2016

PULMONARY EMBOLISM IN A COLLEGIATE SOCCER PLAYER

Jessica Paske

Valerie Moody
University of Montana

PULMONARY EMBOLISM IN A COLLEGIATE SOCCER PLAYER

By

JESSICA ROSE PASKE

Bachelors in Health and Human Performance, University of Montana, Missoula, MT, 2016

Associate of Arts, University of Montana- College of Technology, Missoula, MT, 2011

Professional Paper

presented in partial fulfillment of the requirements

for the degree of

Master's in Athletic Training

The University of Montana

Missoula, MT

May 2016

Approved by:

Scott Whittenburg, Dean of The Graduate School

Graduate School

Dr. Valerie Moody PhD, ATC, LAT, CSCS, Chair

HHP Department

Dr. Gene Burns Ed.D., Member

HHP Department

Melanie Dalpias MS, LAT, ATC

Outside Member- Work-Fit, Seattle, WA

PULMONARY EMBOLISM IN A COLLEGIATE SOCCER PLAYER

Dr. Valerie Moody PhD, ATC, LAT, CSCS, Chair
HHP Department

Dr. Gene Burns Ed.D., Member
HHP Department

Melanie Dalpias MS, LAT, ATC
Outside Member- Work-Fit, Seattle, WA

Background: Pulmonary embolism is a blood clot that occurs in the lungs, which decreases the oxygen levels in the body. Large or multiple pulmonary emboli can be fatal. This case involves a 20 year old female soccer player (goalkeeper) who was diagnosed with a pulmonary embolism during the middle of the regular season. Upon initial assessment, the athlete presented with soreness and redness over her left distal adductors after getting cleated during practice a few days earlier. The initial assessment was adductor tendinitis and treated conservatively. Subsequently, the area became hot, red, and painful and the athlete was removed from practice. Eventually signs and symptoms resolved and the athlete returned to full participation. Several weeks later, the athlete presented with right sided chest pain and visited the campus health center. **Differential diagnosis**: Musculoskeletal pain, pericarditis, pleuritis. **Treatment**: The athlete was referred to the emergency room after blood work was performed. The athlete was told she could not exercise for at least three months. During this time, she was placed on anticoagulants. After the season ended, the athlete was told she could no longer play contact sports after a CT scan revealed pulmonary embolism. **Uniqueness**: Typically, patients with pulmonary embolism will present with chest pain, shortness of breath, and hypoxia. In addition, the incidence of pulmonary embolism is extremely rare in young, healthy athletes with no significant medical history**. Conclusion**: Although most patients with pulmonary embolism have had surgery or are elderly and generally unhealthy, the majority tend to recover. These patients tend to have recurrent pulmonary emboli in the future after their primary embolism. In a young, healthy population, factors that increase the risk of developing pulmonary embolism are cancer, fractures of the hip or leg, oral contraceptives, major surgery, acute medical illness, obesity, or a sedentary lifestyle. It is critical that athletic trainers recognize early signs and symptoms of pulmonary embolism which warrants emergency management. **Word Count**: 316

Table of Contents

Poster Submitted for District 10 Northwest Athletic Trainers Association Symposium

Pulmonary Embolism in a Collegiate Soccer Player

Jessica Paske, Master's Athletic Training Student
Valerie Moody, PhD, ATC, LAT, CSCS, WEMT-B
The University of Montana, Missoula, Montana, U. S. A.

INTRODUCTION

Pulmonary embolism (PE) is a blood clot that blocks a lung artery creating damage to that part of the lung and can lead to hypertension. PE can also cause lower oxygen levels in the blood, damage other organs due to low blood supply, and even death. Pulmonary embolism is generally caused from a blood clot formed in the vein of a leg and travels to the lungs.

PE is commonly found in individuals who have had surgery, are generally unhealthy, or are elderly. The majority of these patients tend to recover.

In a young, healthy population, factors that increase the risk of developing pulmonary embolism are cancer, fractures of the hip or leg, oral contraceptives, major surgery, acute medical illness, obesity, or a sedentary lifestyle. There have only been a few documented cases of competitive athletes sustaining a pulmonary embolism. The occurrence of PE is often times due to the clotting protein mutation some individuals have.

