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Acute Pulmonary Embolism Presenting with Symptomatic Bradycardia: A Case Report and Review of the Literature

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCEFG 1 Rabah Alreshq
EFG 1 Gregory Hsu
ACDEF 2 Mikhail Torosoff

1 Department of Medicine, Albany Medical College, Albany, NY, U.S.A.
2 Department of Cardiology, Albany Medical College, Albany, NY, U.S.A.

Corresponding Author: Rabah Alreshq, e-mail: alreshr@amc.edu
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Patient: Male, 92
Final Diagnosis: Pulmonary embolism
Symptoms: Dizziness
Medication: —
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Challenging differential diagnosis

Background: Acute pulmonary embolism (PE) is a common life-threatening cardiovascular emergency. The diagnosis of PE may be challenging, as there can be a wide range of atypical presentations.

Case Report: A 92-year-old man with asymptomatic first-degree atrioventricular (AV) block, hypertension that was controlled on medication, and a past medical history of deep venous thrombosis (DVT), presented with dizziness, weakness, and collapse while getting dressed. On examination by the attending paramedics, he was noted to have sinus bradycardia at a rate of 18 bpm, which improved to 80 bpm after intravenous injection of atropine. An echocardiogram obtained in the emergency room (ER) showed a markedly dilated right ventricle (RV) with a hypokinetic RV free wall, preserved RV apical contractility, and septal wall motion abnormalities consistent with RV pressure overload. A ventilation/perfusion (V/Q) scan showed a massive PE involving more than 50% of the pulmonary vasculature. Urgent catheter-directed thrombolysis was performed, but the patient's condition deteriorated, and he died shortly afterward.

Conclusions: Sinus bradycardia is an unusual initial presentation of PE, but the diagnosis should be considered in patients with multiple risk factors for thromboembolism.

MeSH Keywords: Bradycardia • Pulmonary Embolism • Thromboembolism

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Background

Acute pulmonary embolism (PE) is a relatively common medical emergency and is a life-threatening condition [1], with a three-month mortality rate of up to 10% [2]. While the symptoms of PE are often non-specific, the most common presentation includes symptoms such as acute dyspnea, tachypnea, and hypoxemia, as at least one of these symptoms is present in 92% of patients with PE [3,4].

Acute dyspnea is the most common presenting sign of PE, noted in approximately 50% of these patients. Tachycardia is another common presenting symptom of PE, noted in at least 25% of these patients. Furthermore, tachycardia is one of diagnostic criteria included in the Well's score, a validated tool for stratification of PE risk [5,6]. However, PE may also present with atypical, non-specific symptoms including syncope, abdominal pain, cardiac arrhythmia, or seizure [1,7]. The wide variation in presenting symptoms complicate ante-mortem PE diagnosis, with up to 84% of PE cases discovered only on autopsy [8].

This report is of an atypical presentation of PE in a 92-year-old man who presented with symptomatic bradycardia.

Case Report

A 92-year-old man presented with first-degree atrioventricular (AV) block. He had a history of hypertension that was controlled on medication, stroke, an asbestos-related lung disease with pleural calcification, rheumatoid arthritis, and deep venous thrombosis (DVT), three years before this presentation. His current medications included diltiazem 180 mg, and combined hydrochlorothiazide (25 mg) and triamterene (37.5 mg) for hypertension, and clopidogrel (75 mg) for the prevention of secondary stroke.

Before his recent presenting event, the patient was in his usual state of health and was ambulatory, but with significantly reduced mobility due to arthritis. On the morning of his presentation to the hospital, the patient experienced dizziness and weakness while getting dressed, fell back on his bed, and slid down onto the floor. There was no loss of consciousness, no seizure activity, and no urinary or fecal incontinence. When the emergency services arrived, the patient was lethargic with a normal sinus rhythm of 18 beats/min and first-degree atrioventricular (AV) block. After receiving two doses of intravenous atropine (0.5 mg), his sinus rate increased to 80 beats/min.

During examination in the emergency room, the patient denied chest pain, shortness of breath, or cough. His blood pressure was 136/90 mmHg, his pulse rate was 73 beats per minute (bpm), his respiratory rate was 16 breaths per minute, and his oxygen saturation was 95% on breathing room air. His cardiovascular, pulmonary, and neurological examinations were unremarkable. There was no significant edema. Laboratory investigations were notable for a leukocytosis of 11.6×10^9/L, serum creatinine of 1.5 mg/dl, troponin of 1.75 ng/mL, and pro-brain natriuretic peptide (pro-BNP) of 1829 pg/ml. An electrocardiogram (ECG) showed sinus bradycardia of 48 bpm with first-degree AV block (Figure 1).

The patient was admitted to the hospital under the care of the internal medicine service and was placed under close observation. A transthoracic echocardiogram (TTE) showed a dilated right ventricle (RV) with a hypokinetic free wall and preserved right RV apical function (McConnell's sign). The interventricular septum was displaced towards the left ventricle, consistent with RV pressure overload. Given the patient's previous history of DVT and findings consistent with acute RV strain, the patient was further evaluated for PE. Computed tomography angiography (CTA) was not undertaken due to renal impairment. However, a ventilation/perfusion (V/Q) scan was performed and showed a high probability of PE with the involvement of more than 50% of the pulmonary vasculature (Figure 2).

