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Bradyarrhythmia in acute massive pulmonary embolism

Jia Wei Tan, Jay Parekh, Anant Shukla

ABSTRACT

Pulmonary embolism (PE) can present with a variety of electrocardiographic findings. Bradycardia is a rare finding in acute PE, which typically manifests with sinus tachycardia. Bradycardia in acute PE might arise from the physiologic Bezold–Jarisch reflex, which describes the constellation of bradycardia, peripheral vasodilation, and hypotension. We report the case of a woman in her 60s who was admitted initially for submassive PE and found to have sinus bradycardia. She had progressed to massive PE with acute worsening of atrioventricular conduction block in the setting of atrial flutter. Her bradyarrhythmia resolved and hemodynamics improved after catheter directed thrombolysis. This case revisits the pathophysiology of Bezold–Jarisch reflex, and the importance of recognizing that it signifies an underlying pathologic insult, especially a life-threatening one like PE.

Keywords: Bradycardia, Bezold–Jarisch reflex, Pulmonary embolism

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INTRODUCTION

Pulmonary embolism (PE) is a life-threatening condition that can cause rapid decompensation in the absence of prompt anticoagulation, thrombolytics, or mechanical thrombectomy. In a patient presenting with dyspnea, hypoxia, and tachypnea, electrographic clues to PE include sinus tachycardia, non-specific ST segment changes and the "S1Q3T3" pattern, which has high specificity and low sensitivity [1]. Bradyarrhythmia is not a common finding in PE, with only handful of cases reported in the literature [2–6].

CASE REPORT

A 65-year-old woman with the past medical history of atrial flutter, heart failure with preserved ejection fraction (left ventricular ejection fraction [LVEF] 50–60%), obesity (class I), and recent left malleolar ankle fracture presented to the emergency department with gradually worsening dyspnea on exertion for several weeks and dizziness. On admission, her vitals were heart rate (HR) of 102 beats per minute (bpm), blood pressure (BP) 130/60 mmHg, respiratory rate (RR) of 18, with an oxygen saturation of 92% on room air. Her presenting electrocardiogram demonstrated atrial flutter at a rate of 150 bpm with 2:1 conduction with a new right axis deviation. Her home medications were resumed, including home dose metoprolol 25 mg twice a day for atrial flutter. She attained rate control with HR of 60s bpm the next day and had subjective improvement in her symptoms. She further revealed being non-adherent with anticoagulation despite having a CHA2DS2-VASc score of 5. A transthoracic echocardiogram (TTE) found a severely elevated estimated right ventricular systolic pressure (RVSP) of 90 mmHg. Because of high clinical suspicion for pulmonary embolism (PE), she was treated with intravenous unfractionated heparin while awaiting inpatient chest tomography pulmonary arteriogram.

On the third day of hospitalization, her routine morning vitals were: HR 58, BP 102/70 mmHg. Hours

later, telemetry revealed that her HR had declined to 35 bpm, and her BP had declined to 52/39 mmHg. Her oxygen saturation on room air was 98%. She had her scheduled dose of 25 mg metoprolol 1.5 hours prior to the bradycardic and hypotensive episode. She complained of new pleuritic chest pain and orthopnea. Physical examination was notable for diaphoresis, elevated jugular venous pressure at 4 cm above the right atrium at 30° of head of bed elevation, and 1+ lower extremity edema. Lungs were clear to auscultation. Within half an hour from the onset of bradycardia, she received 0.5 mg of atropine and 1 liter of normal saline. Her hemodynamic parameters partially improved with HR in the 40–50 bpm and BP 84/59 mmHg.

Her complete blood count and electrolytes included a potassium of 3.7 mg/dL, magnesium of 1.8 mg/dL and creatinine of 1.82 mg/dL (baseline 0.8 mg/dL 1 month ago). D-dimer was significantly elevated at 2.24 mg/L FEU (reference range (Ref): >0.65 mg/L FEU). Troponin T was modestly elevated at 0.10 ng/mL (Ref: <0.01 ng/mL). Lactate was elevated at 4.8 mmol/L (Ref: 0.5–2.2 mmol/L). Arterial blood gas revealed pH of 7.31, PCO_2 54 mmHg, PO_2 88 mmHg, bicarbonate 26.7 mmol/L, and base excess of 0 mmol/L. SARS-CoV-2 polymerase chain reaction test was negative. Electrocardiogram revealed atrial flutter with variable atrioventricular conduction block which ranged from 3:1 to 5:1. Ventricular rate was consistently bradycardic (Figure 1). The narrow width of the QRS complexes suggests that these are impulses being conducted from a supraventricular origin rather than a ventricular or junctional origin. The morphology of the F waves in II, III, and aVF suggests a clockwise typical flutter. There were no ischemic ST-T segment changes. Her electrocardiogram on admission revealed atrial flutter with HR of 100 bpm (Figure 2). Chest X-ray showed no acute changes. Bedside transthoracic echocardiography showed an ejection fraction of 60% with flattened septum and new signs of right ventricular strain (Figure 3).