Factor V Leiden is a mutation that is a risk factor for venous thromboembolism, because it makes it harder for the anticlotting proteins to break up the factor V. Although both men and women can have factor V Leiden, women have an increased tendency to develop blood clots while pregnant or taking hormone estrogen. The majority of people who have factor V Leiden never know because they never exhibit any signs or symptoms. A blood clot may be the first indication of factor V Leiden. Anticoagulants are a common medication for individuals who have factor V Leiden and have developed blood clots.

UNIQUENESS

Typically, patients with pulmonary embolism will present with chest pain, shortness of breath, and hypoxia. In addition, the incidence of pulmonary embolism is extremely rare in young, healthy athletes with no significant medical history.

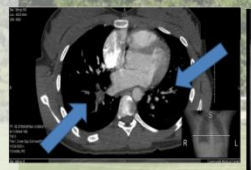
Lab results showed, both alleles contain factor V Leiden. Therefore, the athlete is unable to continue playing contact sports.

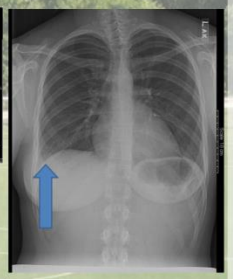
CASE REPORT

A 19-year-old female National Collegiate Athletic Association Division I soccer player (goalie) presented in the athletic training clinic, during pre-season with soreness and redness over her left distal adductors reporting that she had been cleated during a camp she had coached at. The athlete had equal strength, but side to side movement was painful. She was tender to palpate over her distal adductors. Medical history revealed that the athlete was taking oral contraceptive pills daily, had no recent travel, no recent long car trips, was not a smoker, and had no family history of cardiac conditions or blood clots. The initial assessment was adductor tendinitis and she was treated conservatively.

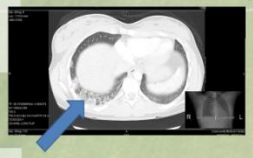
Myofascial release was the only therapy that provided any relief of the pain, besides rest. Subsequently, the area became hot and red for a few days. During this time, the athlete was still removed from participation and was icing the area that was hot and red area. Eventually signs and symptoms resolved and the athlete returned to full participation.

INITIAL IMAGING





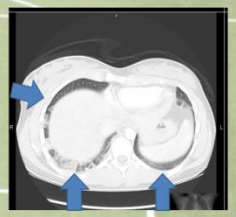
Above: CT scan showing emboli in the left lower lobe and a filling defect in the central right lower lobe pulmonary artery extending into all of the subsegmental



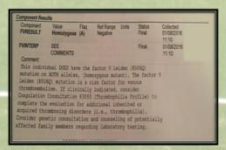
Above: X-Ray demonstrating vague right lower lobe parenchymal opacity

Left: CT scan showing peripheral infiltrate in the left posterior basilar right lower lobe

FOLLOW UP IMAGING



Right: CT scan showing mild residual parenchymal opacity peripherally in the right lung base and interval resolution bilateral pulmonary thromboembolitic disease



Follow up lab testing revealing the Factor V Leiden mutation

CASE REPORT CONTINUED

A few weeks later, the athlete went in one morning to the health center on campus. She was having right sided chest pain and was in obvious discomfort. The exam reported the athlete's vital signs were all within normal limits, her lungs were clear to auscultation with good air movement, there were no rales or wheezing. The athlete was non-tender to palpate along lower right ribcage, no pain reproduced with palpation, her abdomen was soft, not distended, and her upper right quadrant was non-tender to palpate.

The physician at the campus health center gave a 60 mg IM injection of Toradol which offered no relief to the athlete. A chest x-ray showed early signs of pneumonia. A D-dimer was ordered as well, because the symptoms were not equivalent with pneumonia symptoms. The findings were inconclusive that it was truly a pulmonary embolism and the athlete was referred to the emergency department for an evaluation at a hospital in town.

In the emergency department, the athlete was diagnosed with acute pulmonary embolism on the right side. She was given oxycodone-acetaminophen for pain and XAREL(rivaroxaban) twice daily for 21 days.

After three months, the athlete went back to the hospital for a CT Chest Angiography. The CT showed resolution to the pulmonary thromboembolic disease and no evidence of any new pulmonary emboli. The athlete was informed that she could return to full participation which included running, lifting, and soccer practices.

A month later at a follow up appointment at the hospital, the athlete had additional blood work performed which determined that she had a factor V Leiden mutation on both alleles. The factor V Leiden mutation is a risk factor for venous thromboembolism. Because factor V Leiden mutation increasing her chance of a blood contact after possibly getting hit, the athlete was forced to end her soccer career early. However, the athlete is still leads an active lifestyle with basic running and weightlifting.

