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Bilateral Pulmonary Emboli in a Collegiate Gymnast: A Case Report

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Objective: To characterize the diagnosis of pulmonary embolism in collegiate student-athletes and to raise awareness among sports medicine providers of the possibility of this potentially fatal disease in the student-athlete population.

Background: An 18-year-old, previously healthy National Collegiate Athletic Association Division I female gymnast complained of intense pain, bilaterally, deep in her chest. The athlete was referred to her team physician, who identified normal vital signs but referred her to the emergency room because of significant pain. The student-athlete was diagnosed with bilateral pulmonary emboli in the emergency room.

Differential Diagnosis: Pneumonia, renal calculi, upper urinary tract infection, intercostal muscle strain or rib fracture, pancreatitis, gall bladder disease, gastritis, ulceration, esophagitis, infection, tumor, pulmonary embolism.

Treatment: The student-athlete was immediately placed on anticoagulants for 6 months. During that time, she was unable to participate in gymnastics and was limited to light conditioning.

Uniqueness: Documented cases of female student-athletes developing a pulmonary embolism are lacking in the literature. Two cases of pulmonary embolism in male high school student-athletes have been documented, in addition to many cases in elderly and sedentary populations.

Conclusions: All health care providers, including sports medicine professionals, should be aware that this condition may be present among student-athletes. During the initial evaluation, prescreening should include questions about any previous or family history of pulmonary embolism or other blood clots. Athletes who answer positively to these questions may have a higher likelihood of pulmonary embolism and should be referred for testing.

Key Words: cardiovascular diseases, vascular diseases

Pulmonary embolism (PE) is a leading cause of unexpected death in the United States.[1] Pulmonary embolism is estimated to cause 50 000 to 200 000 deaths every year[1]; however, only 2 cases of PE in the young athletic population have been documented. We present this case of PE in a collegiate female gymnast to inform sports medicine health care providers about this uncommon and life-threatening condition. This case is unique in that it is the only published record of a female athlete who has presented with a PE.

PERSONAL DATA AND CHIEF COMPLAINT

An 18-year-old (freshman) previously healthy National Collegiate Athletic Association Division 1 gymnast with no previous history or family history of pulmonary or cardiac conditions presented with upper right and upper left abdominal quadrant pain in the middle of the fall season. The athlete was evaluated during practice and was, therefore, warm and sweaty because of activity. She was visibly distressed and crying, indicating her frustration with a sharp pain in her chest and upper abdomen that was inhibiting her participation. The pain did not radiate or decrease with cessation of activity or change in position (standing, sitting, or lying). Her breathing was shallow because of pain and increased while lying in a prone or supine position. The athlete's only reported medication was an oral contraceptive (Yaz; Bayer HealthCare Pharmaceuticals, Wayne, NJ). The athlete had taken Yaz for irregular menstruation for the 3 months before her symptoms began. Questioning about her sexual activity revealed that pregnancy was not likely. Her pain began the night before her evaluation and was located in the upper right quadrant. At the time of the athletic trainer's evaluation, the team physician's on-campus office hours were ending. Because of the athlete's distress and pain symptoms, the athletic trainer did not want to wait until the next morning for a physician consult. The athlete was, therefore, immediately referred to the team physician.

PHYSICAL EXAMINATION AND MEDICAL HISTORY

The team physician evaluated the athlete promptly (within 10 minutes). Her blood pressure was on the low end of normal limits at 110/64, similar to the measurement on her preparticipation physical examination. Additional vital signs were also similar to those on the preparticipation physical examination, including a heart rate of 80 beats/min, respiratory rate of 28 breaths/min, and oral temperature of 36.6°C (97.88°F). Oxygen saturation by pulse oximetry was 99%, slightly lower than normal.

