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Title: Deep Vein Thrombosis in a Well-trained Masters Cyclist, is Popliteal Vein Entrapment Syndrome to blame?

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ABSTRACT

Whilst athletes are the epitome of health, venous thromboembolisms (VTE) including deep vein thrombosis and pulmonary embolism have been demonstrated to occur in well-trained athletes. VTE is frequently misdiagnosed and poorly treated within this population, often resulting in career or life-threatening ramifications. Furthermore, VTE risk rises with increasing age (>40 years), potentially affecting masters athletes. A 44-year-old well-trained male cyclist volunteered to participate in a research project investigating the influence of exercise on haemostasis in well-trained athletes. The cyclist presented with elevated D-Dimer levels both pre- (2251ng/mL) and post-exercise (2653ng/mL). The cyclist reported constant mild-pain in the left mid-calf region, with a cold tingling sensation in their left foot. Diagnosis of DVT was confirmed via a DVT squeeze test and Doppler ultrasound, with the clot located in the left popliteal vein. During the research project, the cyclist was exposed to numerous thrombogenic risk factors including travel, dehydration, prolonged sitting and exercise. The DVT in the popliteal vein may have resulted from repetitive movements associated with cycling. Additionally, hypertrophy of the gastrocnemius muscle may have impinged the vein. When diagnosing DVT within a cycling population, PVES should not be overlooked as a contributing factor.

KEY WORDS: deep vein thrombosis, doppler ultrasound, sports related compression, athletes

- Popliteal vein entrapment syndrome may lead to deep vein thrombosis in the calf region
- Undiagnosed symptoms of a deep vein thrombosis can replicate muscle strain and is often overlooked in athletic populations
- Travel and sporting position can cause compression of blood vessels, increasing an athletes risk for deep vein thrombosis
- Popliteal vein entrapment syndrome is not commonly looked into as a causation of deep vein thrombosis, however, it should be investigated in cyclists presenting with symptoms associated with deep vein thrombosis

INTRODUCTION

Venous thromboembolism (VTE) is frequently associated with physical inactivity and a sedentary lifestyle. Whilst athletes are the epitome of health, VTE has been demonstrated to occur in athletes [1]. Whilst the risk of VTE in an athletic population is similar to that of the general population (1 in 1000 people) [2], athletes may be at an increased risk due to athlete specific risk factors [3], disrupting factors associated with Virchow's Triad.

Although performing exercise is beneficial in the prevention of chronic diseases [4], exercise is well-demonstrated to disrupt haemostasis, resulting in an increased risk for thrombosis formation [5]. Additionally, to improve performance outcomes, muscular hypertrophy within an athletic population is often required [6]. Consequently, this may result in compression of the venous system, enhancing VTE risk. Indeed hypertrophy of the gastrocnemius muscle may result in entrapment of the popliteal vein, increasing venous stasis and acting as a pathophysiological risk factor for deep vein thrombosis (DVT) [7], especially within the sport of cycling. When cycling, cyclists adopt an aerodynamic position placing the hip into hyperflexion, with this position shown to compress the common iliac artery [8]. The repetitive hyperflexion of the hip, occurring up to ~8 million times a year in cyclists, may also damage the arterial wall due to bending and stretching of the external iliac artery [8], placing cyclists at an increased risk of DVT development. Additionally, cyclists are required to undertake frequent periods of long haul travel, resulting in prolonged periods of immobility, potentially increasing the risk of VTE, with travel promoting venous stasis and hypercoagulability.

Here we present an episode of unprovoked DVT in a well-trained male cyclist and an investigation in to the possible contributing factors that lead to the cyclist exhibiting symptoms whilst completing a 4km cycling time trial (TT) as part of a research project. Additionally, we identify a clear need for the examination of popliteal vein entrapment syndrome when diagnosing deep vein thrombosis within a cycling population.

METHODS AND RESULTS

Initial Assessment

A 44-year-old well-trained male cyclist with no blood disorders or family history of VTE, was prompted to visit a general practitioner (GP) on the discovery of elevated D-Dimer levels prior to and upon completion of five 4km cycling TTs as part of a research project investigating the influence of exercise on haemostasis in well-trained

athletes. Prior to participation, the cyclist was provided with a written description of the risks and benefits of the study and provided signed informed consent. Ethics was obtained from the Institutional Human Research Ethics Committee.

The cyclist trained five times per week, cycling > 10 hours per week. In addition to the cyclist's regular training in which an aerodynamic position was maintained for prolonged periods of time, he completed a 4km cycling TT completed at a high intensity, as part of a research project. Furthermore, the cyclist spent large periods of time in a seated immobile position due to travel required for work (3 ¼ h travel), and an increase in administrative workload at their place of employment.

Six weeks prior to diagnosis, the participant noticed symptoms including: constant mild pain (2/10 perceived pain rating (PPR)) located deep in the left mid-calf region, cold tingling sensation in their left foot, with this sensation especially present whilst in bed at night. Increasing pain (5/10 PPR) was also reported when completing hard cycling efforts (e.g. 4km cycling TT and intense intervals). These symptoms were initially thought to be delayed onset of muscle soreness by the cyclist, however, as they persisted and increased, he was able to distinguish the symptoms. Swelling or edema was not present within the cyclist as confirmed by a calf girth measurement. The cyclist presented with a history of Achilles tendon rupture of the left leg, requiring surgery when he was 28 years of age, which had subsequently resulted in anatomical abnormality of the medial and lateral heads of the left gastrocnemius (**Figure 1**).