DISCUSSION

Early recognition of signs and symptoms is key for appropriate treatment and decreasing morbidity in athletes.

Health care providers should provide proper health screenings to help find athletes with high prevalence of PE.

University of Montana
ATHLETIC TRAINING
Rising Above

Introduction:

Pulmonary embolism (PE) is a blood clot that blocks a lung artery creating damage to that part of the lung and can lead to hypertension.[1] PE can also cause lower oxygen levels in the blood, damage other organs due to low blood supply, and even death. Pulmonary embolism is generally caused from a blood clot formed in the vein of a leg and travels to the lungs.[1]

Pulmonary embolisms can be categorized as either chronic or acute.[2] An acute pulmonary embolism develops in a short period of time. They are either treated and dissolves away or result in mortality.[2] A chronic PE occurs, when an acute PE is unable to completely dissolve.[2] There are two subsets of acute PE that are categorized by the presence or absence of a major predisposing factor, embolus mobility, the size, and the pulmonary involvement. These subsets are submassive and massive.[2] When the right ventricular dysfunctions without hemodynamic instability, which is identified through an electrocardiography. If several submassive pulmonary emboli go undiagnosed, they can lead to a massive pulmonary embolism. A massive PE occurs when blood pressure decreases more than 40 mm Hg for longer than 15 minutes, systolic blood pressure less than 90 mm Hg, and pulmonary vasculature obstruction of more than 50 percent.[2] Knowing what kind of pulmonary embolism the individual had can help health care professionals monitor vital signs and symptoms and reduce the risk of the individual getting more PE in the future.

PE is commonly found in individuals who have had surgery, are generally unhealthy, or are elderly.[1] The majority of these patients tend to recover. In 25% of people with PE, their first symptom is sudden death.[3,4] Approximately one third of the patients that do recover tend to have recurrent pulmonary emboli within ten years after their primary embolism.[3] In a young, healthy population, factors that increase the risk of developing pulmonary embolism are cancer, fractures

of the hip or leg, oral contraceptives, major surgery, acute medical illness, obesity, or a sedentary lifestyle. There have only been a few documented cases of competitive athletes sustaining a pulmonary embolism.[5] The occurrence of PE is often times due to the clotting protein mutation some individuals have.

Factor V is a clotting protein found in the body.[6] There are many anti-clotting proteins that help to break the factor V, to help it from forming clots where they are not needed. Factor V Leiden is a mutation that is a risk factor for venous thromboembolism, because it makes it harder for the anticlotting proteins to break up the factor V.[6] Most people with the mutation never develop abnormal blood clots. However, some develop blood clots that can lead to long-term health problems or become life-threatening. Although both men and women can have factor V Leiden, women have an increased tendency to develop blood clots while pregnant or taking hormone estrogen. The majority of people who have factor V Leiden never know because they never exhibit any signs or symptoms.[6] A blood clot may be the first indication of factor V Leiden.[6] Anticoagulants are a common medication for individuals who have factor V Leiden and have developed blood clots.

CASE REPORT

A 19-year-old female National Collegiate Athletic Association Division I soccer player (goalie) presented in the athletic training clinic, during pre-season with soreness and redness over her left distal adductors reporting that she had been cleated during a camp she had coached at. The athlete had equal strength, but side to side movement was painful. She was tender to palpate over her distal adductors. Medical history revealed that the athlete was taking oral contraceptive pills daily, had no recent travel, no recent long car trips, was not a smoker, and had no family

history of cardiac conditions or blood clots. The initial assessment was adductor tendinitis and she was treated conservatively. Over the next few days, many therapies were attempted: corrective exercises, Graston technique, massage, dry needling, and myofascial release. Myofascial release was the only therapy that provided any relief of the pain, besides rest. Subsequently, the area became hot and red for a few days. During this time, the athlete was still removed from participation and was icing the area that was hot and red area. Eventually signs and symptoms resolved and the athlete returned to full participation. The athlete mentioned later that she had felt intense pain with certain movements during practices that would bother her. She always felt that she could work through this pain.