Upon physical examination, tenderness of the upper right and left abdominal quadrants was noted, in addition to rib cage tenderness with oblique and anterior compression. No organomegaly was identified. A cardiac examination indicated no irregular heart sounds and normal heart rate and rhythm. In addition, auscultation of the lungs was normal. The athlete's legs were palpated and a calf squeeze produced no findings of pain or knots. Based on the athlete's significant pain level and inconclusive evaluation, she was referred to the emergency room by the physician for additional evaluation. The team physician called ahead to apprise the emergency room staff of the referral and evaluation findings and to order blood

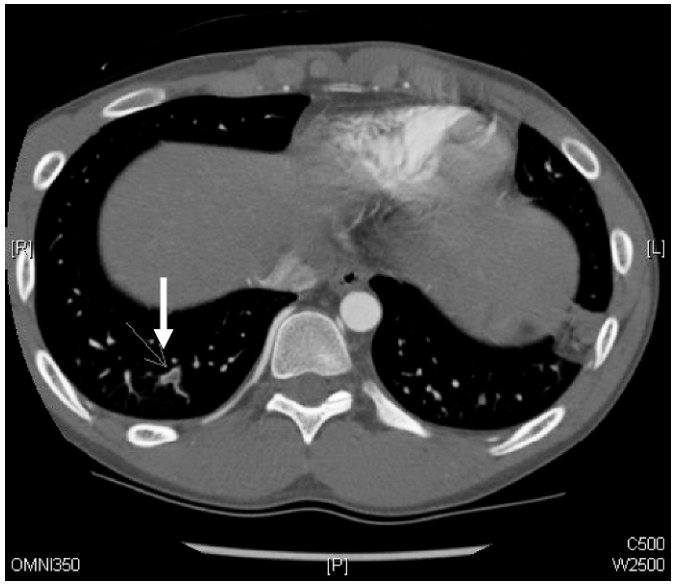

Figure 1. Computed tomography angiogram demonstrating segmental and subsegmental filling defects.

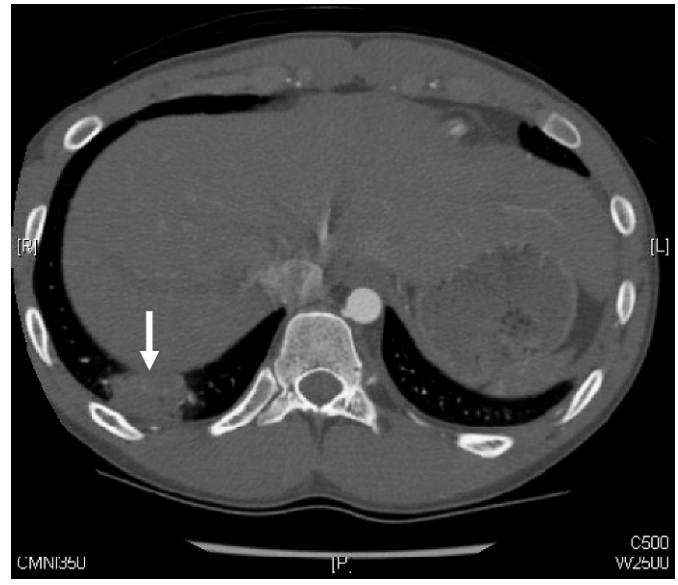

Figure 3. Computed tomography angiogram demonstrating left lower lobe infarction.

laboratory tests and chest radiographs. Abdominal radiographs and urinalysis were not requested at that time.

EMERGENCY ROOM EVALUATION AND LABORATORY STUDIES

The athlete was immediately transported to the emergency room by the athletic trainer in a golf cart, arriving within minutes. Emergency room vital signs evaluated during triage were unchanged from the physician's evaluation. No additional physical examination was conducted. An electrocardiogram indicated T-wave inversion in leads V1 and V2, which characterizes right ventricular outflow obstruction or volume or pressure overload and can reflect pulmonary emboli. Repeat electrocardiogram indicated normal rhythm, which may have accounted for the normal sinus rhythm upon auscultation. A chest radiograph suggested no abnormalities. A computed tomography angiogram (CTA) indicated bilateral basilar, segmental, and subsegmental filling defects consistent with PE (Figures 1 and 2). Wedge-shaped infarcts in the right lower lobe and left lower lobe were also identified, demonstrating that the clots were, in fact, obstructing blood flow (Figures 3 and 4).

