperative, allowing for a return to competitive function. A failure to return to premorbid state has severe implications for quality of life.

A single-center study reported venous pathology contributing to 46% of all vascular complications in competitive athletes,[2] most commonly affecting the subclavian vein. Venous compression in the context of repetitive intimal trauma is thought to lead to fibrous deposition resulting in vascular stenosis. This can predispose to symptomatic thrombus formation.[3]

This article provides an overview of the pathophysiology, clinical features, investigation, and management for the most common venous pathologies attributed to repetitive trauma in these high-functioning individuals. In addition, an outline of expected prognosis specific to the athlete population will provide information allowing for clinicians to counsel these individuals appropriately.

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Infrastructure support for this work was provided by the NIHR Imperial Biomedical Research Centre.

Author conflict of interest: none.

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The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2213-333X

https://doi.org/10.1016/j.jvsv.2019.03.012

A literature review was undertaken in February 2016 with search terms including "athlete," "repetitive strain," and "vascular injury," enabling the identification of articles that were independently reviewed by the authors.

LOWER EXTREMITY DEEP VENOUS THROMBOSIS

Deep venous thrombosis (DVT) is most commonly associated with venous stasis in the deep veins of the calf or pelvis, with an incidence that increases with age. Risk factors, including trauma, surgery, and immobilization, all contribute to promoting thrombus formation, which depends on Virchow's triad: stasis, hypercoagulability, and endothelial injury. Exercise has been shown to disrupt homeostatic mechanisms.[4] The degree of disruption is dependent on exercise intensity, with high-intensity training increasing both prothrombotic and fibrinolytic markers.[5]

Various case reports have identified lower extremity DVT in athletes, including (and not limited to) long distance runners,[6] military cadets,[7] soccer players,[8] and track athletes.[8] There are no specific sports known to predispose to this condition; however, these cases suggest any activity involving lower limb trauma—either blunt trauma or repetitive strain—increases the risk of developing thrombosis. Pathologic mechanisms promoting lower limb venous thromboembolism (VTE) specific to the athlete are multifactorial. In endurance athletes, venous compression by surrounding structures can cause microtrauma, endothelial cell damage, and subsequent activation of the coagulation cascade.[9] Blunt trauma, most common in contact sports, may also predispose these individuals to lower limb DVT.[10] Further physiologic factors contributing to a prothrombotic environment specific to this patient population include dehydration, increased flight time owing to travel, and bradycardia leading to venous stasis.[1,11,12] Idiopathic thrombus formation in the athlete is rare, but should be suspected in low-risk individuals presenting with an edematous, erythematous, and painful limb.[9]

The specific prevalence of VTE events in athletes is unknown. However, Bishop et al[11] report the rate of isolated lower extremity DVT as 27% of total VTE events in an athletic population when compared against upper extremity (UE) DVT, PE and combined DVT, and PE events (n = 55, between 1999 and 2016). The rate of combined DVT and pulmonary embolism (PE) events was 10.9% of all VTE episodes in athletes.[11] In young, active patients, it is important to rule out underlying conditions predisposing to venous thrombosis, including thrombophilias such as factor V Leiden and protein C deficiency; their presence increases recurrence rates.[13]

MANAGEMENT OF VTE IN THE ATHLETIC POPULATION

Diagnosis and initial treatment follow recommendations based on studies derived from the general population. Venous duplex ultrasound imaging is a safe, noninvasive test that can confirm the diagnosis of DVT (sensitivity 93% for proximal lower limb DVT).[14,15] Treatment involves immediate anticoagulation with low-molecular-weight heparin or unfractionated heparin to avoid DVT extension, the development of PE, and of complications such as the post-thrombotic syndrome. Initiation of oral factor Xa inhibitors is also advocated for initial anticoagulation unless contraindicated.[16,17]

