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Risk factors of multiple pulmonary emboli in an elite Ironman triathlete: a case report

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Objective: *To present a unique case of pulmonary embolism (PE) in an elite-Ironman triathlete and review athlete-specific risk factors associated with venous thromboembolism (VTE).*

Case presentation: *A 57-year-old male triathlete presented for chiropractic care of midback pain and dyspnea one day before competition. During competition, he was removed and unable to complete the event with dyspnea, chest, and midback pain. Vitals revealed prolonged elevated resting heart rate. He was triaged to the hospital with a high index of suspicion for PE. He was diagnosed with multiple PE in both lungs.*

Facteurs de risque d'embolies pulmonaires multiples chez un triathlète Ironman d'élite : un rapport de cas

Objectif: *Présenter un cas unique d'embolie pulmonaire (EP) chez un triathlète Ironman d'élite et passer en revue les facteurs de risque spécifiques aux athlètes associés à la thrombo-embolie veineuse.*

Présentation du cas: *Un triathlète de 57 ans s'est présenté pour des soins chiropratiques en raison de douleurs au milieu du dos et de dyspnée un jour avant la compétition. Pendant la compétition, il a été évacué et incapable de terminer l'épreuve en raison d'une dyspnée, d'une douleur thoracique et d'une douleur lombaire. Les signes vitaux ont révélé une élévation prolongée de la fréquence cardiaque au repos. Il a été transféré à l'hôpital avec une forte suspicion d'EP. On lui a diagnostiqué une EP multiple dans les deux poumons.*

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Summary: *VTE is not normally considered in athletes. A combination of athlete-specific risk factors may predispose athletes to a higher propensity of VTE. Due to life-threatening consequences, it is important to include a differential diagnosis of VTE in patients presenting with midback pain and dyspnea.*

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KEY WORDS: triathlete, pulmonary embolism, deep vein thrombosis, venous thromboembolism, endurance sport

Synthèse: *La thrombo-embolie veineuse n'est normalement pas envisagée chez les athlètes. Une combinaison de facteurs de risque spécifiques aux athlètes peut les prédisposer à une plus grande propension à la thrombo-embolie veineuse. En raison des conséquences potentiellement mortelles, il est important d'inclure un diagnostic différentiel de la thrombo-embolie veineuse chez les patients souffrant de douleurs lombaires et de dyspnée.*

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MOTS CLÉS : triathlète, embolie pulmonaire, thrombose veineuse profonde, thrombo-embolie veineuse, sport d'endurance, chiropratique

Introduction

Venous thromboembolism (VTE) encompasses both deep vein thrombosis (DVT) and pulmonary embolism (PE).[1] The pathogenesis of DVT begins with thrombogenesis, of which nearly 80% are accounted for in the lower extremity.[2,3] The most common site for a thrombus is in the valves and cusps of the superficial veins of the calf. However, they have also been reported in the femoral, popliteal, and iliac veins with the risk of PE increasing if the thrombus is located more proximally.[2] The thrombus will break off and ascend as an embolus along the venous pathway into the inferior vena cava into the right side of the heart and then pass through the pulmonary arteries into either the left or right lung.[3] The embolus will typically descend to the lower lobes of the lung due to gravity.[2] Consequently, the embolus will create increased pulmonary vascular resistance due to mechanical obstruction leading to the release of neurohormonal factors including serotonin, thrombin, and histamine.[3] This can lead to back-flow of blood to the right ventricle along with dilation and thinning of the right ventricular wall and higher perfusion demands from the right ventricle.[3] Subsequently, higher pressure from the right ventricle can lead to less distensibility and filling of the left ventricle and lower overall stroke volume.[3] Ultimately, this leads to tachycardia due to impaired systemic perfusion. In severe cases, lead to right-sided heart failure, lung infarctions, and sudden death.[3,4]

