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Case Report

Double Whammy of Pulmonary Embolism Complicated by High-Degree Atrioventricular Block: A Case Report

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ABSTRACT

High-degree Atrioventricular (AV) block is rare in acute Pulmonary Embolism (PE). We present a case of a 62-year-old woman experiencing recurrent PE complicated by Complete Heart Block (CHB). Her PE was classified as intermediate-low, and she was treated with a heparin infusion. Due to persistent CHB, a permanent pacemaker was implanted, and she was discharged with Rivaroxaban. We also conducted a review of 22 case reports involving PE and high-degree AV block. Our study highlights the importance of early identification of high-degree AV block in cases of acute PE. Continuous monitoring and timely intervention are crucial in managing these patients.

Keywords: Pulmonary embolism; High-degree atrioventricular block; Case report

INTRODUCTION

Acute pulmonary embolism (PE) is a life-threatening condition that has emerged as a significant global health concern. The incidence of acute PE in the United States is estimated to be 1.15 cases per 10,000 person-years, accounting for approximately 0.16% of all emergency department visits [1]. Acute PE has a 30-day mortality rate of 30% if left untreated, with 11% of patients succumbing within the first hour of hospital presentation [1].

Acute PE is often referred to as the "great faker" because its symptoms can range from subtle signs, such as pleuritic chest pain and dyspnea, to severe manifestations like syncope or sudden cardiac arrest. Various types of arrhythmias may also be observed in cases of acute PE, including sinus tachycardia, atrial tachycardia, atrial flutter, and atrial fibrillation. Common Electrocardiographic (ECG) findings associated with acute PE include right axis deviation, the S1Q3T3 pattern (McGinn-White sign), Right Bundle Branch Block (RBBB), ST-segment elevation in leads V1 or V3, ST-segment depression in leads V4 to V6, and inverted T waves in leads V2 to V4. These ECG abnormalities are typically indicative of Right Ventricular (RV) compromise.

Transient Left Bundle Branch Block (LBBB) and/or high-degree Atrioventricular (AV) block had been suggested as important atypical clinical manifestations, although being rare. This can complicate the clinical course and lead to adverse outcomes if not promptly recognized. Here, we present a case of submassive PE complicated by severe pulmonary hypertension and persistent Complete Heart Block (CHB) that necessitated permanent pacemaker placement. Additionally, we reviewed the literature on the presentations, ECG findings, clinical course, and treatments of 22 case reports involving PE and high-degree AV block.

CASE PRESENTATION

A 62-year-old female presented to the Emergency Department (ED) with a two-week history of exertional shortness of breath. The patient had a past medical history of unprovoked deep vein thrombosis and PE 20 years ago, for which she was treated with warfarin. She had been compliant with her medication and had not experienced any adverse events. However, two weeks prior, her haematologist discontinued the warfarin. Her other medical conditions included type 2 diabetes, diabetic neuropathy, essential hypertension, hyperlipidemia, and obesity.

Upon arrival, her vital signs blood pressure 149/72 mmHg, heart rate 35 beats per minute, respiratory rate 16 breaths per minute, and oxygen saturation 97% on ambient air. Her laboratory results indicated normal troponin but mildly elevated B-type Natriuretic Peptide (BNP) (340 pg/mL, normal range<100 pg/mL). An ECG revealed CHB with a junctional escape rhythm and a rate of 38

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beats per minute (Figure 1). A Computed Tomography (CT) scan of the chest identified a PE in the right main pulmonary artery, extending into the right interlobular artery (Figure 2). The patient had been taking metoprolol tartrate 100 mg twice daily for hypertension, which was discontinued on arrival. She did not respond to the atropine injection. After consulting with cardiology, the patient began receiving a 5 mg intravenous push of glucagon, followed by a glucagon infusion at 3 mg/hour for potential beta-blocker toxicity, in addition to heparin infusion. The patient continued to exhibit persistent CHB and bradycardia, although her blood pressure remained stable. A cardiac point-of-care ultrasound demonstrated tricuspid regurgitation, but no RV enlargement, septal flattening, paradoxical septal motion, or McConnell's sign. The patient was determined to have an intermediate-low risk PE and no need for advanced treatment, such as systemic or catheter-directed thrombolytics. She was admitted to the intensive care unit to initiate dobutamine infusion for bradycardia and to ensure close hemodynamic monitoring.

