Exertional Dyspnea, Orthopnea, and Tachycardia: A Case Report describing a Pulmonary Embolus Misdiagnosed as Congestive Heart Failure

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ABSTRACT:
Pulmonary embolism presentation can often be insidious in nature and may present like any other cardiopulmonary condition. Symptoms may present classically with a history of immobilization, followed by sudden onset dyspnea, chest pain, and hemoptysis. In a more likely scenario, they will present atypically and symptoms will progress gradually. This case report describes a 71 year old gentleman, with a history of aortic valve stenosis and metastatic prostate cancer presenting with progressive dyspnea, orthopnea, and fatigue. He presented to the emergency department and hospital 3 times in the course of 2 months. Initially he was diagnosed with congestive heart failure secondary to aortic valve stenosis. On subsequent visits, he was treated for decompensated heart failure, without ever investigating a possible alternative cause for his symptoms. It was not until he was referred for a transcatheter aortic valve replacement, or TAVR, that a chest CT with contrast was performed to reveal extensive bilateral pulmonary emboli. This case illustrates the importance maintaining a high index of suspicion for pulmonary embolism, and of avoiding bias when evaluating the same patient multiple times for similar symptoms.

INTRODUCTION:
Pulmonary thromboembolism has been called “the great masquerader” and with good reason. They are easily diagnosed when occurring with a V/Q scan, but in the absence of a DVT, they are easily missed. It occurs in 6 to 70 of 100,000 patients, although this is considered a low estimate. “Silent” or asymptomatic pulmonary embolism in people with DVT occurs around 50% of the time, and autopsies have revealed only 30-45% of PE are diagnosed prior to death. The risk is substantially higher in patients with malignancy, with venous thromboembolism (VTE) occurring in as many as 40% of cancer patients. High mortality is associated with high risk PE, (86-65%), and early detection is key. These patients present with shock and cardiovascular collapse. Lower risk pulmonary embolism, i.e. patient is stable on presentation, and mortality of 5-25% are usually asymptomatic. The classic triad of symptoms are sudden onset of dyspnea, pleuritic chest pain, and hemoptysis. Unfortunately these only present together 5%-7% of the time. Occasionally a PE will present atypically with progressive shortness of breath, productive cough, wheezing, fevers, abdominal pain, fever, hemoptysis, flank pain and syncope. Signs of PE are not any more helpful. Classically, it is tachypnea, tachycardia, rales, and accentuated second heart sound, occasionally new onset atrial fibrillation. Tachypnea occurs in 96% of the cases, all other signs occur at least 60% of the time. The classic triad of symptoms are sudden onset of dyspnea, pleuritic chest pain, and hemoptysis. Unfortunately these only present together 5%-7% of the time. Occasionally a PE will present atypically with progressive shortness of breath, productive cough, wheezing, fevers, abdominal pain, fever, hemoptysis, flank pain and syncope.

Several different criteria have been created to help with diagnosis, but none has been shown to be very specific or sensitive. The Geneva and Wells criteria give a score to determine probability. A moderate probability deserves an initial D dimer, which is very sensitive and will rule out VTE, but is not specific. If there is a high probability or an elevated D dimer a chest CT with contrast or V/Q scan is done to determine presence of pulmonary embolus. CT is the test of choice, but in the presence of contraindications, a V/Q scan can be done - but it is not as sensitive or specific.

CASE DESCRIPTION:
71 year old male presented to the emergency room for worsening non-productive cough, shortness of breath, paroxysmal nocturnal dyspnea, and 2 to 3 times near syncopal episodes associated with his cough. These symptoms had progressively been getting worse over the last month. He also complained of progressive weakness, decreased exercise tolerance, and recent intermittent shortness of breath, which he thought to be secondary to his cough. He denies fevers, chills, nausea, or peripheral edema. He did have a recent hospital admission approximately 6 weeks prior for decompensation with congestive heart failure secondary to aortic valve stenosis. On physical exam his vitals were heart rate 104, blood pressure 114/63 respiratory rate 17, temperature 98.5 F, and oxygen saturation 98% on 2 l nasal cannula. He was visibly distressed, dyspneic with conversation. No jugular venous distention was noted in the supine position. He had a 3/6 crescendo/decrescendo murmur, lung sounds were distant, and there was no pericardial edema. All other physical exam findings were within normal limits.