Blood tests were conducted at this time, indicating an extremely elevated D-dimer concentration of 2566 ng/mL (normal is less than 451 ng/mL). An elevated D-dimer concentration is present when blood clot dissolution occurs. The athlete had a normal international normalized ratio (INR) prothrombin time and partial prothrombo-

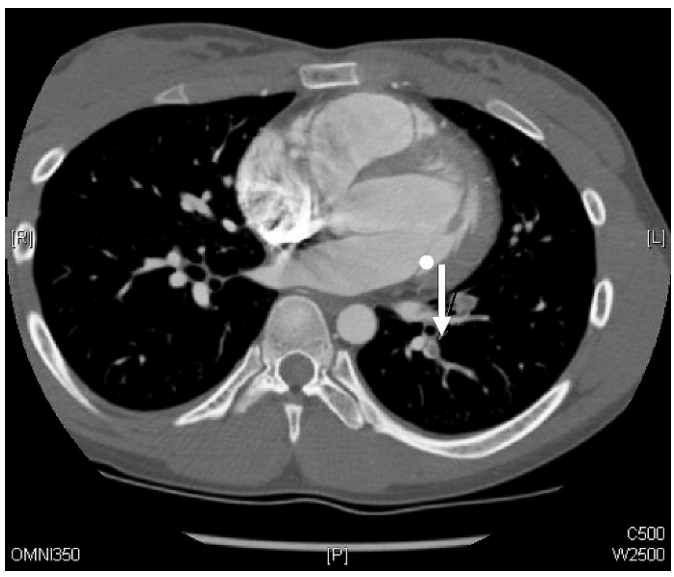

Figure 2. Computed tomography angiogram demonstrating filling defect.

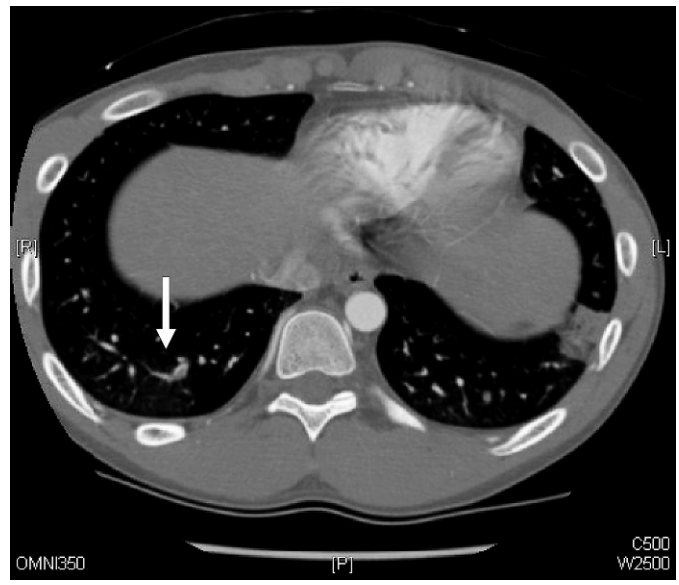

Figure 4. Computed tomography angiogram demonstrating left lower lobe blood clot.

plastin time, and the hypercoagulability evaluation, including homocysteine, antithrombin III, anticardiolipin antibody, prothrombin 20210A, factor V Leiden, and lupus anticoagulant levels, was within normal limits. The D-dimer test was the key to determining that a blood clot was present because all other tests were noted as normal. A complete blood count, urinalysis, and chemistry panel were unremarkable.