The following day, a Transthoracic Echocardiogram (TTE) revealed severe tricuspid regurgitation, pulmonary hypertension (pulmonary systolic pressure 74 mmHg, normal range 15 to 25 mmHg), and preserved RV function (no RV dilation or septal motion abnormality, with tricuspid annular plane systolic excursion measurement of 28.6 mm, normal range 15 to 25 mm, Figure 3). Her Electrocardiogram (ECG) showed Complete Heart Block (CHB) consistently, but her blood pressure and oxygen saturation remained stable. Since no prior TTE for comparison, we reasoned that her severe pulmonary hypertension had developed acutely, given her compensated RV function and adherence to warfarin until its discontinuation two weeks prior. Because of her profound conduction abnormalities associated with severe pulmonary hypertension, that could be the underlying cause of the CHB with no anticipated improvement in the coming days, a shared decision was made to implant a permanent dual-chamber pacemaker. The patient recovered without other complications and was discharged home on anticoagulation therapy (rivaroxaban) two days later.

The literature was searched across multiple databases, including MEDLINE/PubMed, Scopus, Google Scholar, and the Cochrane collaboration database of randomized trials, using search terms such as "pulmonary embolism" or "pulmonary emboli" and "atrioventricular block". The search was conducted up to June 20, 2025, without language limitations. The exclusion criteria included sinus bradycardia without high-degree AV block and Mobitz I AV block. A team of two independent reviewers screened eligible studies and performed data extraction. Continuous variables were presented as mean ± Standard Error (SE), and frequencies were expressed as percentages. The F-test and Chi-square test were applied for comparisons, with a significance level set at $p<0.05$.

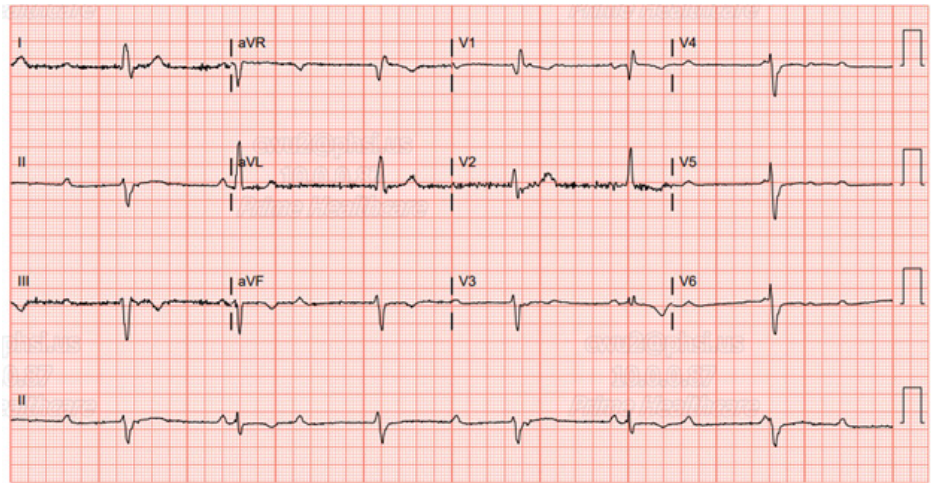

Figure 1: ECG showing complete heart block with bradycardia. A standard 12-lead ECG demonstrated a complete heart block. (Heart rate 38 beats/minute).