Case Progression: Additional Symptoms and Diagnosis

The cyclist visited their GP after the discovery of elevated D-Dimer levels both pre- (2251ng/mL) and post-exercise (2653.0 ng/mL) with these results 35- and 30-fold greater than the group of well-trained cyclists mean, and above the normal guidelines for D-Dimer (<500ng/mL). From this, the cyclists GP conducted a series of tests including a DVT squeeze test and Doppler ultrasound (**Figure 2**), each confirming the diagnosis of a DVT in the left posterior tibial vein ascending into the left popliteal vein. The cyclist was also genetically screened for Factor V Leiden mutation, with no genetic mutations for hypercoagulablity noted.

Follow-up Investigations

Dehydration and May-Thurner syndrome were both ruled as inconclusive contributing mechanisms in the development of DVT, with May-Thurner Syndrome ruled out via a Doppler ultrasound.

Anticoagulation

The cyclist commenced oral anticoagulation medication therapy. Rivaroxaban was administered for the 8 weeks following diagnosis at an initial dosage of 30 mg (1 tablet daily), decreasing to 20 mg from 8 to 16 weeks. No further treatment was prescribed 16 weeks post initial diagnosis as no thrombosis was detected via Doppler ultrasound. Six months post diagnosis, the cyclist reported no complications/ loss of performance when returning to training and competition load.

DISCUSSION

This case report details the development of an unprovoked DVT in a healthy 44-year-old male cyclist after completing a 4km cycling TT. Despite no conclusive mechanisms identified contributing to the DVT development, possible factors such as cycling itself, cycling positioning and prolonged periods of immobility whilst traveling may have been contributing factors.

Whilst May-Thurner Syndrome was ruled out as a likely cause of DVT, similar mechanisms with regards to cycling position could have led to the mechanical compression of the iliac vessels, resulting in stasis rather than the syndrome itself. The aerodynamic position of cycling has been demonstrated to compress the iliac vessels due to hyperflexion of the hip [8]. This compression may result in venous stasis [9], which can stimulate blood clot formation. Indeed, sport specific activities may cause a disruption in normal circulation, with participation in some sports resulting in an extended period of time spent in compressed positions or postures [10]. With the cyclist riding >10 h per week, maintaining an aerodynamic position for much of this time, the aerodynamic position may be a contributing factor in the development of DVT.

Whilst May-Thurner Syndrome was excluded, popliteal vein entrapment syndrome (PVES) may have given rise to the formation of DVT within this cyclist. PVES is a sub-component of popliteal vascular entrapment, which also includes popliteal artery entrapment syndrome [11]. Entrapment of the popliteal artery/ vein results from hypertrophy of the surrounding muscles, specifically of the gastrocnemius, resulting in vessel wall damage and venous stasis [11]. However, little is known about this condition within a cycling population. Of interest is the

association of PVES and the intermittent compression and decompression of the popliteal vein which occurs during hyperextension and flexion of the knee [7], movements associated with cycling. Whilst the cyclist was cycling > 10 hours per week, this high training load may have contributed to the repetitive compression and decompression of the popliteal vein, resulting in possible musculo-tendinous compression [7], contributing to DVT development [9].

Abnormalities of the musculature and tendons have been reported to be contributing factors in PVES [12]. The cyclist presented with a previous history of a left Achilles tendon rupture resulting in remodeling of the medial and lateral heads of the gastrocnemius. Previous studies have demonstrated an association between previous Achilles tendon ruptures and DVT formation, however these studies were conducted over a 2 month period post-acute total Achilles tendon rupture [13] whereas the cyclist experienced this injury 15 years prior to DVT development. In addition, athletes are at an increased risk of type VI PVES [12] whereby the popliteal vein and artery are entrapped because of hypertrophy of the surrounding muscle. From this information, as well as the inconclusive nature of previous tests, it would be of interest to practitioners to conduct further tests to confirm or exclude PVES as a contributing factor in the development of DVT, especially within the cycling population.

Extended travel for training and competition is common for athletes, potentially increasing the risk of DVT through cramped travelling positions [14]. The cyclist reported long travel times and increased sedentary based activity, potentially contributing to DVT development. Indeed, venous stasis occurring at the lower limbs may result from long term immobilization and potential compression of the popliteal vessels when travelling, resulting in an increased risk of VTE [15].

CONCLUSION

The development of DVT in the popliteal vein may have resulted from intermittent compression and decompression of the vessel during hyper-extension and flexion, repetitive movements associated with cycling. Additionally, hypertrophy of the gastrocnemius muscle may have impinged the vein. PVES is a rare pathophysiological factor for DVT, often underdiagnosed in well-trained cyclists. Therefore, when diagnosing VTE within a cycling population, PVES should not be overlooked.

COMPLIANCE WITH ETHICAL STANDARDS

Funding: No funding was obtained for this study

Conflict of Interest: The authors declare that they have no conflict of interest

Ethical Approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all indivudal participants included in the study.

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