DIFFERENTIAL DIAGNOSIS

Possible alternative diagnoses based on symptoms included pneumonia, renal calculi (kidney stones), upper urinary tract infection, intercostal muscle strain or rib fracture, and numerous abdominal conditions, such as pancreatitis, gall bladder disease, gastritis, ulceration, esophagitis, infection, and tumor. After examination, the physician thought that the athlete's cardiac and pulmonary symptoms could be the result of pneumonia; however, because of the amount of pain the athlete was experiencing and her inability to attain a comfortable position, as well as her upper abdominal pain and pain upon palpation, the physician suggested kidney stones might be responsible.

The case history, physical examination, and laboratory results failed to substantiate a diagnosis of either pneumonia or kidney stones. Cardiovascular signs that can occur with PE include tachycardia, hypotension, palpitations, cyanosis, calculated shock index (pulse divided by systolic blood pressure) greater than 0.8, new onset of atrial fibrillation, cardiac murmur, and chest pain.[1–6] This athlete presented with very mild cardiac symptoms except that 1 of 2 electrocardiograms indicated right ventricular flow problems. Pulmonary symptoms of PE can include dyspnea, tachypnea, cough (may be nonproductive), hemoptysis, coarse breath sounds (rhonchi, wheezing, rales), and pleural rub.[1–6] Neurologic symptoms can include syncope; decreased level of consciousness or confusion, with or without seizures; confusion (decrease in orientation); and seizures.[1–6] Psychological symptoms associated with PE are apprehension and anxiety.[1–6] Integumentary symptoms that can occur with PE are diaphoresis and cyanosis,[1–6] and musculoskeletal symptoms consist of leg cramps and edema. Gastrointestinal symptoms include nausea and vomiting, which were absent in the athlete.[1–6] Other possible symptoms, such as fever and chills, were absent in the athlete[1–6] (Table 1). Other than pain, pain upon palpation, and anxiety, the athlete's symptoms were unremarkable. Her blood tests did not indicate any significant inherited markers (ie, factor V Leiden, antithrombin mutations, prothrombin mutations). The key to diagnosis in this athlete was an elevated D-dimer concentration in conjunction with an abnormal CTA scan demonstrating the presence of blood clots, which disrupted blood flow and caused pain.

Table 1. Prodromal Signs and Symptoms of Pulmonary Emboli by Body System[1–6]

Body System	Symptoms of Pulmonary Emboli
Cardiovascular	Tachycardia
	Hypotension (systolic blood pressure 100 mm Hg)
	Palpitations
	Cyanosis
	Chest pain (pleuritic, nonpleuritic, or nonretrosternal)
	Calculated shock index (pulse divided by systolic blood pressure) >0.8
	Gallop rhythm (S2 or S4)
	Increased S2 heart sound and widened P wave (widening of S2 heart sound over pulmonic region)
	New onset of atrial fibrillation
Pulmonary	Dyspnea (new or worsening chronic)
	Tachypnea
	Cough (may be nonproductive)
	Hemoptysis
	Coarse breath sounds (rhonchi, wheezing, rales)
	Pleural rub
Neurologic	Syncope/fainting
	Decreased level of consciousness/confusion with or without seizures
	Confusion (decreased orientation)
	Seizures
Psychological	Apprehension
	Anxiety
Integumentary	Diaphoresis
	Cyanosis
Musculoskeletal	Leg cramps
	Edema
Gastrointestinal	Nausea
	Vomiting
Miscellaneous	Fever
	Chills

TREATMENT COURSE

To quickly reabsorb the clots that had formed, the athlete was administered Lovenox (Sanofi-Aventis, Bridgewater, NJ) 60 mg, a low–molecular-weight heparin, subcutaneously every 12 hours, until her INR increased to 2 to 3 times normal. She was also prescribed warfarin, 2.5 mg orally on a daily basis for 6 months, to maintain her elevated INR. Increasing the athlete's INR was intended to drastically reduce the likelihood of developing further clots. While she was on blood thinners, she was unable to participate in any activity that might subject her to injury. Thus, she was limited to basic gymnastic skills and cardiovascular exercise. After the course of blood thinners ended, she was required to wait for 2 weeks, until the medicine was completely eliminated from her system, and then was allowed to return to full sport activity.