Long-term anticoagulation therapy recommendations for athletes also remain the same as for the general population.[15] Antithrombotic medication selection is patient specific, and includes direct oral anticoagulants, warfarin, or low-molecular-weight heparin. The duration of therapy is guided by causation. First DVT with a transient risk factor present requires anticoagulation for 3 months.[18] Unprovoked DVT requires anticoagulation for at least 3 months, with a subsequent risk-benefit discussion.[18] The American College of Chest Physicians advise anticoagulation to be maintained for an indefinite period in individuals with idiopathic proximal DVT with low bleeding risk, or if a second idiopathic DVT arises.[18]

Although the evidence related to its prognostic outcome is limited, catheter-directed thrombolysis (CDT) may be considered to reduce the risk of recurrence and development of the post-thrombotic syndrome in patients with a proximal (iliofemoral) limb DVT.[19] Despite the recent ATTRACT trial suggesting that there is no benefit in its use, the methodology and results of this study have been the subject of much controversy.[20] Further data are, therefore, required to evaluate the prognostic outcome of CDT, including in the athlete patient group, in which, owing to their young age, the development of the post-thrombotic syndrome is particularly devastating.

With respect to ambulation in all individuals looking to return to premorbid athletic function, Depenbrock[15] advocates for the initiation of activity 24 hours after starting anticoagulation, provided there is no evidence of PE. This recommendation is on the premise that early ambulation may decrease the risk of DVT extension, improve symptoms, and potentially decrease the occurrence of the post-thrombotic syndrome, although data are sparse. This finding has been substantiated in a recent meta-analysis, advocating early mobilization in all acute DVT cases.[21] These individuals should look to gradually improve their function within the first 3 weeks. Between weeks 4 and 6, athletic activity can resume with an aim to return to full noncontact athletic function by week 6.[22]

Historically, it is advised to avoid participation in contact sports while anticoagulated after an acute VTE event owing to bleeding risk. However, in their expert analysis Berkowitz and Moll[22] highlight direct oral anticoagulants, with rapid onset and elimination of anticoagulant action, may play a role in expedited return to play with "personalized anticoagulation." This notion is of particular

relevance to athletes undertaking contact sports—dosing schedules depending on the pharmacokinetics and pharmacodynamics of these medications may allow for a return to activity in these individuals.[22] Anecdotal data from Nazha et al[23] advocate for an initial 3-month period of uninterrupted anticoagulation following VTE diagnosis (without "competitive, collision or high-risk activities"), followed by intermittent anticoagulation based on individual risk factors—in noncontact sports, anticoagulation should be held in competition and high-intensity practice sessions and resumed after; in contact sports, anticoagulation should be held on game days and for 24 to 48 hours after if there were any traumatic contact. Robust evidence-based studies are lacking in this area.

VENOUS ANEURYSMS

Thromboembolic events associated with venous aneurysms have been reported in the literature, most commonly in those affecting the deep venous system.[24] Although rare, these are an important cause of thrombosis and PE, although their pathophysiology is not fully understood.[25] There is evidence that they result from the interplay of innate and acquired risk factors, including blunt trauma and stretching, which promote venous wall weakness.[25] Owing to local trauma being a suspected cause, athletes performing repetitive motions are at risk, as Paes et al[26] demonstrate in a rower with two venous aneurysms (presenting as discrete soft lumps) on the dorsal aspect of the right forearm increasing in size on activity. Further case studies report the superficial femoral vein, common femoral vein and a saphenous vein tributary as sites of venous aneurysm in athletes.[27-29]

Popliteal venous aneurysms are the most common subtype. Owing to their risk of developing VTE, with Sessa et al[30] reporting 24% of popliteal venous aneurysms presenting with PE in their retrospective case study (n = 25), surgical intervention is advised for fusiform popliteal aneurysms greater than 20 mm and all saccular popliteal aneurysms.[31] Tangential excision and lateral venorrhaphy is recommended for saccular aneurysms, resulting in the greatest patency rate, and aneurysm resection with reconstruction for fusiform aneurysms to maintain venous continuity.[25,30] Postoperative anticoagulation is advocated with oral anticoagulation for 3 months.[30,32] Observation for asymptomatic venous aneurysms of the superficial system has been suggested owing to low risk of thromboembolism in these cases.[33]