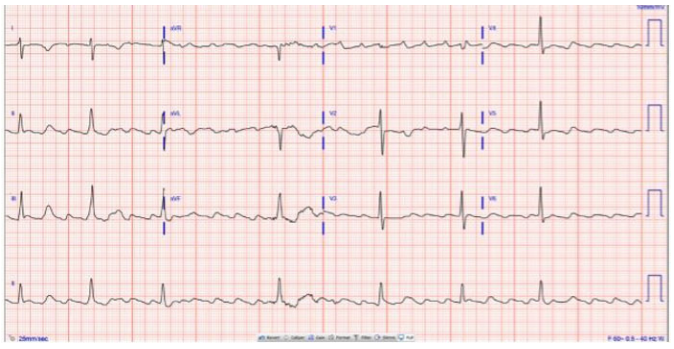

Figure 1: 12-Lead electrocardiogram showed atrial flutter at 150 bpm with variable AV conduction. The morphology of the F waves in II, III, and aVF suggests a typical flutter in clockwise direction. The narrow width of the QRS complexes suggests that these are impulses being conducted from a supraventricular origin rather than a ventricular or junctional origin. There were no ischemic ST-T segment changes.

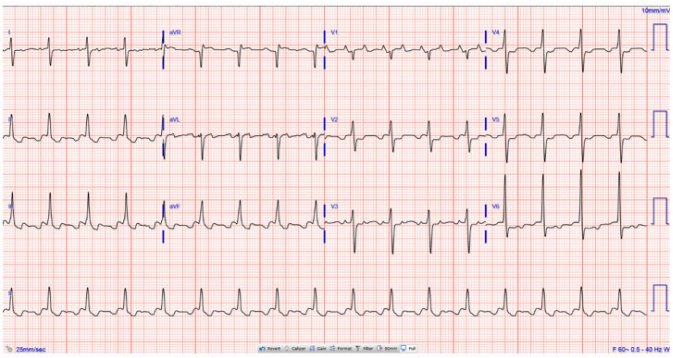

Figure 2: 12 Lead electrocardiogram on admission showed atrial flutter at 100 bpm.

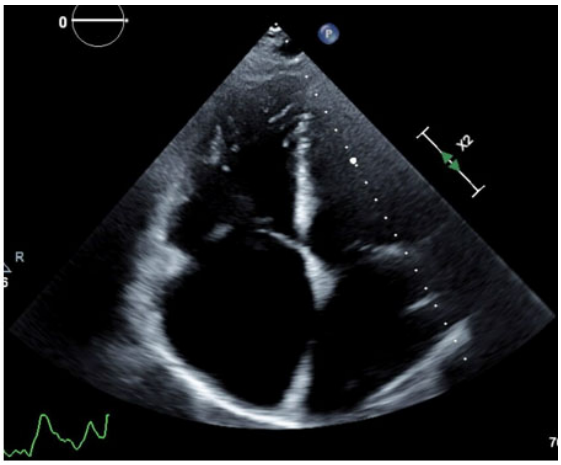

Figure 3: Bedside transthoracic echocardiography showed an ejection fraction of 60% with flattened septum and signs of right ventricular strain. This is an apical 4 chamber view of the heart showing a dilated right ventricle with flattened septum (right side is labeled on the figure).

