Bilateral Pulmonary Embolism Masked by New-Onset Atrial Fibrillation with Rapid Ventricular Rate: The Role of Mechanical Thrombectomy

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Patient: Male, 70-year-old
Final Diagnosis: Atrial fibrillation • pulmonary embolism
Symptoms: Dyspnea
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course
Background: Management of atrial fibrillation (AF) with rapid ventricular rate in the setting of submassive pulmonary emboli (PE) has not been well defined in the literature. It is challenging as the hemodynamics caused by a PE can change the management of AF. We report a case of bilateral PE masked by new-onset AF with rapid ventricular rate that was treated pharmaceutically and mechanically, with thrombectomy.

Case Report: An 85-year-old man presented with gradual dyspnea and was found to be in AF with rapid ventricular rate (~160-180 bpm). The patient had tachypnea and hypoxia requiring oxygen administration. On physical examination, he had euvolemia. Chest X-ray did not reveal pulmonary vascular congestion. He was started on standard AF management with atrioventricular nodal blockers. Laboratory tests revealed a normal troponin level but mildly elevated B-type natriuretic peptide and lactate. Because his dyspnea was out of proportion to the physical examination, radiographic, and laboratory findings, a D-dimer level was obtained and was elevated. Computed tomography with pulmonary angiogram showed extensive bilateral PE. An echocardiogram (TTE) showed evidence of right ventricular failure. The patient underwent mechanical thrombectomy with clot retrieval, deterring the risk of hemodynamic collapse that would have ensued with atrioventricular nodal blockers monotherapy. On repeat TTE, right ventricular dysfunction was completely resolved and the remaining hospitalization was uneventful.

Conclusions: In patients with concomitant AF with rapid ventricular rate and submassive PE, the use of mechanical thrombectomy, in addition to the standard AF management, could be beneficial in deterring the risk of hemodynamic collapse.

Keywords: Atrial Fibrillation • Pulmonary Embolism • Thrombectomy

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Background

Pulmonary embolism (PE) is a serious cause of cardiovascular death that most commonly occurs as a complication of a deep vein thrombosis (DVT). The clots in question often, but not exclusively, arise from the lower extremities and embolize to the pulmonary arteries.

Atrial fibrillation (AF) is a prevalent arrhythmia that is described in up to 2% to 3% of the general population worldwide [1] and can lead to both right-sided and left-sided heart thrombi.

PE and AF have common risk factors, including advanced age, obesity, high blood pressure, and heart failure. It is not unusual for both conditions to coexist, and clinicians should have a high clinical suspicion, as the medical management can differ.

According to the American Heart Association [2], PE is classified as massive, submassive, or low risk. Massive PE is accompanied with hemodynamic changes such as systolic blood pressure below 90 mmHg for more than 15 min or the need for inotropic therapy. Submassive PE is associated with a systolic blood pressure above 90 mmHg but with signs of right ventricle (RV) dysfunction or myocardial injury/necrosis. Lastly, low-risk PE is not associated with hemodynamic changes, RV dysfunction, or myocardial necrosis [2]. Massive PE is treated with thrombolytic therapy, while submassive and low-risk PE are typically treated with anticoagulation [2].

The criterion standard test for diagnosing an acute pulmonary embolism is computed topography with angiography (CTA) of the chest. When approaching a patient with a suspected PE, the likelihood of the condition is first determined to guide appropriate diagnostic testing [3]. This is done by calculating a pre-test probability using the Wells’ criteria for PE [3]. The scoring system includes 6 variables with different grading points. The patient is considered at low risk of PE if the score is less than 2, at moderate risk if the score is from 2 to 6, and at high risk if the score is more than 6. For low and moderate risk of PE, D-dimer is first obtained and, if elevated, a CTA is obtained [3]. Alternatively, for patients with a low risk of PE (score <2), clinicians can also consider using the 8 PE rule-out criteria and, if the patient meets all criteria, then no further testing, including D-dimer, is needed [3]. In contrast, for patients at a high risk of PE (score >6), D-dimer testing is not recommended, and CTA is performed [3].

The presentation of AF in the setting of submassive PE has been reported in the literature; however, the management has not been well defined. The coexistence of both conditions is clinically significant as the hemodynamics of PE can change the management of AF. In this report, we discuss the case of a bilateral PE presenting as new-onset AF with rapid ventricular rate, managed pharmaceutically and mechanically with thrombectomy.

Case Report

An 85-year-old man with a past medical history of hypertension, benign prostatic hyperplasia, resected melanoma over 20 years ago, and a history of unprovoked deep vein thrombosis (DVT) with suspected noncompliance with apixaban presented to the ED with gradually worsening shortness of breath of a 1-week duration. His symptoms started abruptly and were notable at rest and upon exertion. He denied chest pain, palpitations, paroxysmal nocturnal dyspnea, orthopnea, cough, calf pain, fever, or chills.