A few weeks later, the athlete went in one morning to the health center on campus. She was having right sided chest pain and was in obvious discomfort. The day before the athlete reported having the same pain that started in her upper right chest, worked its way into the lower right chest along her ribs. The athlete took 800 mg of ibuprofen the night before with no relief. The exam reported the athlete's vital signs were all within normal limits, her lungs were clear to auscultation with good air movement, there were no rales or wheezing. The athlete was non-tender to palpate along lower right ribcage, no pain reproduced with palpation, her abdomen was soft, not distended, and her upper right quadrant was non-tender to palpate.

The physician at the campus health center gave a 60 mg IM injection of toradol which offered no relief to the athlete. A chest x-ray showed early signs of pneumonia. A D-dimer was ordered as well, because the symptoms were not equivalent with pneumonia symptoms. A D-dimer or Fibrin Degradation Fragment is a lab test that assists in ruling out deep vein thrombosis, pulmonary embolism, and stroke.[7] It may be ordered when the patient presents with sudden shortness of breath, labored breathing, coughing, lung-related chest pain, and/or a rapid heart

rate.[7] A positive D-dimer test indicates abnormally high levels of fibrin degradation products in the blood.[7] The findings were inconclusive that it was truly a pulmonary embolism and the athlete was referred to the emergency department for an evaluation at a hospital in town.

In the emergency department, the athlete was diagnosed with acute pulmonary embolism on the right side. She was given oxycodone-acetaminophen for pain and XAREL(rivaroxaban) twice daily for 21 days. The athlete was instructed to return to the emergency room if she produced a fever, cough, bloody cough, worsening chest pain, or difficulty breathing. The athlete was informed to remain on anticoagulants for three months and removed from practices.

After three months, the athlete went back to the hospital for a CT Chest Angiography. The CT showed resolution to the pulmonary thromboembolic disease and no evidence of any new pulmonary emboli. The athlete was informed that she could return to full participation which included running, lifting, and soccer practices. A month later at a follow up appointment at the hospital, the athlete had additional blood work performed which determined that she had a factor V Leiden mutation on both alleles. The factor V Leiden mutation is a risk factor for venous thromboembolism. Because factor V Leiden mutation increasing her chance of a blood contact after possibly getting hit, the athlete was forced to end her soccer career early. However, the athlete is still leads an active lifestyle with basic running and weightlifting.

References:

1. What is Pulmonary Embolism? Website. https://www.nhlbi.nih.gov/health/health-topics/topics/pe. Updated July 1, 2011. Accessed March 10, 2016.
2. English J. B. Prodromal signs and symptoms of a venous pulmonary embolism. *Medsurg Nurs*.2006;15(6):352–356
3. Venous Thromboembolism (Blood Clots). Website. http://www.cdc.gov/ncbddd/dvt/data.html. Updated June 22, 2015. Accessed March 10, 2016.
4. The Surgeon General's Call to Action to Prevent Deep Vein Thrombosis and Pulmonary Embolism. Website. http://www.ncbi.nlm.nih.gov/books/NBK44181/ Updated 2008. Accessed March 10, 2016.
5. Kahanov L, Daly T. Bilateral Pulmonary Emboli in a Collegiate Gymnast: A Case Report. *Journal of Athletic Training.* 2009; 44(6): 666-671.
6. Diseases and Conditions Factor V Leiden. Website. http://www.mayoclinic.org/diseases-conditions/factor-v-leiden/basics/definition/con-20032637. Updated July 14, 2015. Accessed March 10, 2016.
7. D-dimer. Website. https://labtestsonline.org/understanding/analytes/d-dimer/tab/test/ Updated October 29, 2015. Accessed March 10, 2016.

Provisions

The preceding pages contain the Professional Paper Proposal, and do not reflect any changes made to the design or execution of the project. The subsequent pages contain the final manuscript for submission, and are inclusive of the changes to the project and reflect the updated execution of the study.

Introduction:

Pulmonary embolism (PE) is a blood clot that blocks a lung artery creating damage to the lung and can lead to hypertension.[1] PE can also cause lower oxygen levels in the blood, damage other organs due to low blood supply, and death. Pulmonary embolism is generally caused from a blood clot formed in the vein of a leg and travels to the lungs.[1]