UE DVT

Between 4% and 10% of all DVT events in the general population involve the UE, including the thoracic intlet.[34] Primary upper limb DVTs are idiopathic in nature, contributing to 20% of the total UE DVT case load.[35] Secondary upper limb DVTs occur most commonly owing to systemic disease and venous catheter insertion.[13] Complications include symptomatic PE, post-thrombotic syndrome, and recurrence after treatment.[34] The risk of PE is variable and has been cited as 2% to 36% in UE DVT cases.[34,35] In a recent retrospective cohort study involving 37,366 patients with acute DVT reflective of the general population, concomitant PE in UE DVT is less frequent than in lower extremity DVT cases (9.8% vs 25.0%).[36] However, note is made of PE recurrence rate during treatment in catheter-related UE DVT cases being higher when compared with the lower extremity DVT cohort (1.7% vs 1.3%, respectively).[36]

According to a recent review of injury reports from the National Hockey League, Major League Baseball, National Basketball Association, and National Football League in the United States, including only active professional athletes, UE DVT represented 34.5% of all VTE events reported, followed by lower limb DVT (27.3%), PE (27.3%), and DVT with PE (10.9%).[11] UE DVT was particularly prevalent in Major League Baseball players (68.9% of reported cases) compared with other groups (National Hockey League, 31.8%; National Football League, 8.3%; National Basketball Association, 0%), highlighting their increased risk associated with the repetitive upper limb motions these athletes perform.[11]

Guidelines for diagnosis are the same as for lower extremity DVT. The CHEST guidelines relating to UE DVT treatment advocates anticoagulant therapy alone, with duration as per lower extremity DVT.[37] However, note is made of CDT as a treatment option, which could be of benefit in patients with severe symptoms, axillary and subclavian vein thrombosis, acute-onset symptoms (<14 days), low bleeding risk, and a life expectancy of more than 1 year.[37] Indeed, CDT has been shown to decrease the rate of the post-thrombotic syndrome in proximal lower limb DVT, and improve the rate of iliofemoral patency, when compared with conventional anticoagulation treatment.[19] Further studies are required to validate its use in UE DVT where the development of post-thrombotic changes in the dominant arm can significantly impact on function.

VENOUS THORACIC OUTLET SYNDROME

Athletes are at particular risk of Paget-Schroetter syndrome (PSS), a subgroup of primary UE DVT also known as effort thrombosis. It is part of the thoracic outlet syndrome (TOS), a collection of conditions manifesting with vascular or neurological upper limb symptomatology secondary to compression of neurovascular structures at the level of the thoracic outlet. Venous TOS (VTOS) accounts for 2% to 3% of TOS cases.[38] PSS relates to VTOS associated with activity—60% to 80% of patients have a history of high-stress activities involving the upper limbs.[39] Although rare, with an incidence of 1 to 2 in 100,000 individuals per annum,[39] it is an important complication in athletes performing extensive

upper limb exercises, including baseball players, swimmers, and wrestlers.[40] Bilateral PSS is a reported but rare occurrence; a single study reported a prevalence of 6.1% among a population presenting with PSS.[41]

Although the exact etiology of PSS remains unknown, the presence of scar tissue surrounding the vasculature seen intraoperatively in these individuals is suggestive of a chronic inflammatory process occurring secondary to repetitive trauma from arm abduction and external rotation. This reduces the mobility of the UE, promoting further injury by venous compression and venous stenosis as a consequence of intimal hyperplasia.[39] Thrombosis likely occurs at a later stage after platelet activation and deposition.[2] UE DVT is associated with a reduced costoclavicular distance, suggesting anatomic variations of the thoracic outlet contribute to venous compression and disease.[42] A number of other factors, including muscle hypertrophy, have also been implicated in the pathology of effort thrombosis.[43]

