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(CASE REPORT)

Asymptomatic bradycardia as a manifestation of intermediate high-risk pulmonary embolism

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Abstract

Progressively worsening dyspnea as an initial manifestation of an intermediate high-risk pulmonary embolism (PE) is a readily reported life-threatening cardiac emergency. Atypical presentation of PE may make the initial diagnosis challenging and is often the reason it is missed by clinicians. A 60-year-old woman with asymptomatic bradycardia presented with shortness of breath and dizziness. On examination, she was noted to have a heart rate of 40 beats per minute (bpm) which slightly improved to 55 bpm after intravenous (IV) fluids were administered. A transthoracic echocardiogram (TTE) showed evidence of severe right ventricular strain. Chest computed tomography angiogram (CTA) confirmed PE. After percutaneous aspiration thrombectomy, she was transitioned to direct oral anticoagulant (DOAC) from a heparin infusion and discharged on oral anticoagulation. Notably, her hospital course was marked by labile cycles between normotension and hypertension, with blood pressures exceeding 200/100 mmHg. The possibility that hypertensive states may prevent hemodynamic deterioration and a resulting high-risk pulmonary embolism is considered in patients presenting with multiple risk factors for thromboembolism.

Keywords: Pulmonary; Embolism; Bradycardia; Hypertension; Intermediate; Risk

1. Introduction

Notorious for its diverse and often nonspecific presentations, pulmonary emboli have proven difficult to diagnose in a timely manner. As such, thorough and numerous case descriptions are warranted in order to more completely detail the full breadth and scope of variances in clinical presentations. Recent trends have been encouraging; from 1998 to 2006, identification of pulmonary emboli doubled in the United States without any significant changes in mortality [1]. As technology advances and understanding of disease parameters continues to deepen, wisdom over which lesions warrant more urgent attention and intervention is likely to continue to grow - the ultimate goal being that this highly feared event will in the near future achieve remarkable levels of survival and recovery.

2. Case Presentation

A 60-year-old woman with a history of type 2 diabetes mellitus, hypertension, depression, obesity, and active daily tobacco use presented to the emergency department with progressively worsening dyspnea that had started the day prior. On admission, she was noted to be hypertensive with a blood pressure of 160/88 mmHg and hypoxic with a pulse oximetry of 88% on room air. The patient denied any associated chest pain or palpitations but admitted a dry and productive cough. She was subsequently placed on 4L of supplemental oxygen via nasal cannula. CTA chest with IV contrast was remarkable for extensive bilateral pulmonary artery emboli with evidence supporting the presence of right ventricular (RV) strain.

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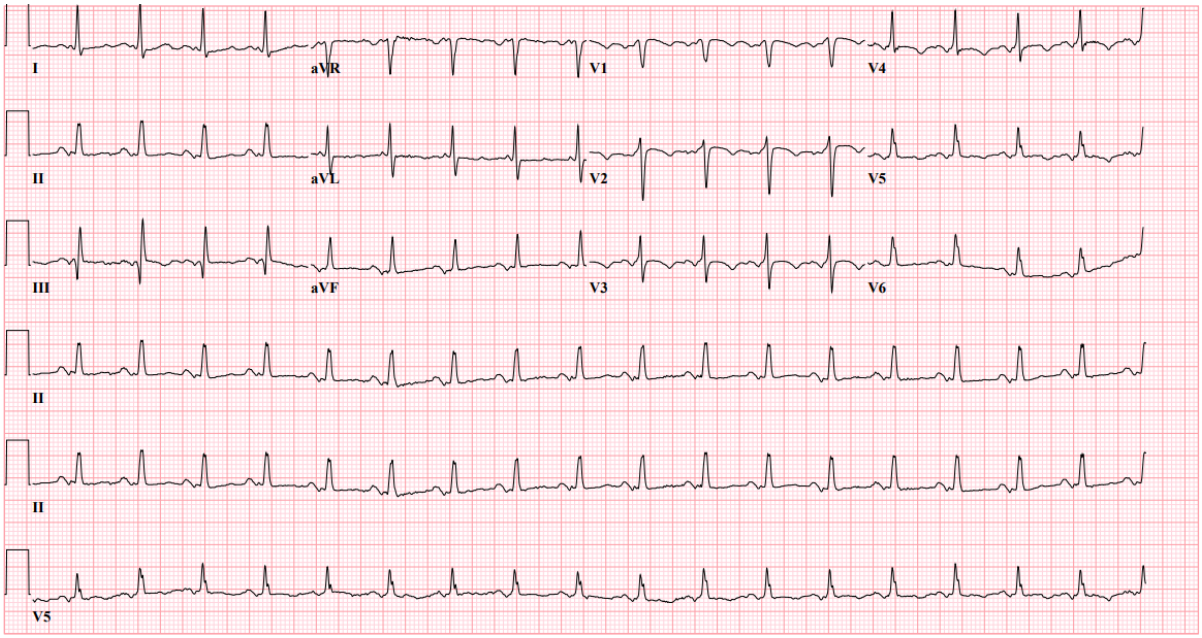