In addition to his CHF and aortic stenosis his past medical history was significant for insulin dependent diabetes with retinopathy, hyperlipidemia, metastatic prostate cancer to the bones. He had sought treatment for his cancer with chemotherapy and radiation. Last radiation treatment had been 8 weeks prior, currently on Lupron injections, with a normal PSA. Family history was non-contributory. He denied tobacco or alcohol use. He was on multiple medications: Novol 70/30 sliding scale, lisinopril 5mg, metoprolol 25mg, furosemide 20mg, omeprazole 20mg, aspirin 81mg, loratidine prn, multivitamin, vitamin D, iron supplements, and Omega 3 fish oil. Reported allergy to amoxicillin, his stated reaction was a rash.

On physical exam his vitals were heart rate 94, respiratory rate 17, temperature 98.5 F, and oxygen saturation 98% on 2 l nasal cannula. He was visibly distressed, dyspneic with conversation. No jugular venous distention was noted in the supine position. He had a 3/6 crescendo/decrescendo murmur, lung sounds were distant, and there was no pericardial edema. All other physical exam findings were within normal limits.

He then presented in Tucson for his pre-operative appointment for the TAVR procedure. A routine chest CT with contrast was done, per protocol. On this exam he was found to have extensive bilateral pulmonary emboli. He was given warfarin, his procedure was cancelled, and he was discharged home on 4 liters of oxygen.

He returned to the emergency department 5 days later for chest pain and worsening shortness of breath. Because he had a diagnosis of decompensated heart failure and was scheduled to be seen in Tucson for his procedure he was sent home from the emergency department without further evaluation. He then returned to Tucson for his pre-operative appointment for the TAVR procedure. A routine chest CT with contrast was done, per protocol. On this exam he was found to have extensive bilateral pulmonary emboli. He was given warfarin, his procedure was cancelled, and he was discharged home on 4 liters of oxygen.

CONCLUSION:
It is difficult for clinicians not to have ‘tunnel vision’ when approaching a patient. If a reason for the complaint is found, that is what is treated without further investigation. It is important to approach each patient, even ‘frequent fliers’ with a fresh perspective, even when they were not expected to improve according to the recommended treatment plan.

This patient was a victim of this bias. He was referred for TAVR, and was not expected to improve until his aortic stenosis was corrected. As a result, every time he was presented with worsening symptoms, no other possible cause was investigated. If PE had been considered, the commonly used tools would have put him at moderate risk, which would have led to further investigation. With PE, he would have scored 4.5 points for tachycardia and PE being equally as likely as other diagnosis. The Geneva criteria put him a moderate risk also, for his age and tachycardia. Unfortunately his recent diagnosis of decompensated heart failure led to bias, and his pulmonary embolus was likely missed, not once, but twice.

On review of his hospital stays, during his first hospitalization, pulmonary embolus was considered and a D dimer was ordered, which was negative. All other laboratory and diagnostic studies supported congestive heart failure. On his return visit to the hospital, his symptoms were very similar, and pulmonary embolism was not considered. In retrospect there was some notable differences. His second hospitalization he required increasing amounts of oxygen. He came into the ED requiring 2 liters, and was discharged on 4 liters oxygen. Prior to this, he did not require supplemental oxygen, even during his previous hospitalization. He was also complaining of new symptoms - near syncope and pleuritic chest pain. He also was tachypneic which is the most common presenting sign, and tachycardic. All of these should have been investigated further.

One week after his pulmonary embolus was diagnosed, he again presented to the local emergency department, this time after a fall and because of an extensive epistaxis. Head CT was done, it was negative for cerebral hemorrhage, but the local ENT was called. There was a nosebleed that was packed, and he was sent home. He was back in the emergency room 2 days later because the nose bleed had not stopped. Again it was packed, but because of his PE they were unable to stop the warfarin.

Unfortunately, the next day he was found in his car after having committed suicide. Unfortunately his recent diagnosis of decompensated heart failure would have changed the outcome of this case. He had been led to believe and was hopeful his TAVR procedure would help resolve his symptoms, when it was canceled because of a different diagnosis, pulmonary embolus, this likely led to disappointment and ultimately hopelessness which caused him to take his life.

Patient’s deserve a fresh perspective every time they present, regardless of past diagnoses and treatment.

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REFERENCES:
2. “Pulmonary Embolism”. Michael S Boesen, MD, MBA, FACEP Kevin Garg, MD., Eugene C Lim, MD, Robert E Uccomber, MD, MPH, Gary Setnik, MD, Eric J Stein, MD, Sara Churchill, MD, MD, FAC,PC, Francisco Talavera, PharmD, PhD, Gregory Tino, MD, Medscape, Oct 9,2015
3. “Pulmonary embolism, part I: Epidemiology, risk factors and risk stratification, pathophysiology, and case series” in Diagnostic pulmonary embolism”. Jan Birnbaum, MD PhD, Vladimir Dmitrov MD, and Aled Llubiner, MD PhD, Medicus, Inc. 2008-2012.