PSS is characterized by acute onset upper limb edema, pain, discoloration, and parasthesia secondary to acute thrombosis, with 85% of individuals presenting less than 24 hours after exercise.[39] Duplex ultrasound examination is recommended as the first-line diagnostic investigation.[44] The use of magnetic resonance venography has been advocated in patients with an inconclusive ultrasound result (sensitivity, 100%; specificity, 97%) rather than catheter-directed venography.[40]

A recognition of a lack of randomized, controlled trials supporting treatment options and outcomes for all TOS conditions has led to the development of reporting standards by the Society for Vascular Surgery.[45] Treatment aims to prevent symptom progression and recurrent thrombosis. In acute cases, evidence supports thrombolysis (provided there are no contraindications) followed by surgical decompression with transaxillary first rib resection owing to high rethrombosis rates.[39] Current opinion supports either immediate or staged rib resection. However, the efficacy of thrombolysis is time dependent.[46] Illig et al[39] propose a treatment algorithm using symptom duration of 14 days as a benchmark for whether thrombolysis is likely to be successful.[39] In cases of total occlusion (ie, failure of thrombolysis or presentation more than 14 days after thrombosis where thrombolysis is likely to fail), surgical decompression with or without venous reconstruction is advised, depending on the severity of symptoms.

Open surgical decompression permits macroscopic assessment of the subclavian vein; where residual scar tissue is present, this is excised (circumferential venolysis). An infraclavicular, supraclavicular, or transaxillary approach may be used—there is no evidence advocating one approach over another.[47] If this procedure does not result in a resolution of subclavian vein stenosis (or in patients with recurrent symptomatology despite decompression), deep venous reconstruction may be required. There are three options where reconstruction is required:

1. Balloon angioplasty (eg, from ipsilateral basilic vein access)[39];
2. Vein patch angioplasty with or without the removal of intraluminal pathology (eg, cicatrix, mural thrombus, or neointima)[48]; and
3. Venous interposition bypass grafting to replace the diseased segment.[48]

Each of these procedures can be done at the initial rib resection in the properly prepared patient, thus avoiding the much more difficult and involved reoperative field. Deep venous reconstruction may also entail the formation of an arteriovenous fistula in the arm to increase UE venous flow in the first 3 months after surgical treatment. All patients with effort thrombosis should remain anticoagulated for at least 3 months.[37,39]

Studies relating to functional outcome following treatment of VTOS and PSS remain limited. However, Chandra et al[49] report a full return to competitive sport in 93% of athletes after surgical treatment of PSS involving thrombolysis, decompression (including venolysis), and venoplasty (n = 14). The mean time to recovery was 4.7 months. This outcome suggests that surgical management provides an excellent outcome for the professional athlete, likely resulting in a return to premorbid functional status and activity. This finding has also been demonstrated by Melby et al,[50] reporting a full return to activity in 100% of competitive athletes surgically treated for effort thrombosis (n = 32). Athletes were involved in a range of sports, including American football, baseball, basketball, swimming, tennis, volleyball, and fencing. Postoperative anticoagulation was maintained for 12 weeks in all cases, with median time to recovery of 3.5 months.

POPLITEAL VEIN ENTRAPMENT SYNDROME

Popliteal vein entrapment syndrome (PVES) results from popliteal vein compression; this syndrome uncommonly occurs in isolation, generally presenting with associated arterial involvement. Individuals with anatomic variations of the gastrocnemius muscle or popliteus hypertrophy are predisposed to PVES[51]; this finding is particularly relevant in athletes with lower limb exertion involving the triceps surae, which includes football, rugby, American football, or cycling.[3,51,52] Interestingly, there is evidence that this condition may represent a spectrum of disease—27% of asymptomatic adults have incidental subclinical PVES, suggesting it may be considered a benign entity in a subgroup of individuals.[53]

