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The Night Sweats and Pulmonary Embolism-controversial Presentation and Differentiation

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Abstract

Pulmonary embolism (PE) is a common and potentially fatal condition where early and accurate diagnosis can significantly reduce mortality and morbidity. It typically presents with a range of associated symptoms such as breathlessness at rest or during exertion, coughing, discomfort or swelling in the legs, and wheezing. However, many atypical symptoms are also possible, including epigastric pain, hypotension, and night sweats, with mechanisms not yet understood. The absence of definitive signs and symptoms makes diagnosing pulmonary embolism challenging. We report the case of a 56-year-old male who presented to the ER with right-sided chest pain following laparoscopic umbilical and bilateral inguinal hernia repair 6 days prior. Workup revealed a PE, for which the patient was started on apixaban. Following the initiation of apixaban, the patient developed drenching night sweats, which, after extensive workup, were determined to be a diagnosis of exclusion.

Keywords: Diagnosis, Heart failure, Night sweats, Pulmonary embolism

Introduction

Pulmonary embolism (PE) is a frequent and potentially deadly cardiovascular disease caused by the migration of deep venous thrombi to one or more pulmonary arteries. The incidence of PE is approximately 60 to 120 per 100,000 people per year [1].

The obstruction of blood flow in the pulmonary arteries leads to various physiological disruptions that can significantly impact respiratory and cardiac function.

The immediate consequence of a clot obstructing the pulmonary artery is reduced blood flow to the alveoli, leading to a ventilation-perfusion (V/Q) mismatch. Parts of the lung may be ventilated but not perfused, resulting in impaired gas exchange. This can manifest clinically as hypoxemia, contributing to the symptoms of dyspnea and hyperventilation observed in patients with PE. Additionally, the obstruction increases pulmonary vascular resistance, which can lead to pulmonary hypertension and right ventricular strain. The increased resistance in the pulmonary circulation due to a PE places an excessive burden on the right ventricle, leading to right ventricular (RV) pressure overload and potential RV failure. Acute RV dilation, as a response to pressure overload, can impair left ventricular filling by altering the interventricular septal position, further compromising cardiac output and leading to systemic hypotension and shock in severe cases.

Diagnosing pulmonary embolism (PE) is difficult, particularly when it displays unusual symptoms like severe night sweats defined as excessive sweating during sleep that is not induced by a hot sleeping environment-"sleep hyperhidrosis". This delay in identification can result in apostponement of proper treatment. Although commonly associated with infectious, oncologic, or endocrine disorders, night sweats have been reported as the primary symptom of PEs in the literature [2].

The aim of detailing this case is to enhance comprehension of the varied clinical spectrum of pulmonary embolism (PE) and underscore the importance of maintaining a high level of suspicion for its uncommon presentations. This will be achieved by discussing the diagnostic process, treatment strategies, and outcomes through an analysis of this unique case study.

Case Presentation

A 56-year-old male presented to the emergency room with a one-day history of right-sided chest pain. He was in his usual state of health the night before when he started having right-sided chest pain that was localized to the right lower chest and radiated to the right abdomen and flank exacerbated by inspiration, causing difficulty breathing. It is worth mentioning that he had laparoscopic umbilical and bilateral inguinal hernia repair 6 days prior, and his perioperative and postoperative course was uneventful. He was not bedbound and was ambulating. The review of systems was significant for the mild discomfort at the incision site but was negative for fever, chills, rigors, cough, palpitations, sick contacts, sore throat, dizziness, headache, dysuria, hematuria, nausea, vomiting, abdominal pain, and change in bowel movement. He had a past medical history of left-sided spontaneous pneumothorax 3 years back that was managed conservatively, exercise-induced ventricular tachycardia. He used to work as an emergency medical technician (EMT). On presentation, he was normothermic and hemodynamically stable with a heart rate (HR) of 104 beats per minute (bpm), blood pressure at 149/98 (115) mmHg, respiratory rate (RR) of 24 breaths per minute, and saturating 92% on room air. Initial blood work revealed leukocytosis with a white blood cell (WBC) count of 14.7 x 10^9/L, hemoglobin (Hgb) level of 14.8 g/dL, a D-dimer level of 742 ng/mL (<318 ng/mL), a normal troponin level of 0.00 ng/mL (0.00-0.02 ng/mL), red blood cell (RBC) count of 4.99 x 10^12/L, hematocrit (HCT) of 44.3%, mean corpuscular volume (MCV) of 88.8 fL, and a platelet count of 188 x 10^9/L. Urinalysis (UA) and liver profile were within normal limits. EKG showed sinus tachycardia **(Figure 1)**.