DVT and PE are typically considered health conditions that are associated with prolonged physical immobility, physical inactivity, and poorer metabolic health.[1,5,6] For instance, hospitalized residents account for nearly 60% of cases of VTE in the community.[6] Overall, the age-adjusted incidence rate is higher for men (130 per 100,000) compared to women (110 per 100,000) with the male to female reported as 1.2:1 historically.[5,6] Additionally, recent epidemiological data has reported a higher incidence of VTE due to an aging population and more specific and sensitive diagnostic tests.[5,6]

One classic triad of risk factors, Virchow's Triad (circa 1854) has been taught historically as factors associated with developing VTE.[1] The three pillars of risk factors in Virchow's Triad includes prolonged venous stasis, increased hypercoagulability, and endothelial injury.[1] Cardiovascular exercise, however, is believed to be a gold-standard preventative measure for VTE.[1,7] However, there are several case reports of DVT and PE in elite endurance athletes that are initially misdiagnosed as musculoskeletal sprain or strain-type injuries.[8] This can unfortunately lead to higher diagnostic lag time and potentially athletic career or life-altering health consequences.[1,6,8] While it is unknown the proportion of patients with VTE who present to a chiropractor for care, several published case reports stress the potential for patients with vascular pathologies that can present for care with musculoskeletal-like symptoms.[9-12] Vascular diseases, such as DVT

and PE are not often considered in the differential diagnosis process for conditions with musculoskeletal manifestations. This is due in part to the fact that pathology associated with these conditions are not commonly seen by physical therapists and chiropractors.[13] As such, the purpose of this case report is to highlight the unique risk factors and presentation of VTE in endurance athletes and to report a case of multiple PE that was suspected in an elite Ironman triathlete during competition.

Case presentation

The information from this case report is based on clinical documentation obtained during the patient's sideline evaluation and hospital records.

Pre-competition: travel, symptoms, and chiropractic management

A 57-year-old male elite-level Ironman triathlete travelled seven hours by car five days prior to the triathlon. He reported that he previously competed in a marathon two weeks prior to the triathlon.

One day prior to competition, he presented to a nearby community chiropractor for management of new-onset left-sided thoracic spine and chest pain and attributed to his long drive. He reported that his chest pain worsened with deep inspiration. While the details and clinical notes of the assessment were not obtained, he reported to received spinal manipulative therapy and taping to the thoracic spine. He reported that his pain reduced following treatment, but he had difficulty sleeping the night before the triathlon due to his thoracic spine and chest pain.

Competition day: sideline evaluation

The average air and water temperature on the day of competition were recorded at 26°C and 20°C, respectively. During the swimming component of the triathlon, he was removed by rescue kayaks for shoreside medical evaluation as he was reportedly struggling to swim and became presyncopal. His chief complaint included intermittent pleuritic chest pain, left periscapular and thoracic spine pain, and worsening exertional dyspnea. On observation by the side-line sport medicine physician, he was alert but appeared uncomfortable. Visual inspection revealed no gross abnormalities, with the exception of slight pallor in his face. His initial resting heart rate (RHR) was recorded at 98 bpm and blood pressure was recorded at 128/78 mmHg. Respiratory rate was 14 breaths per minute. Palpation of his radial pulses were strong bilaterally. Oxygen saturation levels were recorded at 98% SpO_2 with a pulse oximeter. Auscultatory examination of the lung fields were unremarkable bilaterally.

His medical history included a previous DVT three years ago and benign prostate hyperplasia which was well managed with finasteride. His previous DVT occurred following a long duration of travel and was managed with Warfarin and Heparin for three months. His family medical history included a myocardial infarction that his mother suffered at the age of 63. He denied any family history of clotting disorders.

After continued monitoring of the patient for 20-minutes, he reported a complete resolution of chest pain, and he felt ready to complete the triathlon. Sustained measurement of his heart rate remained elevated at 98 bpm and he reported that his normal RHR was historically between 54-56 bpm. Due to a strong suspicion for serious cardiovascular or cardiorespiratory pathology, the side-line sport medicine physician removed him from competition, and he was transported to a local hospital via ambulance.