PVES should be considered an overuse injury with symptoms of venous claudication—lower limb edema on exertion with severe, bursting calf pain that, depending on the period of time spent exercising, will subside within a few minutes to a number of hours.[54] Initial examination and investigation may reveal evidence of raised popliteal compartment pressures—in their prospective cohort study,

Dijkstra et al[55] noted a median popliteal compartment pressure of 53 mm H_2O with the knee fully extended in limbs with PVES (n = 11) compared with a median value of 26 mm H_2O in controls with functional popliteal artery entrapment syndrome without venous compression (n = 13). Of note, compartment pressures in chronic exertion compartment syndrome require defined pre-exercise and postexercise intracompartmental pressure values with standardized knee and ankle positioning—not relevant in the diagnosis of PVES.[56]

In clinical practice, diagnosis depends on duplex ultrasound imaging.[51] Functional ankle movements—active ankle plantarflexion and passive ankle dorsiflexion with full knee extension—can elicit evidence of venous compression on dynamic studies.[54] It should be noted that venous duplex at rest is normal, unless there are signs of chronic venous injury. Although venography remains the gold standard investigation tool, magnetic resonance venography is increasingly being use. It confers diagnostic advantages over catheter-based venography by allowing for regional anatomic assessment, although accessibility remains limited.[57]

Conservative measures (compression stockings and leg elevation) are first-line treatment options.[2] In cases of severe chronic venous insufficiency or unremitting symptomatology in which no other cause can be identified, surgical decompression may be required. Indications include swelling, pain, stasis dermatitis, and ulceration after failure of conservative management.[58] This process involves surgical exploration with resection and popliteal vein release, dependent on the anatomic nature of the compressive mechanism.

Selection criteria for surgical intervention remain controversial. Raju et al[58] found that 100% of patients with an increase or drop in popliteal vein pressure on exercise by more than 15% preoperatively, suggestive of reduced flow, were noted to have improved outcomes on postoperative assessment.[58] Hence, the percutaneous measurement of pre-exercise and postexercise popliteal vein pressures may be used to identify patients who would functionally benefit from intervention. In this study, with 30 symptomatic patients undergoing "entrapment lysis" for venographic findings of popliteal compression, clinical outcome was labelled as excellent or improved in 79.3% of cases (n = 29).[58] Pain improved in 76% of individuals (n = 25). The median time for follow-up was 14 months. Hence, marked symptomatic benefit has been noted. However, with the median age being 49, this may not reflect the prognostic outcome in the competitive athlete. No case series data are available to guide return-to-play guidelines or prognosis after surgery for the athlete suffering from PVES. With total relief of pain and swelling reported as 48% and 41%, respectively, in the aforementioned study,[58] this finding may reflect a low likelihood of a return to premorbid status in this patient cohort. Further data are required.

CONCLUSIONS

This article has highlighted that athletes can present with a number of disorders affecting their venous system that require prompt identification and management to decrease the risk of adverse outcomes and maximize the chances of them returning to their functional baseline. There may be an under-representation of women in the available body of literature. In their retrospective review of VTE incidence in American athletes, Bishop et al[11] only identify male sports players. Indeed, with 21% of single-patient case studies in this review involving female patients (n = 3/14), this may impact clinicians' ability to make recommendations specific to the female athlete.

As a literature review, effect sizes are not measured, a key weakness in this work. Furthermore, limitations are largely determined by the quality of available evidence, which makes guiding management in this patient group difficult. In particular, return-to-play guidelines need validating with further long-term prognostic evidence. This highlights the need for further randomized, controlled trials to validate current recommendations.

AUTHOR CONTRIBUTIONS

Conception and design: AD
Analysis and interpretation: DM, SO, AD
Data collection: DM, SO
Writing the article: DM, SO
Critical revision of the article: SO, AD
Final approval of the article: DM, SO, AD
Statistical analysis: Not applicable
Obtained funding: Not applicable
Overall responsibility: AD

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Submitted Dec 12, 2018; accepted Mar 29, 2019.