

Pulmonary Embolism Presenting as a Complete Heart Block

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Received April 08, 2023; Revised May 13, 2023; Accepted May 24, 2023

Abstract Pulmonary embolism (PE) is also known as the great faker. It is difficult to diagnose and entails a lot of suspicion from a clinician. Our case talks about the appearance of a complete heart block in a patient with PE who had a pre-existing left bundle branch block. We present the case of a 75-year-old lady with a pre-existing left bundle branch block, diabetes and hypertension presented to the emergency department because of shortness of breath which had been ongoing for 4 days. She was tachycardic and hemodynamically stable initially on arrival. Computed tomography pulmonary angiogram done in the emergency department showed an embolus in the distal right main pulmonary artery, which was extending to and involving the proximal right lower lobe of the pulmonary arterial tree. Follow-up cardiac echography showed an enlarged right ventricle with decreased right ventricular systolic function. Initial electrocardiogram (EKG) only showed a left bundle branch block. However later during the day, the patient became hemodynamically unstable before thrombectomy. The thrombectomy was planned for the following day. Repeat electrocardiogram at this point in time showed a third-degree heart block. The patient received atropine and a transvenous pacemaker was placed. She later on underwent thrombectomy. Electrocardiogram post-thrombectomy repeatedly showed complete heart block, hence the decision was made to place a permanent pacemaker. Pulmonary embolism can present in a variety of ways. It could possibly present as a complete heart block. In patients with a preexisting left heart block, a PE could easily cause a complete heart block especially in the scenario where we have a right ventricular strain evident on a transthoracic echocardiogram (TTE). The presence of right ventricular strain on EKG, be it T wave inversion in V1 like in my patient or new onset complete right bundle branch block could be used as an indicator of massive/sub-massive(severe) pulmonary embolism.

Keywords: pulmonary embolism, cardiology, complete heart block

Cite This Article: Antoine Egbe, Agyingi Chris, Hussein Gaith, and Khurram Arshad, "Pulmonary Embolism Presenting as a Complete Heart Block." *American Journal of Medical Case Reports*, vol. 11, no. 5 (2023): 95-97. doi: 10.12691/ajmcr-11-5-3.

1. Introduction

Pulmonary embolism is a well-documented pathology in current medical literature with renowned cardiovascular complications.

There are a multitude of EKG abnormalities which are common in patients afflicted with pulmonary embolism. [1] From review of literature they include; sinus tachycardia, left axis deviation and right axis deviation, a leftward shift of the transition zone from V3-V4 to V5, maximum overall deflection of the QRS complex \leq 5mm in all limb leads, nonspecific ST segment changes which could include ST and T wave changes, the classic S1Q3T3 pattern, p-wave amplitude greater than 2.5mV in lead II, supraventricular tachyarrhythmias such as atrial fibrillation and atrial flutter, and a RBBB pattern. There are rare case reports of transient left bundle branch block induced by PE [2].

Multiple EKG abnormalities have been documented but

rarely is the case of complete heart block in the setting of PE especially in patients with preexisting rhythm abnormalities. In the case of our patient, she had a pre-existing left bundle branch block.

2. Case Presentation

75-year-old woman with past medical history of hypertension, type 2 diabetes mellitus and underlying Left bundle branch block (LBBB), who presented to the emergency department (ED) with shortness of breath over the course of four days, exacerbated by exercise. She did not report chest pain, excessive sweating and she did not report usage of hormone replacement therapy.

On admission, the patient was hemodynamically stable. Vital signs were as follows: Blood pressure was 115/63, heart rate of 115 bpm, temperature of 97.3 °F (36.3 °C), Body mass index of 37.93, and oxygen saturation of 97% on ambient air. Initial EKG was consistent with her baseline EKG of left bundle branch block.

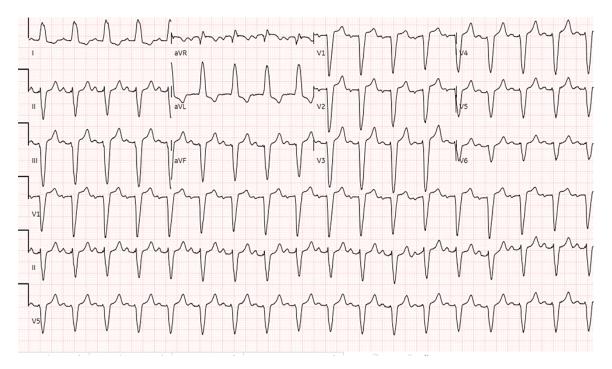
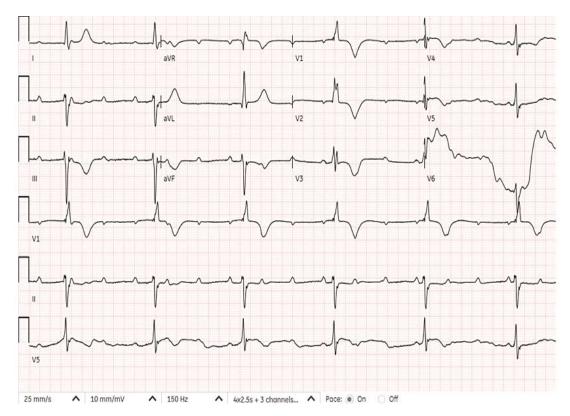


Figure 1.