Figure 1 EKG depicts sinus tachycardia with S1Q3T3 pattern

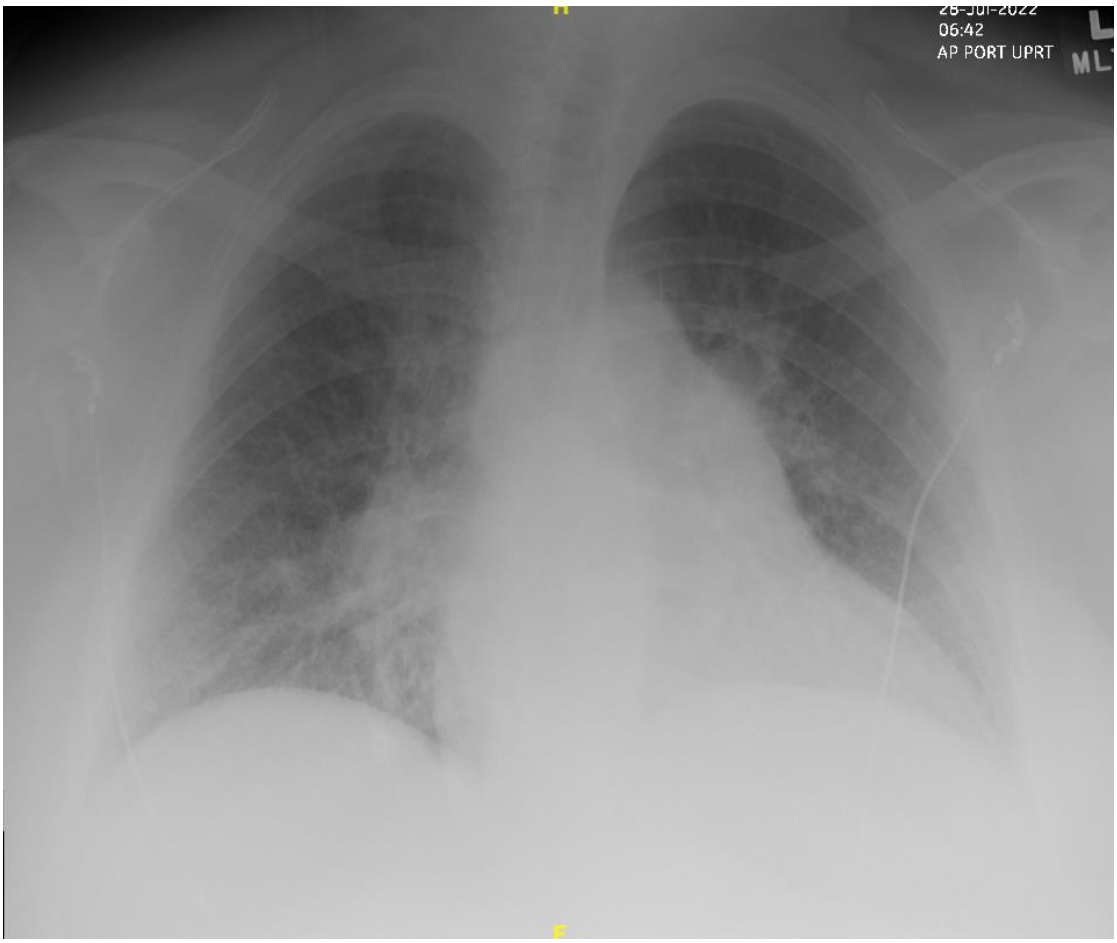

Figure 2 Chest x-ray demonstrates cardiomegaly with mild interstitial edema and prominent superior left heart border suspected to represent pulmonary arterial enlargement