Differential diagnosis

Inferior myocardial ischemia was considered at first because of the constellation of dyspnea, hypotension, and atrioventricular conduction block. However, the ECG showed no ischemic changes and the echo showed no regional wall abnormalities. We considered beta-blocker toxicity. However, she had been on metoprolol for years without such incident and she received the same dosage she takes at home in the hospital. Besides, beta-blocker toxicity will not explain her symptoms of pleuritic chest pain and orthopnea. She did not receive other medications known to cause negative chronotropy other than metoprolol which she has been taking for years. Pericarditis was considered, but electrocardiogram did not reveal ST segment elevation, PR depression, or T wave inversion. Myocarditis due to a variety of causes including rheumatic fever, Lyme disease, diphtheria, Coronavirus disease 2019 pneumonia, and systemic lupus erythematosus were unlikely given her minimally elevated troponin. Hypoxia was considered as a cause for her worsening bradyarrhythmia. However, the ABG revealed normal PaO_2. Electrolyte derangements were

considered as another possibility for bradyarrhythmia but her serum potassium and magnesium were in a relatively normal range.

We considered septic shock but she had no fever or leukocytosis. Although patient had bradycardia instead of tachycardia, obstructive shock secondary to pulmonary embolism is considered given her risk factors (obesity and recent immobilization), new right axis deviation and elevated RVSP. We considered fat embolism syndrome; however, she did not have the classic triad of hypoxemia, petechia, and altered mental status. Her computed tomography of the chest did not reveal findings suggestive of acute respiratory disease syndrome. Further, the incidence of fat embolism is low at 0.17%, and typically due to femur fracture [7]. Bedside TTE showed normal LVEF but had signs of right ventricular strain, further underscoring this suspicion. She underwent computed tomography pulmonary angiogram pulmonary embolism and enlarged right heart chambers with reflux of contrast within the hepatic veins (Figure 4).

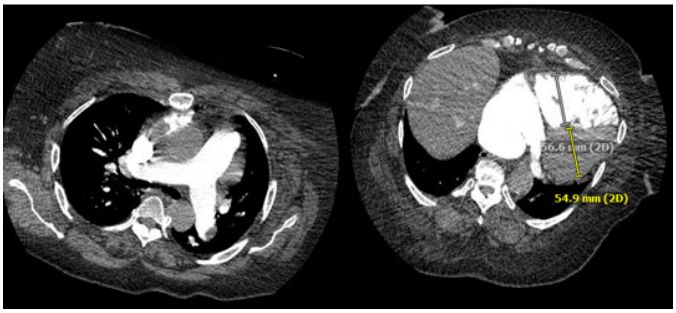

Figure 4: Computed tomography pulmonary angiogram showed bilateral pulmonary embolism and enlarged right heart chambers with reflux of contrast within the hepatic veins.

Treatment

The patient was diagnosed with submassive PE that had progressed into massive PE. She was transferred to the medical intensive care unit and started on dobutamine and norepinephrine. She underwent ultrasound-assisted catheter-directed thrombolytic therapy. Her right heart catheterization revealed a pulmonary artery systolic pressure of 73 mmHg. The next day, TTE continued to show signs of right ventricular strain, but the estimated RVSP had decreased from 90 mmHg on the pre-thrombolysis TTE to 76 mmHg. Her hemodynamics improved after catheter-directed thrombolytic therapy and she weaned off the vasopressors. She eventually transitioned from intravenous heparin to rivaroxaban and was transferred to the general medical ward.

Outcome and follow-up

She was discharged home with rivaroxaban and re-initiation of metoprolol. In subsequent follow-up appointments, she is back to her baseline functional ability and has had no further thrombotic episodes or bradycardia. Coagulopathy workups were non-revealing.

DISCUSSION

Bradyarrhythmia is an unusual finding in acute pulmonary embolism. The overall incidence of sinus bradycardia is 2% and first degree atrioventricular block is 3.5% [1]. Second- or third-degree atrioventricular block in pulmonary embolism is rarely reported [3, 8, 9]. This case is illustrative of the diverse conduction abnormalities and arrhythmias that are pulmonary embolism-related, ranging from a normal electrocardiogram (9–26% of the patients) to findings suggestive of right ventricular strain, including S1Q3T3 pattern, right axis deviation, right bundle branch block. The incidence of atrial flutter in acute pulmonary embolism ranges from 3% to 35%. Right axis deviation is less predictive of PE than left axis deviation [10].

