



Case Report

Pulmonary Embolism in Otherwise Healthy Athlete

Evgeny Shlyakhove¹, David Rott^{2,3*}

¹The University of Nicosia and St George's, University of London Medical School, Gower Street, London

²The Leviev Heart Centre, The Chaim Sheba Medical Centre, Tel Hashomer, Israel

³The Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

***Corresponding author:** David Rott, Cardiac Rehabilitation Institute, The Leviev Heart Centre, The Chaim Sheba Medical Centre, Tel Hashomer 52621, Israel

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Abstract

Pulmonary Embolism (PE) presents a spectrum of hemodynamic consequences, ranging from being asymptomatic to a life-threatening medical emergency. Risk factors of PE are usually related to one or more dimensions of Virchow triad: vessel's walls damage, stasis of the blood and states of increased coagulability. Some common examples of risk factors are genetic or acquired predisposition to blood clots formation, immobilization, hormonal contraception, pregnancy, obesity, increased age, malignancy, recent trauma or surgery. Although cases of PE in otherwise healthy athletes were described in the literature, vigorous physical exercise per se is not usually considered as a risk factor for PE. Here we describe a case of PE in an otherwise healthy woman, where excessive physical exercising appears to be a sole risk factor that could explain the development of venous thromboembolic disease.

Keywords: Morbidity; Pulmonary embolism; Risk factor; Vigorous exercising

Introduction

Pulmonary Embolism (PE) presents a spectrum of hemodynamic consequences, ranging from being asymptomatic to a life-threatening medical emergency [1]. PE caused by occlusion of pulmonary arteries via thrombus that is commonly formed inside deep veins of the lower extremities or pelvis. These thrombi detach from deep venous system, reach inferior vena cava and consequently the right heart, from there they are pumped out and lodged inside pulmonary arteries. The thrombi are formed out of fibrin mesh and erythrocytes trapped inside. Interestingly, there is an omission of platelet aggregation step beforehand [1]. While lodging of small thrombi may be completely asymptomatic, thrombus or thrombi that are large enough can cause significant acute symptomatology as a result of pulmonary compromise by uncoupling perfusion from ventilation in large portions of the lungs. The latter event also usually causes hemodynamic instability and is known in medical literature as "massive pulmonary embolism". Some examples of

possible symptoms are various degrees of chest pain, dyspnoea, loss of consciousness, low blood pressure, and increased heart rate and cough [1].

Predisposition to formation of thrombi inside deep veins is determined by one or more components of Virchow's triad: vessel's walls damage, stasis of the blood and states of increased coagulability. In other words, risk factors may include genetic or acquired predisposition to blood clots formation, immobilization, hormonal contraception, pregnancy, obesity, increased age, malignancy, recent trauma or surgery and more [1]. Most common screening tests for PE are Well's score and D-dimer, while diagnostic tests are Computed Tomography Pulmonary Angiography (CTPA) or ventilation to perfusion (V/Q) mismatch scintigraphy [1,2]. Mainstay of PE treatment consists of administering anticoagulation: Novel Oral Anticoagulants (NOACs), heparin, low molecular heparin or warfarin [1]. While, thrombolysis is reserved for treatment of massive PE [1]. Sometimes venous thromboembolic disease is present without any clear presence of previously mentioned risk factors. For example, cases of PE or Deep Venous Thrombosis (DVT) in otherwise healthy athletes. Here we describe a case of PE in an

otherwise healthy woman, where excessive physical exercising appears to be a sole risk factor that could explain the development of venous thromboembolic disease.

Case Report

A Forty-six-years-old generally healthy non-smoker woman, who is an avid long-time runner (including marathon), without any known VTE risk factors, presented to cardiology clinic complaining on dyspnea on exertion and decreased exercise capacity for the last couple of months. Echocardiography at rest has shown normal left ventricular ejection fraction of 60%, without left ventricular hypertrophy, without valve abnormalities with normal right ventricular size and function. Patients was referred to a stress echocardiogram: Upon 12 minutes of exercise stress testing according to Bruce protocol, the patient hasn't shown any symptoms, pulse was 159 bpm, without any signs of ischemia on electrocardiogram and echocardiography, showed overall improvement of cardiac function.