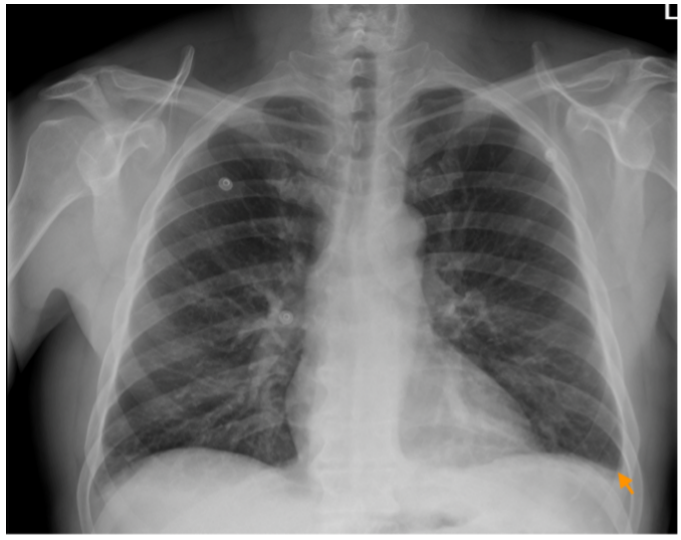

Figure 1a.

X-ray examination of the chest reported as normal. In retrospect there is questionable increased opacity of the basilar zones of the lungs bilaterally, as well as minimal blunting of the left costophrenic angle (yellow arrow). However, none of the classic radiographic findings such as "Westermark" or "Hampton Hump" signs are seen.

Triage and hospital management

Electrocardiography (ECG) in the ambulance revealed a normal sinus rhythm and heart rate of 98 bpm. At the hospital, a chest X-ray was reported as normal (Figure 1a). However, upon retrospective evaluation, there was questionable increased opacity of the basilar zones of the lungs bilaterally, as well as minimal blunting of the left costophrenic angle. A Computed Tomography Pulmonary Angiography (CTPA) confirmed multiple pulmonary emboli in both lungs (Figure 1b). A D-dimer test was over 5000µg/L. Rivaroxaban (15 mg twice daily) was started immediately. The patient was admitted to the hospital overnight and discharged the next day. He was prescribed Rivaroxaban for one month and instructed to follow up with his Family Physician and a Hematologist. Furthermore, he was restricted by the attending physician to participate in sub-symptom threshold exercise and instructed to present to emergency if he continued to experience any exercise-induced dyspnea.

Discussion

The incidence of DVT and PE has not been investigated in the athletic population.[14] Taylor *et al*.[8] published a case report of approximately 50 professional and elite-level

