

## Is a Cath Really Necessary?

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### Abstract

*Pulmonary embolism is associated with elevated troponin levels. Many conditions are known to cause this and are thus regarded to as an NSTEMI in the setting of demand ischemia. We present a 78-year-old male with multiple comorbidities who is found to have an NSTEMI in the setting of a pulmonary embolism. The patient subsequently undergoes catheterization and is found to have mild coronary artery disease. Catheterizations are procedures that come with their own set of risks and may not necessarily need to be performed if troponinemia is deemed to be from demand ischemia. Currently, there is no cut off point for which patients who present with an NSTEMI secondary to these other underlying conditions should undergo catheterization. Further research should be done to determine troponin cut offs in pulmonary embolism and the need for additional evaluation via catheterization.*

**Keywords:** Troponinemia, NSTEMI, Pulmonary Embolism, Coronary Angiography

### Learning Objective

Pulmonary embolism is known to be associated with troponin elevations and result in NSTEMI in the setting of demand ischemia. Formal guidelines should be in place for whether these subgroups of patients require catheterization or if catheterization affects outcomes.

### Introduction

Pulmonary embolism is known to cause some variations in troponin levels. It is also known that troponins can be elevated from a variety of conditions, however do all patients who present with an NSTEMI as well as, rising troponins need to have a catheterization?

### Case Presentation

A 78-year-old male with past medical history of Merkel cell carcinoma status post radiation and chemotherapy with spinal metastasis, atrial fibrillation previously on apixaban, chronic kidney disease stage III, hypertension, heart failure with preserved ejection fraction and hyperlipidemia initially presented with a chief complaint of shortness of breath and was admitted as a code STEMI. He denied any chest pain but had trouble swallowing. Physical exam findings were remarkable for an irregularly irregular rhythm and bilateral crackles over the lung bases. His labs were significant for a BNP of 2600 and a troponin I of 16. EKG showed ST changes over the anterolateral as well as, inferior leads with a bi-fascicular block (Figures A and B). CT angiogram of the chest findings were consistent with an acute pulmonary embolism at the junction of the anterior segment of the right upper lobe, along the

medial as well as, lateral segmental branches of the right middle lobe (Figure C). The patient was immediately started on a heparin drip.

Echocardiogram showed an ejection fraction of 55-60%, mild dilation of the right ventricle with moderate reduction in systolic function and an estimated peak pressure of 28mmHg. Troponins continued to be elevated between 16 and 18. The patient began to deteriorate during his hospital stay and subsequently developed ventricular tachycardia. He was transferred to the ICU for an amiodarone drip as well as, intubation. The patient underwent catheterization which demonstrated mild systolic dysfunction with mild anterolateral hypokinesis, normal left end diastolic pressure and no evidence of flow-limited coronary artery disease (Figure G and Figure H). The patient's telemetry showed sustained ventricular tachycardia. EKG findings initially showed a bifascicular block which later transitioned into a wide left bundle branch block concerning for intracardiac electrical disease warranting the need for pacemaker placement (Figures D, E, F and I). There were plans for IVC filter placement as the patient would likely be unable to tolerate any anticoagulation due to his history of GI bleed for which apixaban was stopped for two weeks prior to presentation. The patient expired secondary to acute hypoxic respiratory failure in the setting of acute pulmonary embolism.

### Discussion

Troponin elevation can occur in a variety of conditions that go beyond acute coronary syndromes. We know that troponins are used as markers of myocardial damage however we know that these

protein complexes are found in both skeletal and cardiac muscle [1]. These elevations can be due to irreversible and reversible cell damage. The relation to noncardiac causes is due to other conditions leading to stress on the myocardium [2]. Troponins may be elevated from sympathetic nervous system activation, cytokine release, infections, etc [3]. In the case of a pulmonary embolism, elevations in troponin occur due to the sudden increase in oxygen demand causing strain on the heart muscle [4].

Current research shows that acute pulmonary embolism may present with elevated troponins. Pulmonary embolism will usually show signs of pulmonary hypertension on echocardiogram with typically normal EKG findings. However, there are exceptions to this rule as presented in this case and in the setting of multiple comorbidities. This case illustrates the importance of elevated troponin levels and its association to acute pulmonary embolism [2].