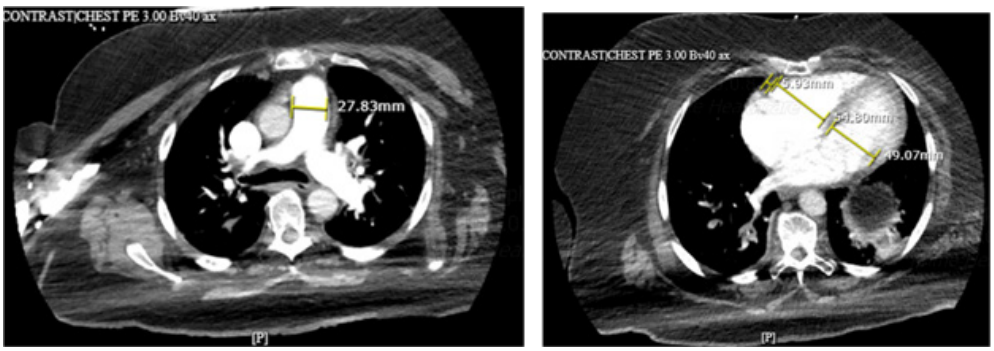

Figure 2: CT PE showing filling defect in the right main pulmonary trunk and lobular pulmonary arteries with right ventricle enlargement. (A): CT chest showed filling defect in the right main pulmonary trunk extending to lobular pulmonary arteries (arrows); (B): Measure of ventricular dimensions showed right ventricular enlargement (RVD/LVD=1.1). **Note:** LV indicates left ventricle; RV, right ventricle.

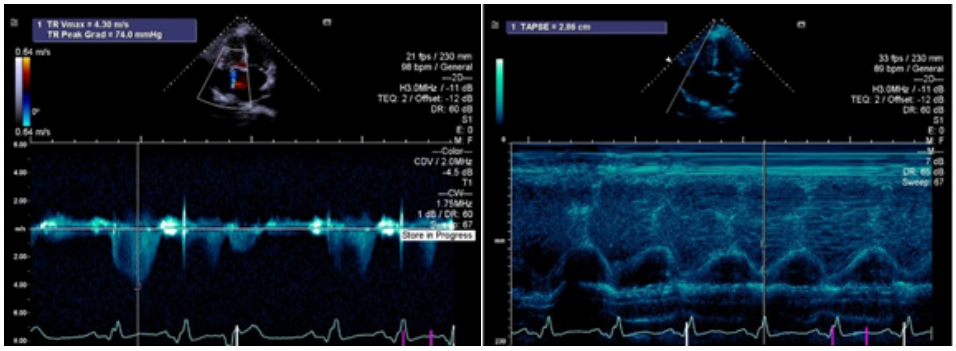

Figure 3: A transthoracic echocardiogram showing severe pulmonary hypertension without right ventricle strain. (A): A Transthoracic Echocardiogram (TTE) showed severe tricuspid regurgitation and pulmonary hypertension (a systolic pulmonary artery pressure of 74mmHg; (B): But preserved RV function as no RV enlargement or septal motion abnormality and an elevated tricuspid annular plane systolic excursion measurement (28.6 mm).

RESULTS

A total of 22 cases of PE with AV block were collected and analyzed (see Supplementary Table 1). Among them, 54.5% (12 cases) presented with persistent CHB, 27.3% (6 cases) exhibited episodic CHB, and 18.2% (4 cases) had Mobitz type II AV block. 54.5% (12 cases) were male, while 45.5% (10 cases) were female. The average age was 68 ± 2.6 years. Patients with persistent CHB had an average age of 70.2 ± 3.6 years, those with episodic CHB averaged 61.8 ± 5.2 years, and those with Mobitz type II AV block averaged 70.8 ± 5.5 years ($p<0.05$ for persistent CHB *versus* episodic CHB).

Among the 22 cases, 50% (11 cases) experienced resolution of Atrioventricular (AV) conduction abnormalities. The resolution rate was 41.7% (5 cases) in the persistent CHB group and 66.7% (4 cases) in the episodic CHB group. Additionally, 36.4% (8 cases) required permanent pacemaker placement, with 75% (6 cases) from the persistent CHB group and 25% (2 cases) from the episodic CHB group. There were three fatalities, resulting in a mortality rate of 13.6%.