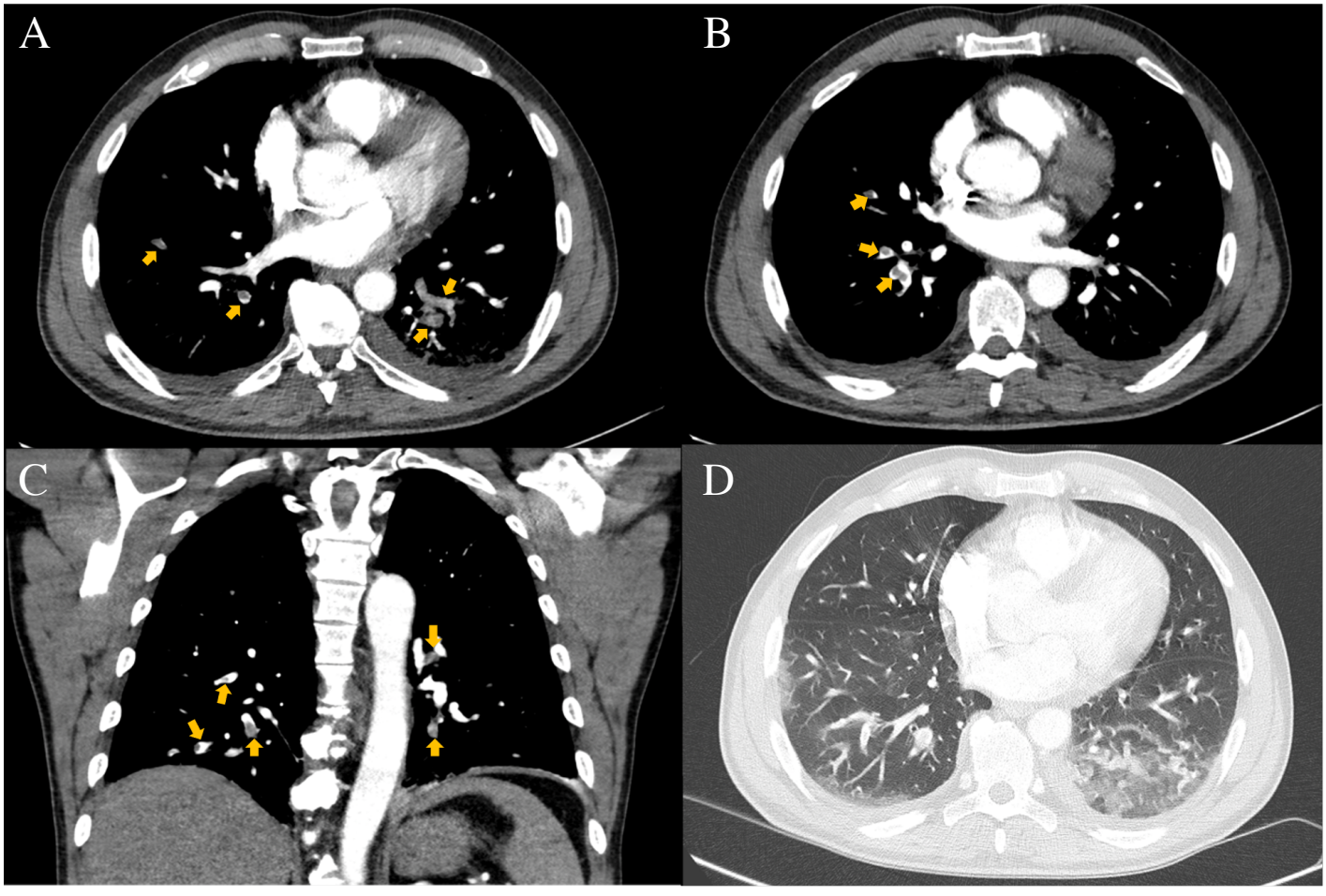

Figure 1b.

Axial and Coronal CTPA, soft tissue windows (A,B,C) demonstrates multiple pulmonary emboli bilaterally in segmental and sub-segmental arteries of the lower, middle and upper lobes. These present as central filling defects in the contrast filled arterial system (yellow arrows). Axial CTPA, lung window (D) demonstrates the resulting consequence to the lung parenchyma; bilateral basilar subsegmental atelectasis with a mild bilateral pleural effusion.

Table 1.
Overview of risk factors for VTE in endurance athletes

Hypercoagulability	Venous stasis	Endothelial injury
• Inherited thrombophilia[15] • Oral contraceptive use[15,16] • Hemoconcentration (prolonged exercise + dehydration + heat)[8,14,15]	• Long duration of travel[18,19] • Athlete's heart (bradycardia + hypotension)[14,20]	• Post-exercise inflammation[14,21] • Repetitive microvascular damage from sport[8]

athletes who experienced PE acutely following exercise. Importantly, over 50% of athletes were initially misdiagnosed, leading to an average diagnostic lag time of greater than one month and return to play times ranging from two to eight months.[8] It is critical for clinicians in sport to understand athlete-specific risk factors from Virchow's triad that can ultimately lead to improved VTE screening and identification (Table 1). This section will dive into the three pillars of Virchow's triad and highlight key areas for clinician awareness.