Pulmonary embolisms can be categorized as either chronic or acute.[2] An acute pulmonary embolism develops in a short period of time. They are either treated and dissolve away, or result in mortality.[2] A chronic PE occurs when an acute PE is unable to completely dissolve.[2] There are two subsets of acute PE that are categorized by the presence or absence of a major predisposing factor, embolus mobility, size, and pulmonary involvement. These subsets are submassive and massive.[2] A submassive pulmonary embolism occurs acutely without system hypotension (systolic blood pressure greater than 90 mm Hg), but with either myocardial necrosis (disorganized breakdown of heart tissue) or right ventricular (RV) dysfunction without a change in hemodynamic instability (abnormal or unstable blood pressure).[2,3] This can only be identified through electrocardiography.[2] Myocardial necrosis occurs if the elevation of troponin I is greater than 0.4 ng/mL or the elevation of troponin T is greater than 0.1 ng/mL.[3] Troponin I and T are specific and sensitive indicators of damage to the myocardium.[4] Right ventricular dysfunction presents in one of the following ways: right ventricle systolic dysfunction on echocardiography, right ventricle dilation on a computerized tomography (CT scan), elevation of B-type natriuretic peptide (BNP) greater than 90 pg/mL, elevation of N-terminal pro-BNP (greater than 500 pg/mL), or electrocardiographic changes (new complete or incomplete right bundle-branch block, anteroseptal ST elevation or depression, or anteroseptal T-wave inversion).[3] All are markers that are proven to be useful in diagnosing cardiovascular disorders.

If several submassive pulmonary emboli go undiagnosed, they can lead to a massive pulmonary embolism. A massive PE occurs when blood pressure decreases more than 40 mm Hg for longer than 15 minutes, systolic blood pressure less than 90 mm Hg, pulmonary vasculature obstruction of more than 50 percent, or requires inotropic support (medicines that change the force of the heart's contractions) not due to a cause other than a pulmonary embolism (arrhythmia, hypovolemia, sepsis, left ventricular dysfunction, pulselessness, or persistent profound bradycardia (heart rate less than 40 bpm with signs of shock).[2,3] Knowing the type of pulmonary embolism, the individual can help health care professionals monitor vital signs and symptoms and reduce the risk of recurring PE.

Pulmonary embolism is commonly found in individuals who have had surgery, are generally unhealthy, or are elderly.[1] The majority of these patients tend to recover. In 25% of people with PE, their first symptom is sudden death.[5,6] Approximately one third of the patients that do recover tend to have recurrent pulmonary emboli within ten years after their primary embolism.[5] In a young, healthy population, factors that increase the risk of developing pulmonary embolism are cancer, fractures of the hip or leg, oral contraceptives, major surgery, acute medical illness, obesity, or a sedentary lifestyle. There has only been one documented case of a collegiate athlete sustaining a pulmonary embolism.[7] The occurrence of PE is often times due to the clotting protein mutation some individuals possess.

Factor V is a clotting protein found in the body.[8] There are many anti-clotting proteins that help to break the factor V, to help it from forming clots where they are not needed. Factor V Leiden is a mutation that is a risk factor for venous thromboembolism because it makes it harder for the anticlotting proteins to break up the factor V.[8] Most people with the mutation never

develop abnormal blood clots. However, some develop blood clots that can lead to long-term health problems or become life-threatening. Although both men and women can have factor V Leiden, women have an increased tendency to develop blood clots while pregnant or taking the hormone estrogen. Factor V Leiden allele is present in about five percent of Caucasian individuals, nearly absent in Africans, Asians, and races with Asian ancestry such as Amerindians, Eskimos, and Polynesians.[9] It is present in eighteen percent of Caucasian patients with venous thrombosis. Conversely, factor V Leiden is usually not found in non-Caucasian thrombotic patients.[9] The majority of people who have this mutation never know because they never exhibit any signs or symptoms.[8] A blood clot may be the first indication of factor V Leiden.[8] Anticoagulants are a common medication for individuals who have factor V Leiden mutation and have developed blood clots.

Case Report:

A 19-year-old female National Collegiate Athletic Association Division I soccer player (goalie) presented in the athletic training clinic with soreness and redness over her left distal adductors being cleated during a camp. The athlete had equal strength, but side to side movement was painful. She was tender to palpate over her distal adductors. Medical history revealed that the athlete was taking oral contraceptive pill Larin Fe daily, had no recent travel, no recent long car trips, was not a smoker, and had no family history of cardiac conditions or blood clots. The initial assessment was adductor tendinitis. She was treated conservatively. Over the next few days, many therapies were attempted: corrective exercises, Graston technique, massage, dry needling, and myofascial release. Myofascial release was the only therapy that provided any relief of the pain, besides rest. Subsequently, the area became hot and red for a few days. During this time, the athlete was still removed from participation and was icing the hot and red area.