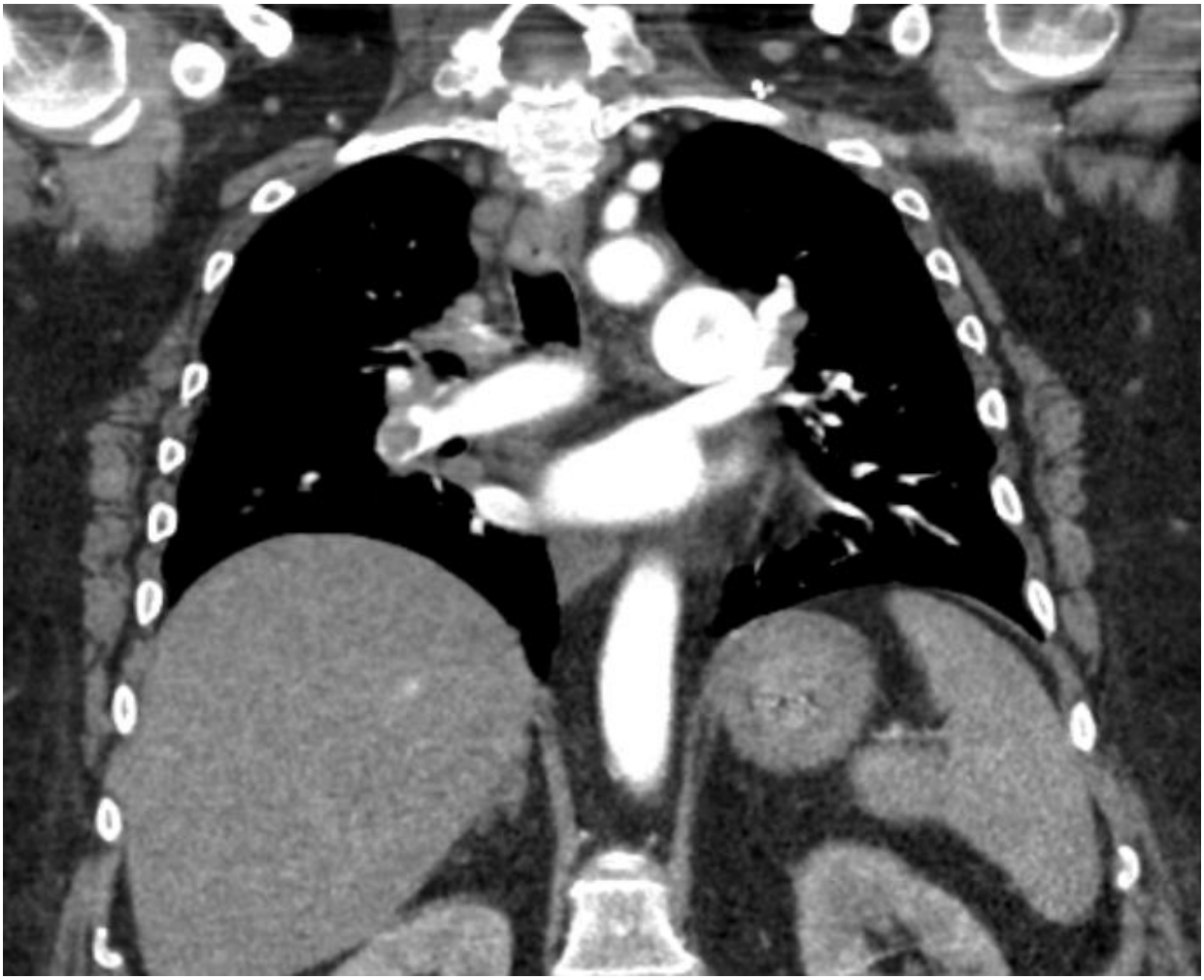

Figure 3 Coronal view of chest computed tomography angiogram shows bilateral pulmonary embolism

Venous Doppler of the lower extremities showed an occlusive thrombus of the left peroneal vein as well as a nonocclusive deep venous thrombosis (DVT) of the upper segment of the left femoral and popliteal veins. Cardiology was consulted, and TTE revealed an ejection fraction of 65-70% with the ventricular septum showing systolic and diastolic flattening suggestive of right ventricular volume and pressure overload; the RVSP was 99.12 mmHg, and McConnell's sign was apparent in this study. High-sensitivity troponin on admission was elevated at 113.