DISCUSSION

Only 2 other cases of PE have been documented in young athletes, and both were in high school-aged, male student-athletes.[2,7] This case study may present the only female athlete with PE described in the literature. Our athlete did not have any genetic predispositions based on history and blood tests. Environmentally, an oral contraceptive may have played a role in predisposing the athlete to PE; however, she did not display other risk factors associated with deep vein thrombosis or PE (Table 2).[3]

Environmental factors such as smoking, obesity, and hospitalization or bed rest typically are not factors in the athletic population, yet they must be reviewed during the history and initial evaluation. Oral contraceptives, travel above 3000 ft (914 m) or for longer than 4 hours, and pregnancy are factors that health care providers for a younger athletic population must investigate during a diagnostic evaluation. Pregnancy may induce PE, in part

Table 2. Pulmonary Emboli Risk Factors[3]

Risk Factor
Genetic risk factors
Hereditary thrombophilia
Deficiencies of protein C
Protein S
Antithrombin
Factor V Leiden
Prothrombin gene mutation
Disease risk factors
Heart disorders
Cancer
Sickle cell anemia
Kidney disease
Liver disease
Diabetes
Environmental risk factors
Smoking
Oral contraceptives
Surgery or trauma
Air travel of more than 3000 ft (914 m) or lasting longer than 4 h
Overweight/obesity
Bed rest or hospitalization
Pregnancy

because of the associated hypercoagulability and obstructed venous return by the enlarged uterus; therefore, it must be eliminated as a potential cause of PE. Oral contraceptives have also been identified as a risk factor, particularly within the first year of treatment.[3,8] In this case the athlete had just started contraceptive medication within 3 months, which may have played a role in development of the PE. Estrogen-containing contraceptives carry a small increased risk of PE that can be exacerbated in the presence of a genetic procoagulant mutation, such as factor V Leiden.[3,8,9] The use of estrogen-containing contraceptives is contraindicated in females with a history of thrombophilic states or inherited thrombophilias. Thus, females with a thrombophilic history should be tested for several inherited conditions, including factor V Leiden, prothrombin gene mutation, protein S deficiency, protein C deficiency, antithrombin deficiency, and dysfibrinogenemia (fibrin disorders) before estrogen-containing contraceptives are prescribed.[3,9] Factor V Leiden mutation accounts for 50% to 60% of thrombophilic cases.[3] Based on her history, the athlete did not have any inherited predisposition to thrombophilic events that would have signaled the need for testing, nor did she have a history suggesting pregnancy. Nonetheless, health care providers should understand and screen for inherited conditions, which, when coupled with oral contraceptive use, can create a high-risk scenario for PE or thromboembolism.[3,8]

We present the case of an athlete who developed 2 acute, submassive PEs. Pulmonary embolisms are categorized as either acute or chronic. Acute PE develops in a short period of time and either is treated and dissolves or results in mortality.[1] Chronic PE occurs when an initially acute PE seems to dissolve, but incompletely, resulting in smaller emboli that travel to the lungs.[1] Acute PE can be further categorized by the amount of pulmonary artery involvement, the presence or absence of a major predisposing factor, embolus mobility, or the interaction of PE size and underlying cardiovascular status.[1] *Massive* and *submassive* are the 2 subsets for PE based on pulmonary artery involvement. A massive PE arises when a pulmonary vasculature obstruction of more than 50% occurs or in more than 1 lobar artery[1] and is defined as shock or hypotension (systolic blood pressure of less than 90 mm Hg) or a blood pressure decrease of more than 40 mm Hg for longer than 15 minutes.[1] Unlike massive PE, submassive PE is characterized by right ventricular dysfunction without hemodynamic instability, which can be identified through electrocardiography.[1] Several undiagnosed submassive PEs can lead to a massive PE.[1] The specific type of PE may not be particularly important for initial evaluation and referral, but the underlying risk factors and primary evaluation provide evidence for the final diagnosis and, therefore, should be understood by the health care practitioner. The knowledge gained from proper differentiation will aid the health care provider in monitoring symptoms should the athlete develop a second PE.