Eventually, signs and symptoms resolved and the athlete returned to full participation. The athlete mentioned later she had felt a bothersome, intense pain with certain movements during practices. She always felt she could work through this pain.

A few weeks later, the athlete went to the health center on campus. She was having right sided chest pain and was in obvious discomfort. The athlete reported having the same pain the day before in her upper right chest, but it worked its way into the lower right chest along her ribs. The athlete took 800 mg of ibuprofen the night before with no relief. The exam reported the athlete's vital signs were all within normal limits, her lungs were clear to auscultation with good air movement, and there were no rales or wheezing. The athlete was non-tender to palpate along lower right ribcage, no pain reproduced with palpation, her abdomen was soft, not distended, and her upper right quadrant was non-tender to palpate.

The physician at the campus health center gave a 60 mg IM injection of toradol which offered no relief to the athlete. A chest x-ray showed early signs of pneumonia, shown in Figure 1. A D-dimer was ordered as well because the symptoms were not equivalent with pneumonia symptoms. A D-dimer or Fibrin Degradation Fragment is a lab test that assists in ruling out deep vein thrombosis, pulmonary embolism, and stroke.[10] It may be ordered when the patient presents with sudden shortness of breath, labored breathing, coughing, lung-related chest pain, and/or a rapid heart rate.[10] A positive D-dimer test indicates abnormally high levels of fibrin degradation products in the blood.[10] The findings were inconclusive for pulmonary embolism and the athlete was referred to the emergency department for an evaluation at a local hospital.

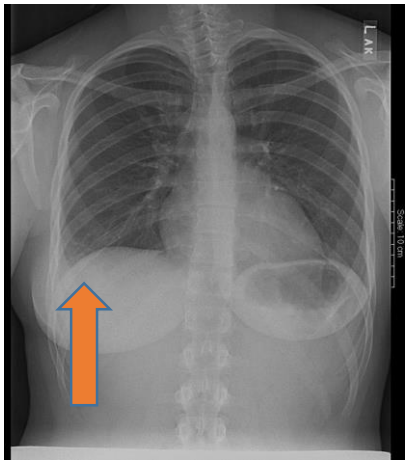


Figure 1. Vague right lower lobe parenchymal opacity

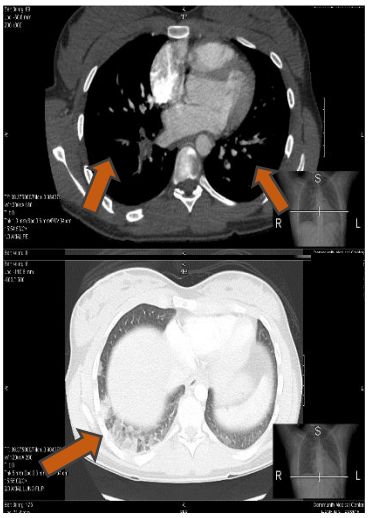


Figure 2. Top- Emboli in the left lower lobe and a filling defect in the central right lower lobe pulmonary artery extending into all of the subsegmental. Bottom- Peripheral infiltrate in the left

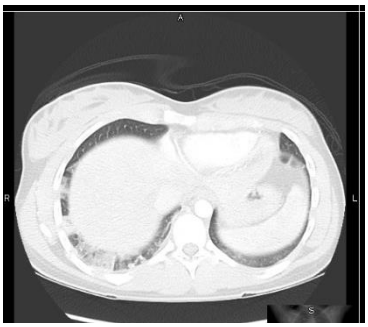


Figure 3. Mild residual parenchymal opacity peripherally in the right lung base and interval resolution bilateral pulmonary thromembolitic disease

In the emergency department, the athlete was diagnosed with acute pulmonary embolism on the right side. She was given oxycodone-acetaminophen for pain and XAREL (rivaroxaban) twice daily for 21 days. The athlete was instructed to return to the emergency room if she produced a fever, cough, bloody cough, worsening chest pain, or difficulty breathing. Figure 2 illustrates the computerized tomography (CT) scans that were performed that show pulmonary embolism in both the right and left lungs. Because of the PE, the athlete was informed to remain on anticoagulants for three months and was removed from practices.