On admission, the patient was ill-looking. He was cooperative during history taking but had some trouble communicating his symptoms due to severe conversational dyspnea. He was afebrile with a blood pressure of 150/67 mmHg and a heart rate >160 beats per min. He had tachypnea and hypoxemia, requiring 3 L nasal cannula to keep oxygen saturation above 90%. Electrocardiogram revealed AF with a rapid ventricular rate. On physical examination, the patient was alert and awake but mildly confused. The lungs were clear to auscultation, with no wheezes or rales. His cardiovascular examination was notable for a rapid irregularly irregular rhythm. There was no jugular vein distention, hepatomegaly, or peripheral edema noted. In addition, his neurological and abdominal examinations were unrevealing.

Blood laboratory results were notable for B-type natriuretic peptide of 325 pg/mL, anion gap metabolic acidosis with an elevated lactate level of 2.9 mmol/L, and an acute kidney injury with a BUN/Cr ratio of 20:1. Troponin, electrolytes, and TSH were within normal limits. A chest X-ray showed no evidence of vascular congestion.

Given that the heart rate was >160 beats per min, the patient received in the ED 3 doses of 5 mg intravenous metoprolol and was then started on oral metoprolol 25 mg every 6 h for the management of symptomatic AF. However, on further evaluation by the medical team, his dyspnea was found to be out of proportion to his examination and imaging findings, and therefore a D-dimer was ordered and was >20 (reference range 0-0.50 ug/mL). A subsequent CTA (Figure 1) showed extensive bilateral pulmonary emboli with a completely occlusive thrombus of the right lower lobe segmental and subsegmental pulmonary arteries, a near occlusive thrombus of the right upper lobe proximal segmental pulmonary artery, and a
large clot burden in the distal left main pulmonary artery with subtotal thrombus of the left lower lobe subsegmental and left upper lobe segmental and subsegmental pulmonary arteries. Right heart strain was noted as well.

An urgent transthoracic echocardiogram (TTE) was performed and showed a severely dilated and hypokinetic RV with apical hypercontractility (McConnell’s sign) as well as septal flattening consistent with increased ventricular pressure and volume overload. The right atrium was also severely dilated with elevated pressures with atrial septum bowing from right to left. The left ventricular (LV) systolic function was reduced, with an ejection fraction of 40% to 45%. In addition, there was evidence of LV underfilling and reduced stroke volume with a left ventricular outflow tract velocity time integral (LVOT VTI) as low as 10.5 cm (normal: 20±3 cm).

The patient was started on a heparin drip. Given the reduced stroke volume, as indicated by the low LVOT VTI, the patient was heart rate-dependent for cardiac output, and we could not safely control the rate without further hemodynamic support. Therefore, the decision was to perform a mechanical thrombectomy in the hope of reducing clot burden and improving forward flow. He was transported to the catheterization laboratory for bilateral pulmonary embolectomy to mitigate the risk of hemodynamic deterioration. Multiple clots were retrieved (Figure 2) and an inferior vena cava filter was placed, given the extensive lower extremity clot burden.

Following the procedure, the patient’s symptoms improved and he was saturating well on room air. A repeat TTE done 36 h following the procedure showed resolution of the RV apical hypercontractility and significant improvement of RV size and function.
systolic function. This was also noted by the increase in the tricuspid annular plane systolic excursion (TAPSE), a representative measurement of RV function, from 1.4 to 2.2 cm. The right atrial pressure decreased to 3 mmHg and the atrial septum bowing resolved. He remained in rate-controlled AF after the procedure and was able to tolerate beta-blocker therapy. He was later discharged on metoprolol and oral anticoagulation.

At follow-up 2 months later, the patient was in an asymptomatic rate-controlled AF, and a TTE showed a preserved LV systolic function with an estimated ejection fraction of 70% and an LVOT VTI of 16 to 18 cm. In addition, the RV size and systolic function completely recovered.

Discussion

The hemodynamic complications that ensue from PE depend on the size of the embolus and the degree of obstruction it causes. This leads to an acute increase in pulmonary vascular resistance. In addition, the interruption in blood flow induces hypoxia that results in the release of vasoactive substances, such as serotonin, thromboxane, and histamine, which cause vasoconstriction and amplify the elevation in pressures [4]. Compared to the LV, the RV is much thinner and has a crescent-shaped geometry. This causes intolerance to any abrupt increase in pulmonary vascular pressure, resulting in a linear decline in its stroke volume, leading to ventricular failure [4].

Our patient was in AF with a rapid ventricular rate, which can severely affect the diastolic filling of the LV. Additionally, in the setting of concomitant acute RV failure and underfilled LV with low stroke volume, our patient was showing clinical signs of cardiogenic shock.

A study done on 782 normotensive patients presenting with acute PE found that a TAPSE of 1.6 cm or less was associated with an increase in all-cause and PE-associated mortality [5]. In addition, a retrospective study of 188 patients found that a LVOT VTI of 15 cm or less in patients with acute PE was associated with an increased risk of in-hospital death, cardiac arrest, and shock [6]. It was also associated with an increased need for reperfusion [6]. In our case, LVOT VTI and TAPSE were found to be as low as 10.5 cm and 1.4 cm, respectively.