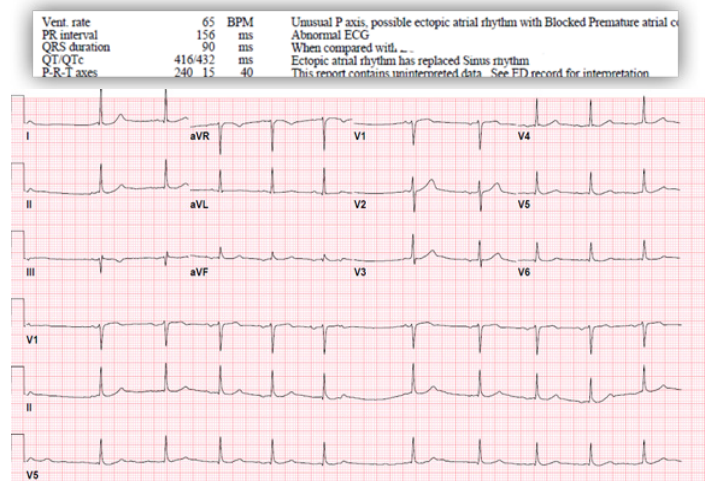
Catheterization generally is conducted for patients who have cardiac symptoms or its equivalent for whom there is a possible narrowing or blockage causing the symptoms, for dysfunctional valves, prior to surgical intervention (i.e.: bypass), etc. This procedure however, comes with risks that include but are not limited to bleeding, arrhythmias, heart attack, stroke, damage to a blood vessel or nerve, allergic reaction, and even death [5]. In patients who are found to have a pulmonary embolism, catheterization may measure atrial and pulmonary pressures as well as, differentiate from coronary artery disease (in the setting of troponin elevations as seen in this patient) [6].

**Conclusion**

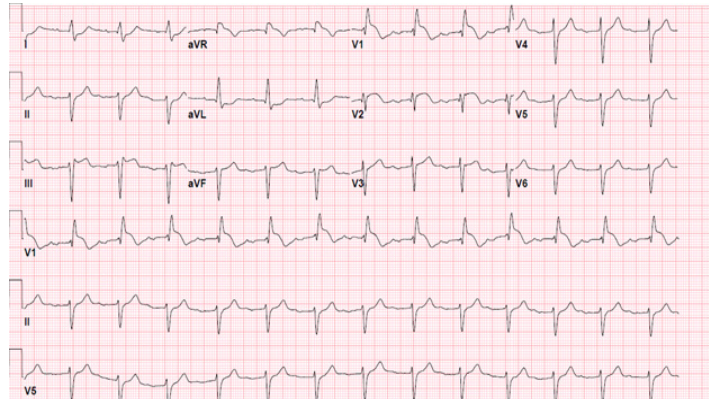
In situations like these, it would be favorable to have a cutoff value for troponin levels to differentiate those who undergo catheterization and those who do not. Patients with multiple comorbidities as seen here make this decision a complex one. However, there have been no studies to date on the effectiveness of catheterization in these patients and its impact on management. Further studies should be conducted to analyze the need for catheterization in this subset population and how it will impact subsequent management.

Supplemental Materials/Figures and Legends:

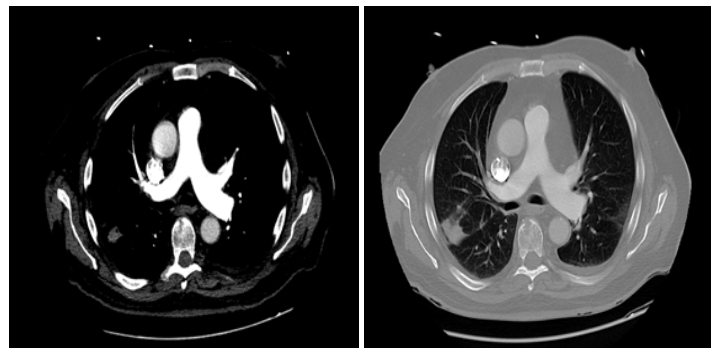
**Figure A&B:** EKG on admission; ST changes over the anterolateral and inferior leads and bifascicular block