The first published study on a high school wrestler with this condition involved a massive PE.[7] The second study was of a high school soccer player with many acute PEs of unknown origin.[2] We report on submassive acute PEs in both lower lobes of the athlete's lungs. Thus, despite the paucity of information in the literature, clinicians should be aware of the range of forms PE can take in the young student-athlete population.

Preventing PE is difficult for health care providers dealing with athletes, who tend to be young and healthy. A family history questionnaire during the preparticipation physical examination might allow the clinician to screen for predisposing genetic risk factors. Further testing to assess inherent conditions should be performed based on the initial screening, particularly for female athletes seeking oral contraceptives. Questions that may indicate predisposing factors include a personal or familial history of PE or deep vein thrombosis or a history of stroke or heart attack, all of which could be triggered by a blood clot. Athletes who have predisposing factors for PE should be aware of their risk and informed as to the signs and symptoms that may develop in the presence of a PE.

Three techniques for evaluating PEs have been described in the literature.[4,5] The first is based on the clinical examination and the likelihood of PE: the Wells ''Prediction Rule for Deep Vein Thrombosis'' (Table 3). Each factor for which the answer is *yes* adds 1 point; factors include active cancer, paralysis or immobilization, being recently bedridden for more than 3 days, swelling of the entire leg, and calf swelling.[4] A score of 3 or greater indicates a high chance of deep venous thrombosis. A second test for PE is the Geneva and Wells ''Prediction Rules for Pulmonary Embolism'' (Table 4). This test examines other factors, including age and previous history of PE or deep vein thrombosis.[4] Both prediction tests are performed by a physician whenever an athlete or patient has a suspected PE. Based on the prediction rules, the likelihood of PE for our athlete was not high.

The second method used to evaluate PE is diagnostic imaging: for example, ultrasonography. Ultrasonography has high specificity and sensitivity of diagnosing deep vein thrombosis of the lower extremity. In asymptomatic patients with a high probability of having the condition,

Table 3. Wells Prediction Rule for Deep Vein Thrombosis: Clinical Evaluation Table for Predicting Pretest Probability of Deep Vein Thrombosis[a]

Clinical Characteristic	Score
Active cancer (treatment ongoing, within previous 6 months or palliative)	1
Paralysis, paresis, or recent plaster immobilization of the lower extremities	1
Recently bedridden >3 days or major surgery within 12 weeks requiring general or regional anesthesia	1
Localized tenderness along the distribution of the deep venous system	1
Calf swelling 3 cm larger than asymptomatic side measured 10 cm below tibial tuberosity	1
Pitting edema confined to symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Alternative diagnosis at least as likely as deep vein thrombosis	−2

[a] A score of 3 or higher indicates *a high probability of deep vein thrombosis*; 1 or 2, *a moderate probability*; and 0 or lower, *a low probability*. In patients with symptoms in both legs, the more symptomatic leg is rated. Adapted from *The Lancet*, 350(9094), Wells S, Anderson DR, Bormanis J, et al,Value of assessment of pretest probability of deep-vein thrombosis in clinical management, 1795–1798, 1997, with permission from Elsevier. www.thelancet.com.

such as postoperative patients, specificity is maintained but sensitivity may be decreased.[4] Helical computed tomography scanning is another example of diagnostic imaging used for PE and deep vein thrombosis. A computed tomography scan combined with an elevated D-dimer level (despite the low likelihood of PE on the prediction scales) was used to correctly diagnose the athlete in the presence of unremarkable symptoms.