This phenomenon might be attributed to the Bezold–Jarisch reflex (BJR), which describes the stimulation of cardiac vagal afferent endings that evokes reflex hypotension, peripheral vasodilation and bradycardia (Figure 5). During periods of ischemia and reperfusion, active metabolites (prostaglandin, bradykinin, reactive oxygen species) can activate the chemosensitive receptors with vagal afferent, and evoke this cardio-inhibitory reflex [11]. The BJR could be cardioprotective—by causing bradycardia and afterload reduction via peripheral vasodilation, and thus avoiding cardiac ischemia through the reduction in workload of the heart. The recognition of the triad of bradycardia, peripheral vasodilation, and hypotension should prompt a search for underlying pathologic state that activates the BJR. Pulmonary embolism should be considered in this scenario, especially in patients who have multiple risk factors for thromboembolism.

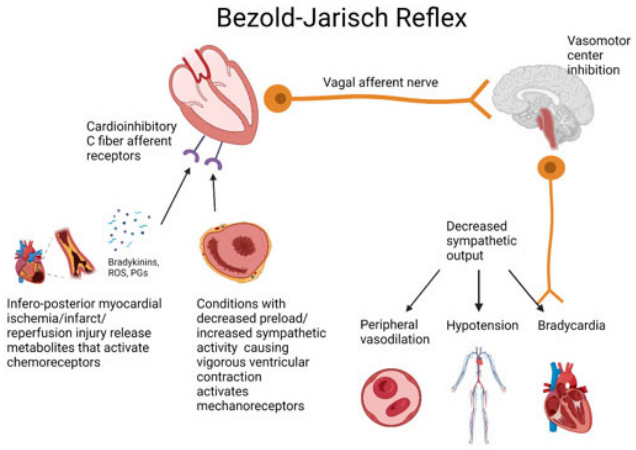

Figure 5: The Bezold–Jarisch reflex—the triad of bradycardia, hypotension, and vasodilation that occurs upon stimulation of cardiac receptors. Information referenced from reference numbers [11] and [12]. Figure created by BioRender.

Our patient was empirically anticoagulated for pulmonary embolism prior to computed tomography pulmonary angiogram due to her high pre-test probability and potentially fatal consequences of untreated submassive pulmonary embolism. She likely has preexisting pulmonary hypertension (elevated RSVP of 90 mmHg on encounter) and hence, a small degree of obstruction can disproportionately increase the pulmonary artery systolic pressure and precipitate right ventricle failure. The combination of impaired coronary perfusion (from reduction in cardiac output and aortic pressure) and elevated right ventricle afterload can cause myocardial ischemia [12].

In the era of intracoronary thrombolysis, it was reported that majority of the patients with right coronary artery reperfusion developed transient bradycardia and hypotension during intracoronary thrombolysis. Most of these patients had spontaneous resolution of the bradycardia and hypotension and the rest recovered with administration of atropine and intravenous fluids. Patients with persistent occlusion did not demonstrate such reflex cardiovascular changes [13]. Hence, BJR may signify reperfusion in ischemic myocardium. The decreased sympathetic tone and increased cholinergic-mediated coronary blood flow might be beneficial to the stressed myocardium.

Efforts should be directed in treating underlying disease and providing supportive care during period of bradycardia secondary to cardioinhibitory reflex. Supportive treatment such as atropine and fluid challenge help in reversing the pathophysiology of BJR. Pacemaker insertion is not recommended in BJR, as the bradyarrhythmia phenomenon is transient and reversible with the treatment of underlying disease state. Temporary pacemaker placement is unlikely to correct the vagally mediated hypotension. Atropine is more effective than pacemaker because it antagonizes the reflex cholinergic vasodilation as well as the bradycardia.

CONCLUSION

In rare cases, pulmonary embolism can present with bradyarrhythmia. The Bezold–Jarisch reflex reflects an underlying pathologic insult that results in decreased venous return and low preload, such as myocardial ischemia, cardiac reperfusion injury, or pulmonary embolism. Recognition of the Bezold–Jarisch reflex is essential for management as the bradyarrhythmia is reversible and transient, and treatment should be directed at the underlying pathologic insult (e.g., catheter-directed thrombolytics for massive pulmonary embolism). Bradyarrhythmias arising from the Bezold–Jarisch reflex mediated are unlikely to be corrected by pacemaker and thus cardiac pacing is not indicated.

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Author Contributions

Jia Wei Tan – Conception of the work, Design of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Jay Parekh – Analysis of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related

to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Anant Shukla – Analysis of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission
The corresponding author is the guarantor of submission.

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Consent Statement
Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest
Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.