Hypercoagulability

Previous literature has reported conflicting conclusions on oral contraceptive (OC) use and increased risk of VTE.[15,16] A systematic review by de Bastos *et al*.[16] reported a two-fold increase of risk of thrombosis from all individual types of combined (estrogen with progesterone) OCs and upwards of a fourfold increase in contraceptives with an estrogen dosage greater than 30 μg. It is unclear whether progesterone-based oral contraceptives may be protective against VTE.[16] While the use of OCs in athletes has been reported as similar compared to the non-athletic population, OCs have been shown to decrease the fibrinolytic response and increase coagulative factors during exercise.[15] Taylor *et al*.[8] also reported that nearly 74% of female athletes with VTE reported OC use at the time of diagnosis or symptom presentation with PE. Endurance athletes are also susceptible to other physiological changes in blood that occur after acute exercise. For instance, El-Sayed *et al*.[17] reported that prolonged high-intensity exercise leads to an increase in platelet aggregation and increases in both coagulatory and fibrinolytic factors. Hemoconcentration can be further exacerbated in endurance athletes due to dehydration and prolonged heat exposure from their sport-related demands, but the combined relationship of all these variables for VTE risk has yet to be investigated in endurance athletes.[8,14,15] Another important risk factor to consider are athletes with hereditary thrombophilia.[14] Reportedly, the prevalence of hereditary thrombophilia is similar in the athletic and non-athletic population.[15] Taylor *et al*.[8] reported that 25.5% of athletes were found to have some form of inherited thrombophilia that was later diagnosed following a DVT or PE. While the types of prothrombotic mutations were not disclosed, this is an important consideration as more common heredity mutations including Factor V Leiden or prothrombin deficiencies can put patients at a higher risk of a VTE event between two-to-eighty times greater.[15]

Venous stasis

Long duration of travel (e.g., car or flight) has classically been associated with increased VTE risk.[18] Philbrick *et al*.[18] reported the incidence of VTE at 4.7 per one million travelers with an average flight duration of greater than hours in the general population. Only one observation study in athletes investigated travel of greater than four hours compared to less than two hours in trained marathon athletes and thrombogenic risk.[19] They reported that athletes that traveled greater than four hours had a statistically significant increase in serum coagulatory factors, however, one limitation is that the long-travel group was on average, 10-years-older than the short-travel group.[19] One additional factor for increased circulatory stasis includes characteristic structural, functional, and electrical remodeling associated with regular athletic training, otherwise known as athlete's heart.[20] High-level endurance athletes commonly show greater left ventricular hypertrophy, increased stroke volume and parasympathetic tone, leading to an average resting heart rate of less than 50 bpm and hypotensive blood pressure measurements compared to the general population.[20] This slower circulatory flow has been postulated to increased venous stasis

and the potential for DVT formation in the athletic population.[14]

Endothelial injury

Taylor *et al*.[8] reported that approximately 15% of athletes were diagnosed with a recent lower limb strain or sprain injury prior to experiencing a PE. Additionally, the pathogenesis of upper extremity DVTs including Paget-Schroetter syndrome have been associated with repetitive arm use and injury to the axillary and/or subclavian vein.[8] While previous literature has shown a transient increase in inflammatory and muscle injury biomarkers peaking 24-48 hours post-marathon, it is hypothesized that this leads to a period of susceptibility within the vascular endothelium for developing a VTE following aerobic exercise.[14,21]

Recognition

Reflecting on the patient in this case, there are several factors of Virchow's Triad that can be considered. Our patient did not have any family history of clotting disorders or oral contraceptive use. Second, his medical documentation reported that his previous DVT was attributed to traveling on a very long trip. He subsequently developed symptoms of DVT within one week of his travel. Similarly, prior to this triathlon, our patient had a history of long duration of travel and developed symptoms of VTE within one week of his travel. Next, our patient was an experienced endurance athlete that understood his normal RHR range around 54-56 bpm. After 20-minutes of resting for sideline medical evaluation, his RHR continued to be sustained at 98 bpm. Lastly, while this patient's hydration status at the triathlon and previous strenuous training was unclear, it is possible that these factors could contribute to higher hemoconcentration and aggregation of coagulatory factors.[17,21]