Cardiopulmonary Exercise Testing (CPET): Respiratory Exchange Ratio (RER) was 0.97, maximal oxygen consumption was 36.4 ml/min/kg which is equivalent to 142% of predicted for her age, anaerobic threshold was shown to be normal, and spirometry at rest has shown mild restrictive anomaly and mild ventilation to perfusion mismatch. Moreover, pulmonary function tests have shown irreversible obstructive abnormality. Consequently, the patient was referred for Computed Tomography Angiography (CTA), the latter had shown pulmonary emboli in arteries of the right lung. Treatment with rivaroxaban was commenced.

Discussion

Current report presents a case of PE in otherwise healthy 46-year-old female athlete, where vigorous exercising appears to be the sole risk factor. As our patient had denied any use of combined oral contraceptive pill, without any recent history of immobilization or long-time airplane or car travel, without any known family history of blood clotting disorders. Cases of PE in healthy athletes where described in the literature previously, [3-5]. However, it is important to emphasise that all these reports had possible alternative explanation for development of pulmonary embolism: use of combined contraceptive pill [3], previous DVT and prolonged immobilisation [4] and immobilisation during car trip [5]. Though in the latter case car trip was interrupted by short bouts of physical activity [5], still relative immobilisation as confounder couldn't entirely to be ruled out. Moreover, in our case there are no apparent confounders other than vigorous exercising which could explain VTE. As been discussed already in [5], strenuous exercising practiced by many athletes can predispose for venous thromboembolic disease through various possible mechanisms in consistence with dogma of Virchow's triad. For example, muscle hypertrophy may predispose for venous stasis, dehydration during exercise can lead to hemoconcentration and

potentially to hyper-coagulability, while increased blood flow can cause vascular micro-traumas through possible endothelial injury [5]. Interestingly, in our case the suspicion of PE was raised through results of CPET: VE/VCO₂ ratio was 37.2, respiratory reserve was 0.1%, and VO₂ in milliliter oxygen per kilogram weight was 36.4. In other words, VE/VCO₂ ratio was increased which could point towards decreased efficiency of ventilation or increase in dead space. Furthermore, respiratory reserve was almost lacking which could point to lung anomaly such as restrictive lung anomaly. In addition, evidence of mild ventilation to perfusion mismatch was observed via spirometry at rest. In consideration with all these findings of impairments in lung function the patient was sent for further diagnostic evaluation of PE via CTA. The latter confirmed diagnosis of PE and anticoagulation treatment was initiated. Curiously, RV function on echocardiography and many parameters of CPET in our patient were normal or even better than expected for average person like oxygen consumption which was higher than average (142% of predicted). This finding is striking, because usually there is an impairment in CPET parameters amongst PE patients and even in PE survivors [6]. This finding could be explained by excellent physical condition of our patient, as was shown [6], deranged CPET results in population of PE survivors stem mainly from physical de-conditioning, rather than from acquired cardiopulmonary impairment.

Apparently, physical exercising in moderation has many benefits for health and crucial for protection from morbidity and mortality [7]. However, exercising in excess may pose threats for overall health and increase risk for VTE [7]. For example, in one large study conducted in UK, women who practiced exercising for 2-3 times a week had decreased Relative Risk (RR) for VTE of 0.83 (0.79-0.87) with Confidence Interval (CI) of 95%, while women who practiced vigorous exercising every day had increased RR of 1.08 (0.99-1.17) for VTE with CI 95% [8]. Although, increased levels of smoking in every day exercising group may partially explain these results [8]. In conclusion, current case suggests that PE could potentially be caused by vigorous exercise. Importantly, strenuous physical exercising practices like those employed by professional athletes could pose a risk factor by itself for development of venous thromboembolic disease. Apparently, exercising up to a certain threshold is beneficial for overall health, however passing this threshold could potentially increase risk of morbidity. To get statistical significance, this notion should be further confirmed by additional studies with large number of cases.

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