After three months, the athlete went back to the hospital for a CT Chest Angiography. The CT showed resolution to the pulmonary thromboembolic disease and no evidence of any new pulmonary emboli as shown in Figure 3. The athlete was informed she could return to full participation which included running, lifting, and soccer practices. A month later, at a follow up hospital appointment, the athlete had additional blood work performed which determined she had a factor V Leiden mutation on both alleles. The factor V Leiden mutation is a risk factor for venous thromboembolism. Because factor V Leiden mutation increases her chance of a blood clot after possibly getting hit, the athlete was forced to end her soccer career early.

To this day, the athlete still leads an active lifestyle with basic running and weightlifting. The athlete has learned to be cautious and aware of how she is feeling while working out. She stays active to promote good blood flow, but is unable to play any contact sports. Future lifestyle considerations for this athlete now include taking precautions while traveling (baby aspirin and compression socks), avoiding prescriptions or medications that contain estrogen, and obtaining regular blood work if placed on blood thinners again or wants to start having children.

Discussion:

There are several documented cases of PE in young athletic populations. The first pulmonary embolism case report was in 1979 in a high school wrestler.[11] The athlete reported flu like symptoms (fever, nausea, weakness, and myalgias) two days prior to a match.[11] After the match, he was having trouble breathing for 30 minutes. Four days later, he competed again, this time being pinned; he became cyanotic and oxygen was administered.[11] The next day his physician found a heart murmur after he complained of left scapular pain.[11] The doctor also found that the gas pressure and pH in his heart were elevated enough that he needed heparin therapy. With no resolve, the athlete underwent median sternotomy bypass and total cardiopulmonary bypass.[11] Emboli of unknown age were removed from both pulmonary arteries. The athlete was discharged from the hospital and received warafin sodium therapy.[11] The athlete did fully recover, but it is not documented if he continued to play sports. In 2007, a high school male soccer player with no apparent risk factors had a sudden onset of dyspnea with no discernable cause.[12] This athlete had an elevated D-dimer, but all lab results were within normal limits.[12] The athlete had recurrent pulmonary emboli. The athlete was treated with the placement of a Greenfield inferior vena cava filter (prevent life-threatening pulmonary emboli). Two and a half years later, the athlete subsequently discontinued all anticoagulation therapy and remains

asymptomatic. It is not documented if the athlete continued to participate in contact sports. The last documented case of a pulmonary embolism in a high school athlete occurred in 2015.[13] The athlete had exertional dyspnea for a year and a half. She was diagnosed with a massive PE.[13] In 2009, the first collegiate athlete with PE was documented.[7] She was a gymnast with no previous history or family history of pulmonary or cardiac conditions and she presented with upper right and left quadrant pain.[7] Her symptoms became so bad that, in less than five minutes, she would experience presyncope. Presyncope is a state consisting of lightheadedness, muscular weakness, blurred vision, and feeling faint. Because of these feelings she decided to go to a clinic. After further evaluation, she was referred to the team physician who referred her to the emergency room.[7] She had an elevated D-dimer and all other blood tests were within normal limits. After a course of blood thinners, the athlete was allowed to return to full participation.

Young, healthy athletes often times will not present with the classic symptoms associated with PE. Early diagnosis of pulmonary emboli is critical, but vague symptoms make it difficult to recognize. A differential diagnosis such as musculoskeletal pain, pleuritic, pericarditis, hyperventilation, and lung trauma should be considered when a patient presenting with abdominal pain, fever, productive cough, new onset of atrial fibrillation, hyperventilation, tachycardia, bradycardia, course breath sounds, dyspnea, or tachypnea (common symptoms found with pulmonary embolism).[7,11,14] Athletes rarely sustain a PE, but delayed treatment could be fatal.

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 pulmonary emboli. Screening for factor V Leiden mutation in individuals with no history of blood clots is currently recommended for individuals who are relatives of patients identified as carriers.[9] There

is no data available to justify screening all athletes for the mutation; however, health care providers should take into consideration that oral contraceptives, pregnancy, or high-risk surgery increase the chance of pulmonary emboli.[9]

Our soccer athlete may be the first athlete to sustain a career ending pulmonary embolism. Furthermore, she is the only case that presented with a positive factor V Leiden mutation and a non-elevated D-dimer. The athlete did not have any cardiac or pulmonary history, therefore there was no need for blood testing prior to this event. Therefore, as healthcare providers, we should be asking questions about family history or previous history of pulmonary embolism. Athletes who answer yes should receive further screening because they may have a higher likelihood of pulmonary embolism.