Duplex ultrasound examination of the lower extremities was performed to evaluate the source of the PE and showed acute

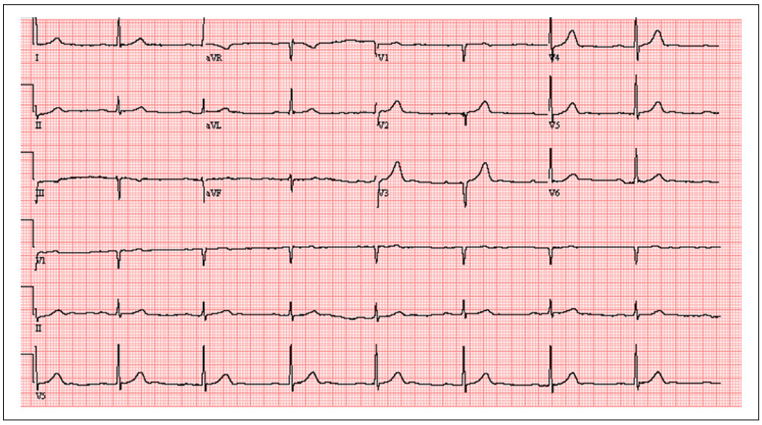

Figure 1. The electrocardiogram (ECG) demonstrates sinus bradycardia of 48 beats per minute (bpm) with first-degree atrioventricular (AV) block.

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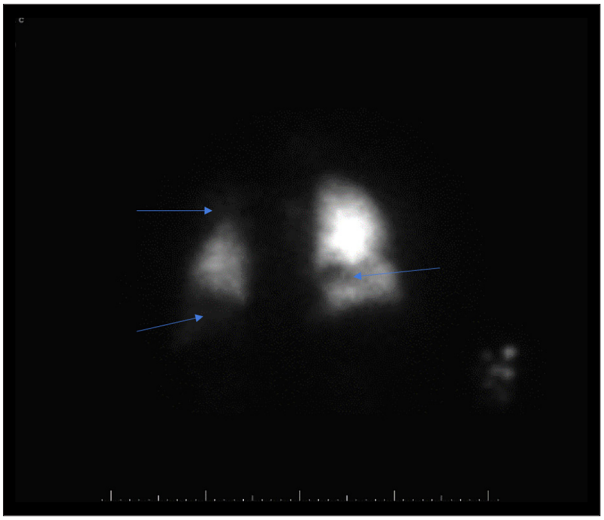

Figure 2. The ventilation/perfusion (V/Q) scan shows a high probability of pulmonary embolism (PE) in both lungs. More than 50% of the pulmonary vasculature contains pulmonary thromboembolus. Perfusion defects are identified by arrows.

occlusion of the left common femoral vein. As systemic administration of thrombolytic agents was contraindicated given the patient's age, history of multiple comorbidities and high risk of bleeding, the patient was started on a high dose infusion of unfractionated heparin and underwent catheter-directed thrombolysis. However, the patient's condition deteriorated due to progressive hypoxemia, and he died shortly afterward. Endotracheal intubation was not performed, as the patient had an advanced directive not to be resuscitated.

Discussion

This report is of an unusual case of acute submassive pulmonary embolism (PE) presenting as symptomatic bradycardia. This patient lacked the typical signs and symptoms of PE. The definitive diagnostic evaluation for PE was undertaken after the discovery of a dilated right ventricle (RV) with evidence of RV strain and positive McConnell's sign. Despite not having pleuritic chest pain and shortness of breath, the patient suffered from reduced mobility and also had a history of treated deep venous thrombosis, both of which increased his risk of PE [9]. The clinical risk of PE in this patient was intermediate, based on his Well's score, but the findings from his ventilation/perfusion (V/Q) scan indicated that he had an 88% chance of having a PE [10]. Computed tomography angiography (CTA) was not performed in his case, as the patient had renal impairment.

This case report highlights the importance of recognizing that PE may present with a wide spectrum of manifestations, which range from lack of symptoms to sudden death [11]. The diagnosis of acute PE continues to be difficult due to variability in the clinical picture at presentation. The atypical presentations of PE, as reported in the literature, are presented in Table 1.