As a last diagnostic resort, pulmonary angiography can be performed. Pulmonary angiography is usually reserved as a measure of last resort because of high cost, invasiveness, and potential complications in patients with acute respiratory failure.[5] Pulmonary angiography was not conducted on our athlete because a conclusive image was obtained with computed tomography angiography. Side effects are most likely in patients who have had surgical complications (eg, difficulty with anesthesia).

The standard treatment of PE consists of a short regimen of heparin, followed by warfarin. However, the exact length of time patients must remain on an anticoagulant is unclear. Physicians typically make decisions on a case-by-case basis with appropriate monitoring. Some investigators have suggested 3 months; the risk of PE recurrence is higher within the first 3 months of treatment than after that time, regardless of treatment. Six months of drug treatment has also been suggested, but the risk of hemorrhaging during the anticoagulation treatment persists, and careful monitoring is required.[6] Longer regimens of anticoagulants are usually reserved for patients with many risk factors, especially recurring PE. The physician treating our athlete selected a 6-month course of anticoagulants as the best option for decreasing the likelihood of recurrent PE in this young, highly active person. To obtain the best outcome for the athlete, health care providers should communicate with the treating physician to monitor or deter activity as warranted.

Athletes prescribed anticoagulation medicines are at much greater risk for secondary damage resulting from

Table 4. Geneva and Wells Prediction Rules for Pulmonary Embolism[a]

Geneva	Points	Wells	Points
Previous pulmonary embolism or deep vein thrombosis	+2	Previous pulmonary embolism or deep vein thrombosis	+1.5
Heart rate >100 beats/min	+1	Heart rate >100 beats/min	+1.5
Recent surgery	+3	Recent surgery or immobilization	+1.5
Age, y		Clinical signs of deep vein thrombosis	+3
60–79	+1	Alternative diagnosis less likely than pulmonary embolism	+3
≥80	+2	Hemoptysis	+1
$PaCO_2$		Cancer	+1
<4.8 kPa (36 mm Hg)	+1		
4.9–5.19 kPa (37–38.9 mm Hg)	+4		
PaO_2			
<6.5 kPa (48.7 mm Hg)	+2		
6.5–7.99 kPa (48.7–55.0 mm Hg)	+1		
8–9.49 kPa (60–71.2 mm Hg)	+1		
9.5–10.99 kPa (71.4–82.4 mm Hg)	+1		
Atelectasis	0–4		
Elevated hemidiaphragm	5–8		
Clinical probability	≥9	Clinical probability	
Low		Low	0–1
Intermediate		Intermediate	2–6
High		High	≥7

Abbreviations: $PaCO_2$, partial pressure of carbon dioxide; PaO_2, partial pressure of oxygen (arterial).

[a] Adapted from *American Journal of* Medicine, 113(4), Chagnon I, Bounameaux H, Aujesky D, et al, Comparison of two clinical prediction rules and implicit assessment among patients with suspected pulmonary embolism, 269–275, 2002, with permission from Elsevier. http://www.amjmed.com.

injuries because normal blood clotting is compromised. Thus, a simple abrasion could cause excessive bleeding or hemorrhaging, bruising, and concussion. Athletes taking anticoagulation medication should not participate in any contact sport or activity in which they might collide with another person or forcefully hit the ground. In addition, the athlete should avoid any heavy lifting or training in which strains or sprains might occur. Low-level cardiovascular training, with little to no risk of falling, is permissible, as is low-level strength training. Treatment for PE can be effective but may end an athlete's season or athletic career.

CONCLUSIONS

Early diagnosis of PE is the key to appropriate treatment, decreased morbidity, and the likelihood of continued athletic participation. Health care providers should ensure proper screening and follow-up treatment if necessary and should monitor athletes diagnosed with PE to facilitate compliance. Physician involvement in diagnosis and continued treatment is essential and should align with sports medicine health care providers' policies and procedures regarding continuity of care and disqualification from participation. Future authors should investigate both the occurrence of PE in the athletic population and optimal treatment to ensure continued participation.

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