The patient was diagnosed with intermediate high-risk pulmonary emboli due to labile blood pressures, echocardiographic evidence of RV strain, and elevated N-terminal prohormone of brain natriuretic peptide (NT-proBNP) level of 2327 and high-sensitivity troponin level of 113.

She exhibited numerous vacillations between times of hypotension to periods of hypertension, with systolic pressures exceeding 200 on occasion. After heparin infusion was initiated, interventional radiology performed a successful aspiration thrombectomy. Follow-up TTE demonstrate a normal-sized right ventricle with normal systolic function and an RVSP of 63.48 mmHg. Pulmonology was consulted for continued post-thrombectomy respiratory support. The patient remained stable after the procedure and was eventually downgraded from the intensive care unit (ICU); her blood pressure soon stabilized thereafter. On hospital day 5, she was transitioned from the heparin drip to DOAC before being discharged the following day with instructions to continue oral anticoagulation and for close follow-up with cardiology and pulmonology.

3. Discussion

A pulmonary embolism is defined as an obstruction of the pulmonary artery or one of its branches by material (eg, thrombus, tumor, air, or fat) that originated elsewhere in the body [2]. It can be classified by the temporal pattern of presentation (acute, subacute, or chronic), the presence or absence of hemodynamic stability, elevated troponins or

BNP, the anatomic location (saddle, lobar, segmental, subsegmental), or the presence or absence of symptoms (symptomatic or asymptomatic). Our patient presented with sudden onset shortness of breath while at rest, hypoxia with initial oxygen level of 88%, chronic history of smoking, and no history of prior DVTs, PEs, recent travel, malignancy, recent surgeries, or hormone therapy. Patients with signs and symptoms immediately after the obstruction of the pulmonary vessels, such as our patient, are likely to have an acute PE.

According to the American Heart Association, high-risk PEs are characterized by the presence of sustained hypotension (systolic blood pressure <90 mm Hg) not due to arrhythmia, hypovolemia, sepsis, or left ventricular dysfunction, and lasting for at least 15 minutes [2]. Intermediate high-risk PEs are characterized by right ventricular dysfunction or myocardial necrosis and the absence of systemic hypotension (systolic blood pressure >90 mm Hg) [2,3]. In general, intermediate high-risk PEs occur in hemodynamically stable patients and are less likely to cause death within two hours from obstructive shock than high-risk PEs. Therefore, prompt diagnosis on the basis of the slightest clinical suspicion for a PE, followed by immediate and adequate anticoagulant therapy is key in preventing the detrimental effects of PEs [5,6].

Patients typically present with dyspnea followed by chest pain that is usually, but not always, pleuritic in nature similar to our patient's initial presentation. Hemoptysis is an unusual presenting symptom, and with intermediate high-risk PEs, patients can develop shock, arrhythmia, or syncope [2].

Epidemiologic investigations of venous thromboembolisms (VTEs) have demonstrated encouraging patterns in the rates of recurrent VTEs, but they have also shown increased rates of first-time diagnoses of VTEs over recent decades [4]. While this is almost certainly in part due to improved detection from advancements in clinical knowledge and medical technologies, an increased propensity for coagulopathies in today's patients by virtue of sedentary lifestyles, greater incidences of certain malignancies, and longer lifespans cannot be overlooked. In order to capture the full breadth and scope of PEs as well as strategies for management, reports detailing the clinical intricacies and how they vary in presentations in different patients and situations are warranted. An obvious inquiry for further investigation posed by this specific clinical vignette concerns the potential beneficial role of hypertension in preventing decompensation into more dangerous (i.e., high-risk) forms of PE, much in the same manner of permissive hypertension and its utility in cerebrovascular accidents.

4. Conclusion

Owing to their varying and non-specific presentations that can often include clinical silence, along with their potential for fatal outcomes, pulmonary emboli are highly feared events. Recent decades have seen considerable improvements in identifying these lesions, but much remains to be accomplished in the realms of timely diagnosis and survival rates. In this clinical vignette, we presented a highly unusual case in which a patient with an intermediate high-risk pulmonary embolism presented with asymptomatic bradycardia. Vigilance on the part of physicians in confronting this potentially silent culprit is paramount to ensuring accurate and punctual diagnosis, which is necessarily requisite for survival and complete recovery.

Compliance with ethical standards

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Disclosure of conflict of interest

All listed authors have no conflicts of interest to declare.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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