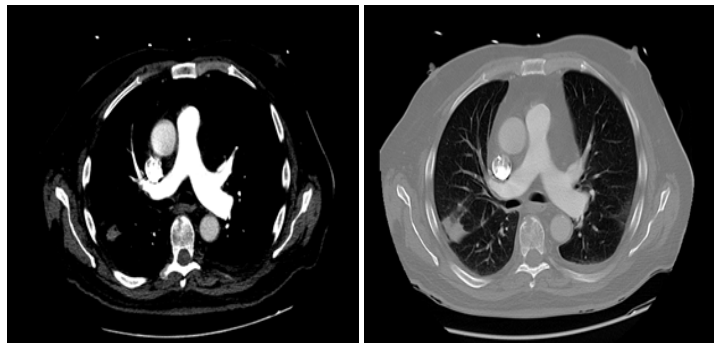
Sinus rhythm with 1st degree A-V block  
 Right bundle branch block  
 Left anterior fascicular block  
 \*\*\* Bifascicular block \*\*\*  
 Abnormal ECG  
 When compared  
 Sinus rhythm has replaced Ectopic atrial rhythm  
 (RBBB and left anterior fascicular block) is now Present



**Figure C:** CTA findings of an acute pulmonary embolism at the right upper and middle lobe



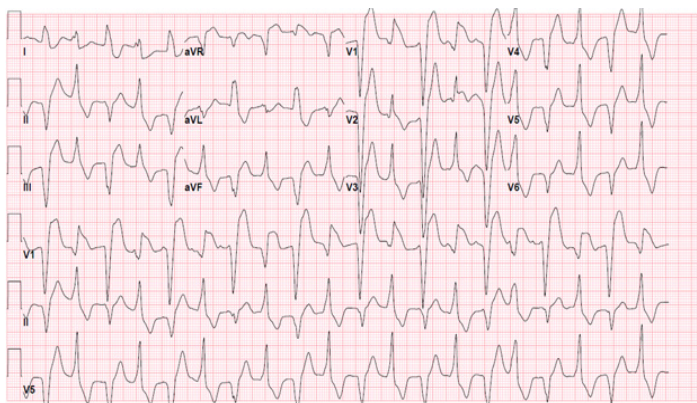
**Figure D:** EKG demonstrating a new onset LBBB



**Figure E:** EKG findings showing Ventricular Tachycardia

Vent. rate	123	BPM	Undetermined rhythm
PR interval	*	ms	Non-specific intra-ventricular conduction block
QRS duration	138	ms	Septal infarct, age undetermined
QT/QTc	382/546	ms	Marked T wave abnormality, consider inferior ischemia
P-R-T axes	* 75 260		Abnormal ECG

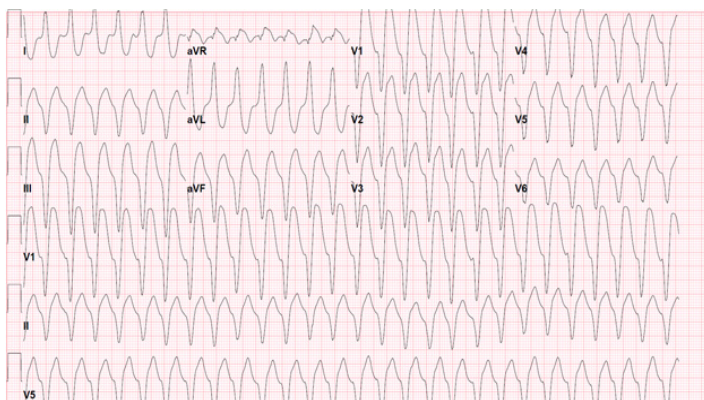




**Figure F:** EKG demonstrating AV dissociation

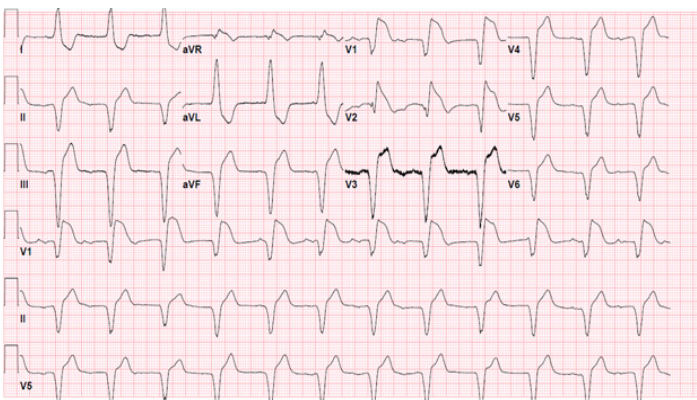
Vent. rate	165	BPM	Wide QRS tachycardia
PR interval	*	ms	Left axis deviation
QRS duration	146	ms	Left ventricular hypertrophy with QRS widening and repolarization abnormality
QT/QTc	276/457	ms	Lateral infarct , age undetermined
P-R-T axes	* -62	116	Inferior infarct , age undetermined