Currently, there is no gold-standard for the sideline assessment of VTE in endurance athletes. Historically, the Wells score, a clinical prediction score criteria, is the most widely used tool for risk stratification of DVT or PE in the general population due to its high sensitivity (0.92).[22] The Wells score consists of a diagnostic, point-based algorithm for patients with suspected DVT/PE that is calculated based on specific patient history or physical examination findings that are present.[22] For instance, patients with a RHR of >100 bpm would receive a +1 score indicating a higher suspicion for PE based on the Wells score. However, when tested retrospectively in athletes who developed a DVT or PE, the Wells score had a 100% failure rate for correctly identifying athletes with known VTE.[22] Furthermore, the patient in our case would have a total score indicating DVT or PE is unlikely according to the Wells score criteria. As such, clinicians may be challenged when screening and identifying VTE in athletes when non-athletic interventions are applied.

Additional testing including ECG, D-dimer, or diagnostic imaging is often required for the diagnosis of PE.[23,24] Sinus tachycardia, a non-specific finding, is the most common abnormality observed on ECG.[2] D-dimer, a commonly investigated biological marker, is the product of breakdown of the fibrous mesh of a blood clot.[24] High levels of D-dimer (>5,000 μg/L) are another non-specific marker associated with several severe diseases including malignancy, sepsis, VTE, trauma, and aortic aneurysm as examples.[25] Lastly, characteristic signs of VTE observed on X-ray include Westermark sign, a focal area of hyperlucency in the vessels distal to the site of occlusion, and Hampton Hump, a dome-shaped pleural opacity in the lung.[23] The sensitivity and specificity for PE of Westermark sign is 14% and 92%, and the Hampton Hump is 22% and 82%, respectively.[23] Chest radiographs are often the first-line imaging modality used to assess differential diagnoses for pleuritic chest pain including pneumonia, pneumothorax, and PE.[23] CT pulmonary angiography is the gold-standard for the diagnosis of PE.[24] This involves injecting the patient with contrast and examining sequential axial images to identify 'filling defects' in the pulmonary artery vasculature.[26] Filling defects refers to areas where the normal opacification (of contrast) in the lumen of the pulmonary arteries is disrupted.[26] This is clearly observed by the yellow arrows in Figure 1b. Our patient was treated with an anti-coagulant Rivaroxaban, an oral direct Factor Xa inhibitor in the coagulation cascade.[27] Common side effects include bleeding complications, cough, vomiting, and gasteroenteritis.[27]

We believe clinicians should focus on obtaining a thorough health history and a sustained vitals examination of athletes presenting with thoracic pain and exercise-induced dyspnea both in office and on the sideline. Specifically, clinicians should ask about family history of clotting disorders. Additionally, clinicians should ask athletes about type and usage of OC, if applicable. Secondly, clinicians should anticipate that vitals measurements for elite-

level endurance athletes may be significantly lower due to physiological adaptations to exercise. As such, when observing normal ranges in the general population in an athlete with symptoms of DVT or PE, clinicians should have a high index of suspicion of VTE when faced with clinical uncertainty. Typically, the symptoms of PE include tachycardia and hypoxemia.[2] Clinicians should also consider the combined risks of dehydration, high-intensity exercise, OC use, and heat exposure on VTE risk to educate athletes potentially better on heat-reduction strategies, hydration frequency, and the symptoms of VTE.

Summary

This case report details a unique case of VTE that presented in an elite 57-year-old Ironman triathlete following a long duration of travel. This patient was treated by a chiropractor one day prior to competition with symptoms of mechanical thoracic spine pain. During the swimming component of the triathlon, he was rescued, triaged, and transported to a nearby hospital and later diagnosed with multiple PE. This case report describes the unique risk factors of VTE in athletes for clinicians to consider along with the conditions of a non-musculoskeletal origin that can present with musculoskeletal manifestations. This case posed a diagnostic challenge from a clinical perspective as traditional risk stratification criteria may not be applicable to an athletic population. In summary, this case highlights the importance of a thorough health history, sustained vitals, and performing a cardiorespiratory examination that proved timely in assisting with this patient's pre-hospital management. While rare, VTE is a condition that can appear in athletes and should be considered as a differential diagnosis in athletes with exertional dyspnea, chest pain, and thoracic spine pain.

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