Computed tomography pulmonary angiography (CTPA) demonstrated evidence of segmental pulmonary emboli (PE) in the right lower lobe alongside bilateral areas of atelectasis. Due to recent surgery and abdominal radiation of the pain, CT of the abdomen and pelvis (CT A/P), was done which showed post-operative changes in the abdomen and pelvis and focal soft tissue in the right inguinal canal **(Figure 2)**.

The patient was admitted and was started on supplemental oxygen and apixaban 10 mg twice daily for 7 days and decreased to 5mg daily later on. A venous duplex scan was done that ruled out deep vein thrombosis. Transthoracic echocardiogram (TTE) showed an ejection fraction of 60-65% with normal RV function. He was discharged with a cardiology follow-up. After the discharge, the patient reported new-onset drenching night sweats. The patient had no history of night sweats. He denied any fever, rigors, chills, chronic cough, weight loss, palpable lymph nodes, recent travel to a tuberculosis-endemic country, or use of Selective serotonin reuptake inhibitors (SSRIs). CT scans done during hospitalization ruled out any enlarged lymph nodes, cavitary lung lesions, or occult malignancy. TTE ruled out the presence of any valvular vegetation. Hence, given the negative workup, these drenching night sweats were attributed to the inflammatory response generated by the breakdown of the clot following the use of apixaban.

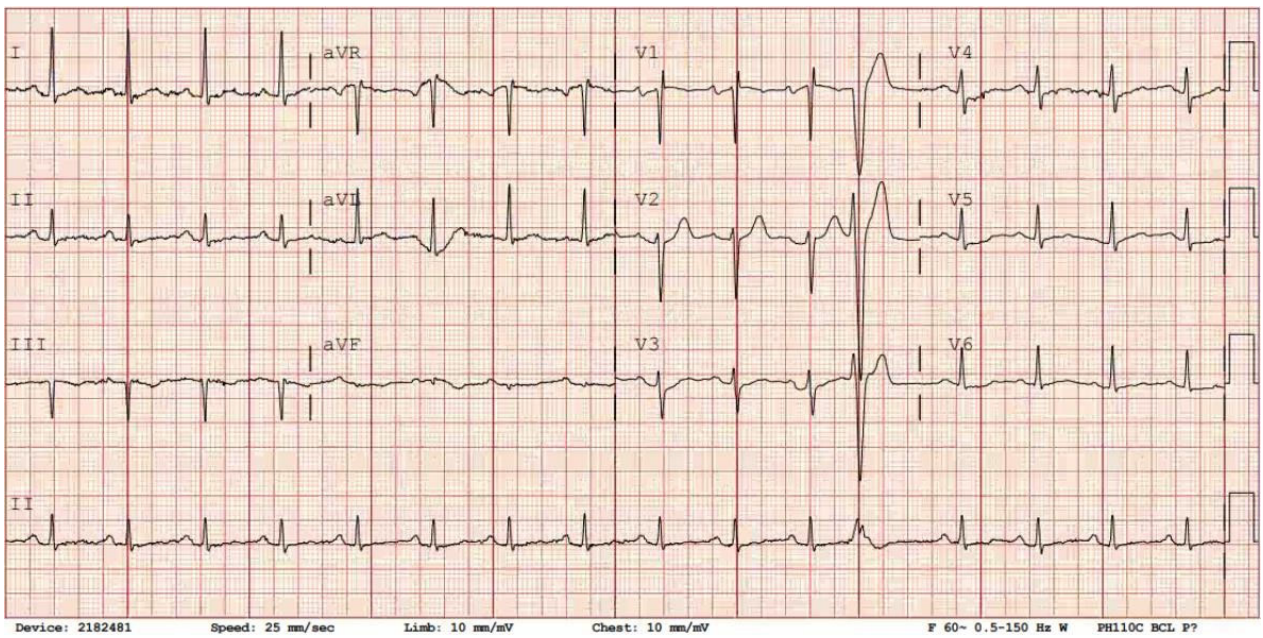

Figure 1. EKG showing sinus tachycardia.