Conclusion:

Pulmonary embolism is not commonly found in athletes; few cases have been reported. In this case, when the adductor injury became red and hot, referral was warranted. Although, common symptoms of deep vein thrombosis (DVT) are swelling, warm skin, red or discolored skin, or tired legs, DVTs are not commonly found in the adductors.[15] Because the initial evaluation was diagnosed as adductor tendinitis, the possible deep vein thrombosis was missed. Making sure that health care professionals know the signs and symptoms of DVT and PE is important in making sure athletes are seeking proper medical treatment. If an athlete does complain of chest pain, difficulty breathing, or differences in normal heart beats, they should be referred to the team physician or emergency room. An early diagnosis of these symptoms is key for appropriate treatment and decreases the likelihood of mortality. Health care providers should

ensure proper screening and follow-up treatment, as well as monitor diagnosed athletes with PE to facilitate compliance.[7]

References:

1. What is Pulmonary Embolism? Website. https://www.nhlbi.nih.gov/health/health-topics/topics/pe. Updated July 1, 2011. Accessed March 10, 2016.
2. English J. B. Prodromal signs and symptoms of a venous pulmonary embolism. *Medsurg Nurs*.2006;15(6):352–356
3. Jaff, McMurtry, Archer, Cushman, Goldenberg, Goldhaber, Jenkins, Kline, Michaels, Thistlethwaite, Vedantham, White, Zierler. Management of Massive and Sumbmassive Pulmonary Embolism, Iliofemoral Deep Vein Thrombosis, and Chronic Thromboembolic Pulmonary Hypertension. *Circulation.* 2011; 123: 1788-1830.
4. Medscape: Troponins. Website. http://emedicine.medscape.com/article/2073935-overview. Updated January 14, 2015. Accessed April 21, 2016.
5. Venous Thromboembolism (Blood Clots). Website. http://www.cdc.gov/ncbddd/dvt/data.html. Updated June 22, 2015. Accessed March 10, 2016.
6. Diseases and Conditions Factor V Leiden. Website. http://www.mayoclinic.org/diseases-conditions/factor-v-leiden/basics/definition/con-20032637. Updated July 14, 2015. Accessed March 10, 2016.
7. D-dimer. Website. https://labtestsonline.org/understanding/analytes/d-dimer/tab/test/ Updated October 29, 2015. Accessed March 10, 2016.
8. The Surgeon General's Call to Action to Prevent Deep Vein Thrombosis and Pulmonary Embolism. Website. http://www.ncbi.nlm.nih.gov/books/NBK44181/ Updated 2008. Accessed March 10, 2016.

9. Kahanov, Daly. Bilateral Pulmonary Emboli in a Collegiate Gymnast: A Case Report. *International Journal of Athletic Therapy and Training.* 2009; 44(6): 666-671.
10. Diseases and Conditions Factor V Leiden. Website. http://www.mayoclinic.org/diseases-conditions/factor-v-leiden/basics/definition/con-20032637. Updated July 14, 2015. Accessed March 10, 2016.
11. Stefano, Chiusolo, Paciaroni, Leone. Epidemiology of Factor V Leiden: Clinical Implications. *Seminars in Thrombosis and Hemostasis.* 1998; 24(4): 367-379.
12. D-dimer. Website. https://labtestsonline.org/understanding/analytes/d-dimer/tab/test/ Updated October 29, 2015. Accessed March 10, 2016.
13. Croyle, Place, Hilgenberg. Massive Pulmonary Embolism in a High School Wrestler. *The Journal of the American Medical Association.* 1979; 241(8): 827-828.
14. Moffatt, Silberberg, Gnara. Pulmonary Embolism in an Adolescent Soccer Player: A Case Report. *Med. Sci. Sports Exerc.* 2007; 39(6): 899-902.
15. Larsen, Ball. Chronic Pulmonary Embolism in a Young Athletic Woman. *Baylor University Medical Center Proceedings.* 2007; 28(3): 371
16. Pulmonary Embolism Clinical Presentation. Website. http://emedicine.medscape.com/article/300901-clinical#b3. Updated October 9,2015. Accessed April 20, 2016.
17. Symptoms and Tests for DVT. Website. http://www.webmd.com/dvt/deep-vein-thrombosis-dvt-symptoms-diagnosis. Updated 2016. Accessed April 20, 2016.