Pertinent labs include a positive troponin level of 0.15 which trended up to 0.24, brain natriuretic peptide was elevated at 406, elevated D-dimer which stood at 2500. This prompted the request for a computed tomography pulmonary angiogram. It revealed an embolus in the distal right main pulmonary artery, which was extending to and involving the proximal right lower lobe of the pulmonary arterial tree. Follow-up transthoracic echocardiography was consistent with a moderately enlarged ventricle and a mildly enlarged right atrium. The right ventricular systolic function was moderately decreased.

Due the right heart strain and her overall clinical picture, she was scheduled for thrombectomy. Prior to the procedure, she suddenly became bradycardic with HR 20-30s, tachycardic and hypotensive. A rapid response team was activated. An EKG (Figure 2) was ordered, and it revealed complete heart block. During the rapid response, the patient did not respond to atropine.



A transvenous pacemaker was placed and after achieving hemodynamic stabilization, the patient underwent emergent thrombectomy. Post-thrombectomy, Electrophysiology department was consulted due to persistent complete heart block, and they opted for permanent pacemaker implantation. Pacemaker was placed and patient remained stable thereafter. At the end of her hospital stay, the patient was discharged on apixaban 5 mg twice daily since her pulmonary embolism was unprovoked with plans for follow up in the cardiology clinic.

3. Discussion

Pulmonary embolism (PE) is defined as a blood clot found in the pulmonary circulatory system. PE is known as one of the top-rated causes of cardiovascular deaths worldwide [3]. PE usually arises as result of deep venous thrombosis and mainstay stay management for PE is adequate anticoagulation and invasive procedures like thrombectomy in high-risk patients. Some of the EKG changes associated with PE are thought to be as a result of right ventricular dilation. The right ventricular dilation is believed to be as a result of the back-up pressure from the pulmonary vasculature obstruction. Common EKG changes include sinus tachycardia, RBBB, S1Q3T3 pattern. Though most of the EKG changes are generally not concerning, RBBB has been shown to be a predictor for negative outcomes in PE. [4]

Third degree heart block is a rare complication of PE. The pathophysiology is poorly understood, however one of the current hypotheses [5] is that a PE could trigger an adrenergic surge stimulating the Bezold-Jarish reflex, a vagal response and decreased sympathetic tone, hence leading to complete heart block particularly in patients with an already diseased conduction system. In our patient, she had a pre-existing LBBB with possible new RBBB leading to complete heart block with profound bradycardia despite the removal of the inciting event [PE] leading to the placement of a permanent pacemaker. RBBB was likely as a result of right ventricular dilatation and strain since the right bundle branch is relatively superficial in its path along the right ventricular side of the septum, it may be particularly sensitive to acute distension of the right ventricular cavity [4,5].

Complete heart block (CHB) with reversible causes is thought to be reversible after the resolution of the inciting factor. However, this patient had persistent complete heart block and was dependent on the transvenous pacemaker despite having undergone thrombectomy. This is suggestive of a profound lesion to the right bundle branch with no recovery despite thrombectomy.

Jacob D. et al [6] showed that six EKG findings are associated with hemodynamic collapse and death within

30 days of acute PE. The EKG on Figure 2 has 2 of these six EKG findings which is the inversion of T-wave in V1 - V5 and a complete right bundle branch block. This corroborates to the assertion that right ventricular strain on EKG is an important prognostic indicator of pulmonary embolism severity. The recent spike in the usage of bedside ultrasound may provide a better and easy way to determine right ventricular strain with bedside TTE. However, we could still use our EKG together with our TTE to come to a conclusion of whether we have or do not have right ventricular strain in a patient acutely presenting with PE. Most importantly in places where a TTE is not readily available, an EKG might prove to be an awesome tool for prognostication and triage in patients with pulmonary embolism.

This case also highlights the importance of continuous cardiac monitoring in patients with massive PE especially those with pre-existing conduction abnormalities. The mechanism of this complication is yet to be understood and will require further investigation to explain this newly presented relationship.

4. Conclusions

Pulmonary embolism can manifest in different ways on an EKG. One of those ways is by causing a right heart strain which could show up as a right bundle branch block on EKG. Special attention should be paid to patients who have a pre-existing left bundle branch block prior to the PE because they can then develop a complete heart block. Right bundle branch block on EKG is associated with severe PE and carries a poor prognosis if not treated accordingly and promptly.

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