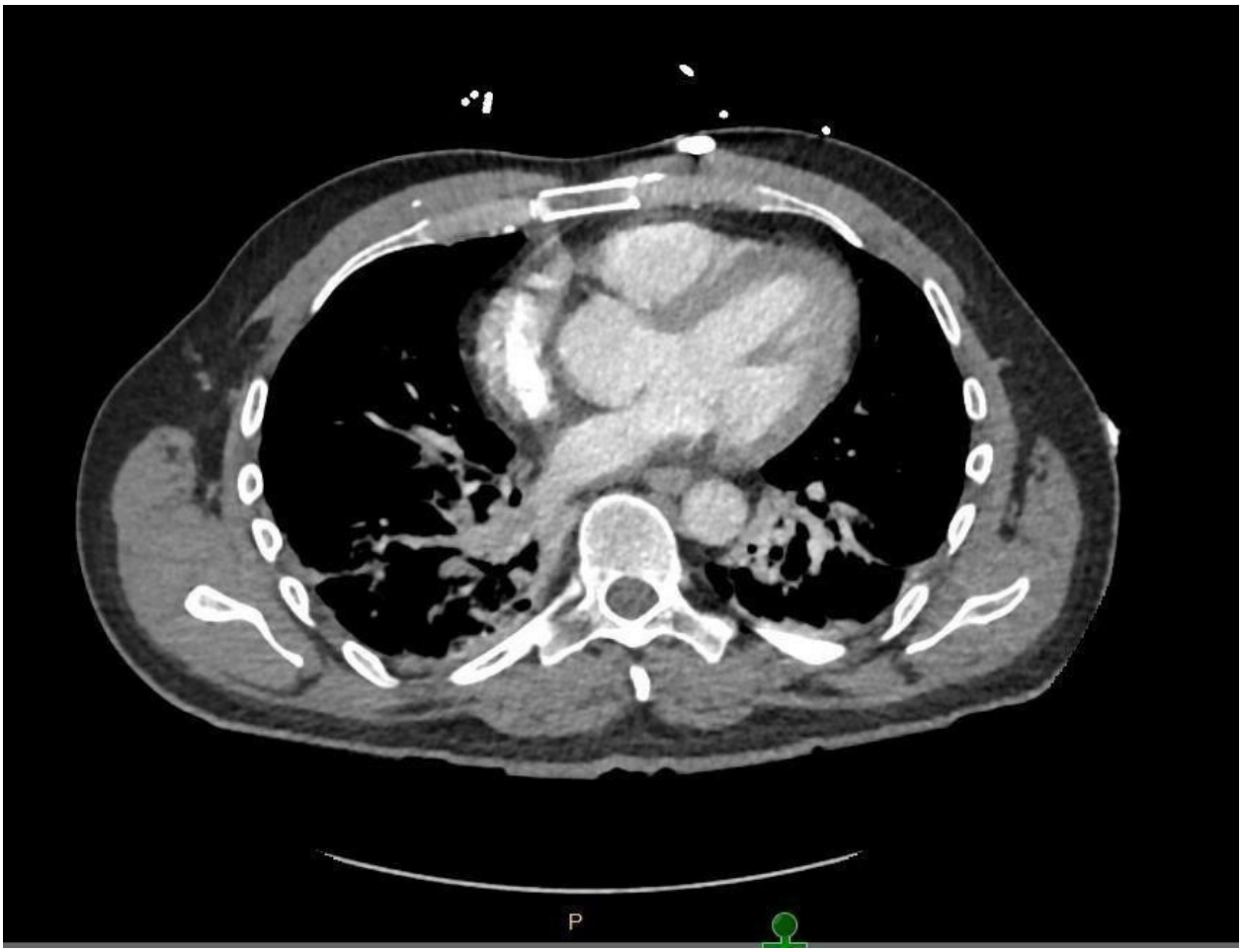

Figure 2. CTPA revealing pulmonary embolism in subsegmental pulmonary arterial branchesof the right lower lobe.