Large cohort clinical studies have shown that sinus bradycardia may be present in more than 2% of patients with PE, and 3.5% may have first-degree AV block at presentation [34]. PE complicated by cardiogenic shock may present as syncope in 26% of patients [35]. Patients with large, hemodynamically significant PE that results in syncope are expected to present with compensatory tachycardia [36]. However, sinus tachycardia has been documented in only 30% of patients with an acute embolism of the pulmonary trunk and the main pulmonary arteries [37]. It has been proposed that bradycardia in patients with PE may be related to vagal stimulation which lowers heart rate and causes concurrent atrioventricular (AV) block. It is presumed that RV dilation and pressure overload from PE lead to excess vagal stimulation [4]. In the presented case report, bradycardia could also be a side effect of prolonged treatment with diltiazem. However, this patient had never experienced symptomatic bradycardia prior to the development of PE, and his baseline asymptomatic first-degree AV block was unchanged. The management of patients with PE presenting with bradycardia is not well described. In one case report, symptomatic bradycardia resolved following theophylline treatment [12]. In this patient, symptomatic bradycardia resolved following intravenous administration of atropine.

Increased serum levels of troponin and pro-brain natriuretic peptide (pro-BNP) in patients with acute PE indicate RV strain and dysfunction and are associated with an increased risk of poor clinical outcome and increased mortality [13]. The report of this case supports this association, as this patient had markedly raised serum levels of pro-BNP and troponin on hospital admission, and his echocardiogram showed severe right heart strain with impaired RV systolic function, which indicated a poor prognosis. These findings also altered the clinical management, as increased serum levels of cardiac biomarkers and the presence of RV strain on transthoracic echocardiography (TTE) in patients with submassive PE are indications for fibrinolysis, which can be administered systematically, or using catheter-directed thrombolysis, as in this case [38,39].

Conclusions

Pulmonary embolism (PE) presenting with sinus bradycardia is rare. However, PE should be considered in patients who have multiple risk factors for thromboembolism. Missing the diagnosis of PE can increase patient morbidity and mortality. Early diagnosis and appropriate treatment interventions are required to improve clinical outcome in patients with acute PE.

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Table 1. Case reports of atypical presentation of pulmonary embolism (PE), including the current case.

First Author	Atypical presenting symptom/s	Age (years)	Gender	Diagnosis	Treatment	Outcome	No. of cases
Alreshq et al.	Bradycardia	92	M	V/Q scan	Catheter-directed thrombolysis	Death	1
Catella et al. [12]	Bradycardia + dyspnea	32	F	CTA	Anticoagulation + theophylline	Survival	1
Majidi et al. [4]	Epigastric pain	42	M	CTA	Heparin	Death	1
Amesquita et al. [13]	Flank pain	63	F	CTA	Heparin	Unknown	1
Viswanath et al. [14]	New atrial fibrillation	82	M	CTA	Heparin + warfarin	Survival	1
Amesquita et al. [13]	Flank pain	64	F	CTA	Unknown	Unknown	1
Rehman et al. [15]	RUQ + back pain	53	M	CTA	Enoxparin + rivaroxaban	Survival	1
Migneault et al. [16]	RUQ + back pain	63	F	CTA	Tenecteplase + heparin	Unknown	1
Amesquita et al. [13]	RUQ + flank pain	52	M	CTA	Heparin	Unknown	1
Gantner et al. [17]	RUQ pain	48	M	CTA	Heparin	Survival	1
Fosmire et al. [18]	RUQ pain	34	F	CTA	Self-resolved	Survival	1
Allou et al. [19]	Seizure	40	M	CTA	Thrombolysis	Death	1
Volz et al. [20]	Seizure	76	M	CTA	Enoxparin + warfarin	Survival	1
Shah et al. [21]	Seizure	20	F	Autopsy	Thrombolysis + heparin	Death	1
Marine et al. [22]	Seizure	33	M	CTA	Thrombolysis + heparin + warfarin	Survival	1
Marine et al. [22]	Seizure	34	F	CTA	Mechanical thrombolysis + urokinase + heparin	Survival	1
Hashmani et al. [23]	Seizure	38	M	CTA	Alteplase + heparin	Survival	1
Lam et al. [24]	Seizure	46	M/F	Varied	Varying	70% (7/10) Mortality	10
Kimura et al. [25]	Seizure	–	M/F	Varied	Varying	N/A	(2/285)
Baloch et al. [26]	Syncope	74	F	CTA	Catheter-directed thrombolysis + heparin	Survival	1
Altinsoy et al. [7]	Syncope	60	N/A	Varied	Varying	N/A	(23/179)
Koutkia et al. [27]	Syncope	54	M/F	Varied	Varying	38% (8/21) Mortality	21
Keller et al. [28]	Syncope	76	M/F	Varied	Varying	N/A	(6792/ 293,640)
Keller et al. [29]	Syncope	78.5	M/F	Varied	Varying	20% (2/10) mortality	(20/182)
Misra et al. [30]	Syncope + fever	43	M	Autopsy	Unknown	Death	1
Meyer et al. [31]	Syncope + seizure	50	M	V/Q scan	Heparin	Death	1
Fred et al. [32]	Syncope + seizure	37	M	Autopsy	Thrombolysis	Death	1
Ronco et al. [33]	Syncope + seizure	7	F	CTA	Thrombolysis + heparin	Survival	1

RUQ – right upper quadrant; M – Male; F – Female; V/Q – ventilation/perfusion; CTA – computed tomography angiography.

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Conflict of interest

None.

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