DISCUSSION

PE can result in different types of arrhythmias. In our study of 22 cases, approximately 50% experienced persistent CHB, a quarter had episodic CHB, and the remaining cases presented with Mobitz II AV block. This data indicates that, despite the frequent association of PE with specific types of tachycardia arrhythmias, a high-degree AV block can occur in acute PE, albeit infrequently.

The pathophysiology of AV block in PE is not well understood, but it may involve several potential mechanisms. Firstly, the right bundle branch of the His bundle has a superficial subendocardial trajectory in the RV wall and intraventricular septum, making it susceptible to injury from rapid elevations in RV pressure and dilation, which can result in RBBB [2]. Particularly in patients with pre-existing conduction system abnormalities, such as LBBB, there is a risk of developing a combination of RBBB and LBBB, potentially progressing to CHB [2-4]. Secondly, acute RV pressure elevation and volume overload can lead to excessive vagal stimulation, known as the Bezold-Jarisch reflex. Acute PE triggers the right atrial stretch receptors, leading to decreased left ventricular filling, while hypoxia increases heart rate and contractility, stimulating mechanoreceptors and causing bradycardia and AV block [5]. Thirdly, a pulmonary embolic event can induce myocardial ischemia at the atrioventricular node, resulting in ischemia-associated CHB. Additionally, the aging process renders the conduction system more susceptible to rapid changes in RV volume and pressure, with older age being a risk factor for developing CHB. Our data consistently indicate that patients with persistent CHB tend to be older than those with episodic CHB, suggesting that aging contributes to AV block in acute PE.

In our case, the likely mechanism for CHB was injury to the AV node and His bundle due to severe pulmonary hypertension. Although there were no baseline ECHO for comparison, her normal troponin levels and the absence of regional left ventricular wall motion abnormalities made ischemia-related AV block less probable. Her stable sinus node rate and blood pressure reduced the likelihood of the Bezold-Jarisch reflex, and her young age did not support age-related AV block. She had been consistently taking warfarin, which made Chronic Thromboembolic Pulmonary Hypertension (CTEPH) less likely, although preserved RV systolic function can still be observed in patients with CTEPH. Therefore, it is likely that the patient experienced an acute embolic burden, resulting in severely elevated pulmonary artery pressure, which impaired the conduction system and led to CHB. This situation may create a vicious cycle involving pulmonary hypertension, CHB with bradycardia, and RV remodeling (see Figure 4).

Current guidelines recommend a risk-stratified approach to the treatment of PE, with anticoagulation serving as a cornerstone for all risk groups [6]. A multidisciplinary team, including cardiologists, electrophysiologists, and pulmonologists, is typically involved in the decision-making process. Our patient was deemed as intermediate-low risk group based on her stable BP, physiological troponin, very mildly elevated BNP, and no evidence of RV strain in echocardiography, although CT chest revealed mildly increased RV:LV ratio. Accordingly, she was treated with intravenous heparin infusion without advanced treatment, such as systematic thrombolysis or catheter-directed thrombectomy.

The treatment plans for patients with PE complicated by CHB or high-grade AV block should be individualized, with permanent pacemaker placement being the primary intervention. Among our 22 cases, 36.4% required permanent pacemaker placement, while 50% experienced resolution of the AV block following appropriate PE treatment, with a relatively higher resolution rate in the episodic CHB group compared to the persistent CHB group.

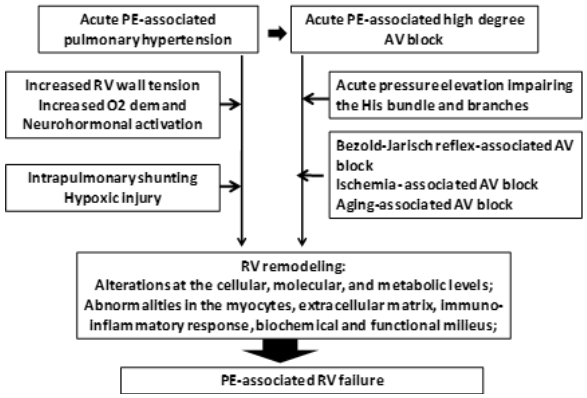

Figure 4: A vicious cycle among pulmonary embolism-associated acute pulmonary hypertension, CHB with bradycardia, and RV remodeling.
Note: PE: Pulmonary Embolism; CHB: Complete Heart Block; RV: Right Ventricle; AV: Atrioventricular.