Discussion

The drenching night sweats, characterized by an excessive outpouring of perspiration during sleep, presents a vexing symptom with multifaceted origins. Night sweats, though seemingly benign, can be a precursor of underlying systemic conditions ranging from infectious diseases to malignancies and endocrine disorders. The systematic exclusion of these potential causes is crucial for an accurate diagnosis. One common cause of drenching night sweats is infections, particularly bacterial infections like tuberculosis (TB) or endocarditis, and viral infections such as HIV/AIDS or influenza. These infections can trigger fevers and night sweats as part of the body's immune response. Hormonal imbalances, notably during menopause in women or due to conditions like hyperthyroidism (overactive thyroid), can also lead to night sweats. Certain medications, including antidepressants or hormone therapy drugs, are known to cause night sweats as a side effect. Night sweats may sometimes be associated with underlying malignancieslike lymphomas, which can produce fever and night sweats as part of systemic symptoms.

Importantly, pulmonary embolism can also be associated with night sweats. The resolution of athrombus involves a complex interplay of cellular activities, inflammatory responses, and fibrotic remodeling. PE can trigger a systemic inflammatory response and neutrophils, and monocytes invade into the thrombus. Neutrophils facilitate early thrombus resolution by promoting fibrinolysis and collagenolytic and release proinflammatory factors like IL-1β and TNF-α [3]. The release of proinflammatory cytokines can alter the hypothalamic temperature regulation center. This could lead to alterations in body temperature regulation, manifesting as night sweats. Another possible theory is that the obstruction of the pulmonary arteries leads to reduced blood oxygenation (hypoxemia), which the body can perceive as a stressor. The resultant stress response activates the hypothalamic-pituitary-adrenal (HPA) axis,affecting cortisol levels and the autonomic nervous system, further disrupting thermal regulation [4]. Another potential mechanism for PE-associated night sweats could be the direct stimulation of sweat glands by cytokines as eccrine sweat glands have been reported to express receptors for most PE-associated cytokines. PTE as a cause

of night sweats is considered a diagnosis of exclusion due to the rarity of night sweats as a symptom of this condition. It becomes a possiblediagnosis only after more common causes of night sweats have been systematically excluded through comprehensive testing and evaluation [5].

In medical literature, the earliest documented case of pulmonary embolism associated with night sweats was reported in The Lancet in 1899. This case described a patient who developed apulmonary embolism following abdominal surgery and subsequently experienced night sweats [6]. Similarly, a more recent case report from 2015 documented a patient with night sweats, despite having no fever, who was diagnosed with pulmonary embolism [7]. Another case report from 2019 highlighted a patient presenting with night sweats and hemoptysis who was found tohave a pulmonary embolism [8]. These reports underscore the diverse and sometimes subtle ways in which pulmonary embolism can manifest, including the occurrence of night sweats as a possible symptom.

Conclusion

Night sweats, historically considered nonspecific, may be an early or concurrent symptom of pulmonary embolism (PE), suggesting the need for further investigation. Documented cases show diverse PE presentations, emphasizing the importance of clinical suspicion for unexplainednight sweats, especially without fever or infection. Prospective studies are needed to understand the prevalence and diagnostic significance of night sweats in PE, as current literature is sparse.

Increased awareness among healthcare providers and appropriate use of diagnostic tools can enhance PE recognition, improve patient outcomes, and inform better management of thromboembolic diseases. Further research is crucial to refine knowledge and optimize patientcare.

Author Contributions Statement

Azka Naeem; Writer, Resident assigned to the patient, Literature Review.

Vahagn Tamazyan; Review, Resident assigned to patient, Obtained the images.

Muhammad Hashim Khan; Literature review, final manuscript preparation.

Vijay Shetty: Attending to the patient, Critical review.

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