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

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

Corresponding Author: Rabah Alreshq, e-mail: alreshr@amc.edu
Conflict of interest: None declared

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

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/915609
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
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 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].

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.
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]      | RUQ + 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            |
| Santner 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            |
| Altimsoy 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    | 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.
Conflict of interest

None.

References:

1. Torbicki A, Perrier A, Konstantinides S et al: Guidelines on the diagnosis and management of acute pulmonary embolism: The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC). Eur Heart J, 2008; 29(18): 2276–315

2. Meyer G, Vicaud E, Danays T et al: Fibrinolysis for patients with intermediate-risk pulmonary embolism. N Engl J Med, 2014; 370(15): 1402–11

3. Esponda O, Tafur A: Management of pulmonary embolism: State of the art treatment and emerging research. Curr Treat Options Cardiovasc Med, 2013; 15(2): 137–52

4. Majidi A, Mahmodi S, Baratloo A, Mirhabsa S: Atypical presentation of massive pulmonary embolism, a case report. Emerg (Tehran), 2014; 2(1): 46–47

5. Stein PD, Beemath A, Matta S et al: Clinical characteristics of patients with acute pulmonary embolism: Data from PiOPED II. Am J Med, 2007; 120: 871–79

6. Wells PS, Anderson DR, Rodger M et al: Excluding pulmonary embolus at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and D-dimer. Ann Intern Med, 2001; 135: 98–107

7. Altinsoy B, Erboy F, Tannriverdii H et al: Syncope as a presentation of acute pulmonary embolism. Ther Clin Risk Manag, 2016; 12: 1023–28

8. Stein PD, Stein PD: Pulmonary embolism, 2nd ed. Prevalence, risks, and prognosis of pulmonary embolism and deep vein thrombosis. Oxford: Blackwell Publishing, 2007; 3–15

9. Goldhaber SZ, Grosdstein F, Stampfer MJ et al: A prospective study of risk factors for pulmonary embolism in women. JAMA, 1997; 277(8): 642–45

10. Value of the ventilation/perfusion scan in acute pulmonary embolism. Results of the prospective investigation of pulmonary embolism diagnosis (PiOPED). JAMA, 1990; 263(20): 2753–59

11. Lega JC, Lacasse Y, Lakhal L, Provencer S: Natriuretic peptides and troponins in pulmonary embolism: A meta-analysis. Thorax, 2009; 64(10): 869–75

12. Catella P, Wiecel S, Siddiqui AH, Chalhoub M: A rare case of pulmonary embolism induced symptomatic bradycardia. Am J Respir Crit Care Med, 2017; 195: A5492

13. Amesquita M, Cocchi MN, Donnino MW: Pulmonary embolism presenting as flank pain: A case series. J Emerg Med, 2012; 42(5): 97–100

14. Viswanath O, Simpao AF, Santhosh S: Atypical presentation of a pulmonary embolism in the perioperative setting. AA Case Rep, 2015; 5(4): 54–56

15. Rehman H, John E, Parikh P: Pulmonary embolism presenting as abdominal pain: An atypical presentation of a common diagnosis. Case Rep Emerg Med, 2016; 2016: 7832895

16. Migneault D, Levine Z, de Champlian F: An unusual presentation of a massive pulmonary embolism with misleading investigation results treated with tenecteplase. Case Rep Emerg Med, 2015; 2015: 868519

17. Gantner J, Keffeler JE, Derr C: Pulmonary embolism: An abdominal pain masquerader. J Emerg Trauma Shock, 2013; 6(4): 280–82

18. Fosmire ST, Gibson GN, Copeland JCI et al: Pulmonary infarction: Right upper quadrant pain as a presenting symptom with review of typical computed tomography imaging features. Mil Med, 2018; 183(11–12): 779–82

19. Allou N, Coolen-Allou N, Delmas B et al: Severe pulmonary embolism revealed by status epilepticus. Rev Pneumol Clin, 2016; 72(6): 377–79

20. Volz EE, Jasani N: Seizure as a presentation of pulmonary embolism. J Emerg Med, 2014; 46(1): 1–4

21. Shah AK, Darwent M: Acute pulmonary embolism presenting as seizures. Emerg Med J, 2009; 26(4): 299–300

22. Marine JE, Goldhaber SZ: Pulmonary embolism presenting as seizures. Chest, 1997; 112(3): 840–42

23. Hashmani S, Tippoo Sultan RA et al: Massive pulmonary embolism presenting as seizures. J Pak Med Assoc, 2016; 66(12): 1656–58

24. Lam M, Jammal M, Tiev K et al: Pulmonary embolism revealed by a seizure: A case report and literature review. Rev Med Interne, 2012; 33(8): 457–60

25. Kimura K, Mori H, Kitaguchi H et al: Pulmonary embolism as a cause of seizure. Am J Emerg Med, 2013; 31(10): 1525–27

26. Baloch ZQ, Ayyaz M, Hussain M et al: Syncope: An atypical presentation of pulmonary embolism secondary to occult uterine malignancy. Case Rep Med, 2018; 2018: 9141529

27. Koutkia P, Wachtel TJ: Pulmonary embolism presenting as syncope: A case report and review of the literature. Heart Lung, 1999; 28(5): 342–47

28. Keller K, Hohbom L, Munzel T et al: Syncope in haemodynamically stable and unstable patients with acute pulmonary embolism. Results of the German nationwide inpatient sample. Sci Rep, 2018; 8: 15789

29. Keller K, Beule J, Balzer JO, Dippold W: Syncope and collapse in acute pulmonary embolism. Am J Emerg Med, 2016; 34(7): 1251–57

30. Misra P, Ghosh AK, Jassar A: Autopsy findings in an atypical case of occult massive fat pulmonary embolism in a backdrop of hyperhomocysteinemia. Indian J Pathol Microbiol, 2018; 61(1): 116–19

31. Meyer MA: Seizure as the presenting sign for massive pulmonary embolism. Circulation, 2010; 122(11): 1124–29

32. Fred HL, Yang M: Sudden loss of consciousness, dyspnea, and hypoxemia in a previously healthy young man. Circulation, 1995; 91(12): 3017–19

33. Ronco R, Catalan J, Salgado C, Vogel A: Syncope: A rare presentation of massive pulmonary embolism in a previously healthy girl. Pediatr Emerg Care, 2010; 26(4): 287–89

34. Stein PD, Matta F, Sabra MI et al: Relation of electrocardiographic changes in pulmonary embolism to right ventricular enlargement. Am J Cardiol, 2013; 112(2): 1958–61

35. Kukla P, McIntyre WF, Fijorek K et al: Use of ischemic ECG patterns for risk stratification in intermediate-risk patients with acute PE. Am J Emerg Med, 2013; 32(10): 1248–52

36. Shopp JD, Stewart LK, Emmett TW, Kline JA: Findings from 12-lead electrocardiography that predict circulatory shock from pulmonary embolism: Systematic review and meta-analysis. Acad Emerg Med, 2015; 22(10): 1127–37

37. Zhang J, Liu G, Wang S et al: The electrocardiographic characteristics of an acute embolism in the pulmonary trunk and the main pulmonary arteries. Am J Emerg Med, 2016; 34(2): 212–17

38. Piazza G, Goldhaber SZ: Management of submassive pulmonary embolism. Circulation, 2010; 122(11): 1124–29

39. Agnelli G, Becattini C: Acute pulmonary embolism. N Engl J Med, 2010; 363(3): 266–74