The resolution of AV block generally occurred within 48 hours after thrombolytic therapy or thrombectomy. Due to the rarity and limited data available, predicting the need for pacemaker placement based on factors such as pulmonary artery pressure, right ventricular dysfunction, and troponin/BNP levels remains challenging. Dobutamine infusion has limitations in resolving high-degree AV block, and patients ultimately required permanent pacemaker placement due to a high risk of recurrence [7,8]. Although the presence of pre-existing AV conduction abnormalities, such as RBBB or LBBB, poses a significant risk for progression to high-degree AV block, our data did not suggest an association between these pre-existing AV issues and the necessity for permanent pacemaker implantation.

An Electrophysiology Study (EPS) can help identify the site of the block and guide treatment decisions. Not all patients with Mobitz type II and/or CHB require an EPS; however, it can provide valuable information for further treatment. Mortality rates in cases of acute PE vary, with our study observing a 13.6% mortality rate, often due to complications such as massive PE and CHB, which can lead to hemodynamic instability, or due to multiple comorbidities.

CONCLUSION

Our study highlights the significance of identifying high-degree AV block in acute PE. Continuous monitoring and timely intervention are essential for effectively managing these cases. Additional research is necessary to refine treatment strategies for patients with acute PE and high-degree AV block.

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ETHICAL COMMITTEE

This study involves human subjects. We declare having received the patient's consent for the case and its publication.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Chaoneng Wu: Writing-original draft, investigation, conceptualization, methodology, formal analysis, reviewing and editing.

Sajith Silva: investigation and data curation.

Anish Kondur: methodology and formal analysis.

Mahi Shah: investigation and data curation.

William E Harder: conceptualization, reviewing and editing.

Sujata Kambhatla: conceptualization, reviewing and editing.

CONFLICT OF INTEREST STATEMENTS

This project doesn't receive any fund. All authors have no conflicts of interest to disclose.

REFERENCES

1. Hsu SH, Ko CH, Chou EH, Herrala J, Lu TC, Wang CH, et al. Pulmonary embolism in United States emergency departments, 2010-2018. Sci Rep. 2023;3(1):9070.
2. Elias J, Kuniyoshi R, Moulin B, Cunha F, Castro E, Nunes A, et al. Syncope and complete atrioventricular block related to pulmonary thromboembolism. Arq Bras Cardiol. 2004;83(5):438-441.
3. Olson PC, Cinelli M, Kurtovic E, Barsoum E, Spagnola J, Lafferty J. Complete atrioventricular block caused by pulmonary embolism: A case report and review of literature. Hear Lung. 2020;49(2):198-201.
4. Kim M, Seo CO, Kim H, Kim HR, Kim K, Kang MG, et al. Case Report: Complete atrioventricular block in an elderly patient with acute pulmonary embolism. Front Cardiovasc Med. 2024;11:1355000.
5. Grubb BP. Clinical practice. Neurocardiogenic syncope. N Engl J Med. 2005;352(10):1004-1010.
6. Marco Z, Behnood B, Jennifer BH, Stefano B, Elisabeth MB, George G, et al. International clinical practice guideline recommendations for acute pulmonary embolism: Harmony, dissonance, and silence. JACC 2024;84:1561-1577.
7. Akinboboye OC, Brown EJ, Queirroz R, Cusi VP, Horowitz LF, Jonas EA, et al. Recurrent pulmonary embolism with second-degree atrioventricular block and near syncope. Am Heart J. 1993;126(3 Pt 1):731-732.
8. Yee P, Reese J. Bradycardia and ST-segment elevation in the setting of acute pulmonary embolism. Chest. 2020;158(4):A2111.