Therefore, our patient was at a higher risk of a worse clinical outcome. A mechanical thrombectomy, using the Flowtrierve system, was simultaneously performed, as it has been shown in the FLARE trial to significantly reduce RV strain (calculated as RV to LV ratio) by up to 25.1% following the procedure [7].

On repeated TTE, RV size and systolic function completely recovered. In addition, TAPSE and LVOT VTI improved to 2.2 cm and 16 to 18 cm, respectively.

In our case, the patient was started on an atrioventricular nodal blocker in the Emergency Department (ED), which is standard AF management. However, this therapy alone could have decompensated this patient given his severe underlying RV dysfunction and underfilled LV that was heart rate-dependent for cardiac output given the low stroke volume. Therefore, the concomitant use of mechanical thrombectomy helped improve the RV function and deter the risk of hemodynamic collapse.

In addition, this case illustrates the importance of obtaining a TTE, if possible, prior to the initiation of therapy in relatively stable patients presenting with new-onset AF with a rapid ventricular rate, as it would help better understand the intracardiac hemodynamics and establish safe treatment plans.

The use of thrombolytic therapy is another treatment approach for the management of patients with acute PE. While thrombolytic therapy is advised in patients with massive PE to mitigate hemodynamic collapse, its benefit in patients with submassive PE is less well-defined given the increased risk of major and minor bleeding [8]. A meta-analysis of 16 studies was performed to compare the use of thrombolytic therapy and anticoagulation in patients with PE. More than 70% of the included patients had submassive PE. The study found that, while...
thrombolytic therapy had a 47% reduction in mortality, the treatment was associated with a 2.7- and 4.6-fold increase in major bleeding and intracranial hemorrhage, respectively [9]. In addition, the risk of major bleeding was triple in patients ≥65 years compared with in younger patients [9].

Therefore, in our 85-year-old patient with submassive PE, whose heart rate could not be controlled safely without supporting his hemodynamics, the decision was to perform manual thrombectomy rather than treating with thrombolysis therapy. Following the procedure, the patient was symptom-free, and the rest of the hospitalization was uneventful. The patient remained in rate-controlled AF and was discharged on metoprolol and oral anticoagulation.

Conclusions

When dealing with new-onset AF with a rapid ventricular rate, it is important to understand intracardiac hemodynamics and identify masked concomitant underlying conditions, such as pulmonary embolism, prior to initiation of standard therapy with atroventricular nodal blockers. Obtaining a 2-dimensional echocardiogram and testing for troponin, B-type natriuretic peptide, and a D-dimer could be helpful. A coexisting PE, if significant, can cause right heart strain and/or failure as well as shock. Therefore, in addition to standard AF management, mechanical thrombectomy, if appropriate, could be considered. This can reverse structural cardiac changes and prevent hemodynamic collapse.

Declaration of Figures’ Authenticity

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References:

1. Ptaszynska-Kopczynska K, Kluk I, Sobkowicz B. Atrial fibrillation in patients with acute pulmonary embolism: Clinical significance and impact on prognosis. Biomed Res Int. 2019;2019:7846291
2. Jaff MR, McMurtry MS, Archer SL, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: A scientific statement from the American Heart Association. Circulation. 2011;123(16):1788-830
3. Raja AS, Greenberg JO, Qaseem A, et al. Evaluation of patients with suspected acute pulmonary embolism: Best practice advice from the Clinical Guidelines Committee of the American College of Physicians. Ann Intern Med. 2015;163(9):701-11
4. Matthews JC, McLaughlin V. Acute right ventricular failure in the setting of acute pulmonary embolism or chronic pulmonary hypertension: A detailed review of the pathophysiology, diagnosis, and management. Curr Cardiol Rev. 2008;4(1):49-59
5. Lobo JL, Holley A, Tapson V, et al. Prognostic significance of tricuspid annular displacement in normotensive patients with acute symptomatic pulmonary embolism. J Thromb Haemost. 2014;12(7):1020-27
6. Yuriditsky E, Mitchell OIL, Sibley RA, et al. Low left ventricular outflow tract velocity time integral is associated with poor outcomes in acute pulmonary embolism. Vasc Med. 2020;25(2):133-40
7. Tu T, Toma C, Tapson VF, et al. A prospective, single-arm, multicenter trial of catheter-directed mechanical thrombectomy for intermediate-risk acute pulmonary embolism. JACC Cardiovasc Interv. 2019;12(9):859-69
8. Igneri LA, Hammer JM. Systemic thrombolytic therapy for massive and submassive pulmonary embolism. J Pharm Pract. 2020;33(1):74-89
9. Chatterjee S, Chakraborty A, Weinberg I, et al. Thrombolysis for pulmonary embolism and risk of all-cause mortality, major bleeding, and intracranial hemorrhage: A meta-analysis. JAMA. 2014;311(23):2414-21.