Abnormal ECG  
When compared ...  
Wide QRS tachycardia has replaced Wide QRS rhythm

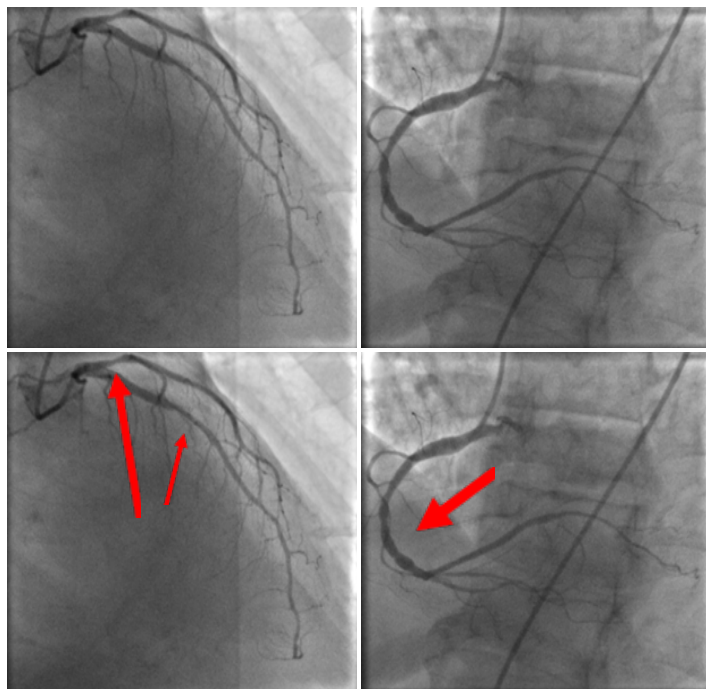


**Figure G:** Angiography Left image corresponds to LCX and LAD, Right image corresponds to RCA, Mild coronary disease appreciated

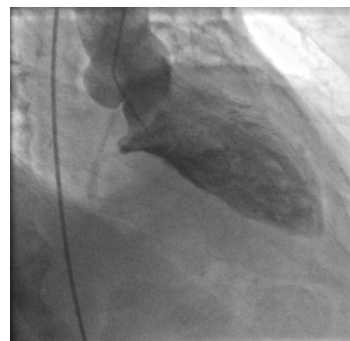
Sinus rhythm with A-V dissociation and Wide QRS rhythm  
Left axis deviation  
Left ventricular hypertrophy with QRS widening and repolarization abnormality  
Lateral infarct , age undetermined  
Inferior infarct , age undetermined  
Abnormal ECG



**Figure H:** Angiography, EF estimate via angiography



**Figure I:** AV dissociation with possible acute STEMI



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#### References

1. Sabrina Arshed, Hong Xiu Luo, Shoaib Zafar, Kalyani Regeti, Nilma Malik, et al. (2015) "Elevated Troponin I in the Absence of Coronary Artery Disease: A Case Report With Review of Literature." *Journal of Clinical Medicine Research* 7: 820-824.
2. Conroy S, I Kamal, J Cooper (2004) "Troponin Testing: Beware Pulmonary Embolus." *Emergency Medicine Journal* 21: 123-124.
3. Skeik Nedaa, Deevia Chandrakant Patel (2007) "A Review of Troponins in Ischemic Heart Disease and Other Conditions." *International Journal of Angiology* 16: 53-58.
4. Tanindi Asli, Mustafa Cemri (2011) "Troponin Elevation in

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Conditions Other than Acute Coronary Syndromes.” *Vascular Health and Risk Management* 7: 597-603.

5. “What Happens during Cardiac Catheterization?” (2016) *InformedHealth.org*. U.S. National Library of Medicine.
6. Willman Vallee L (1965) “Cardiac Catheterization in the Diagnosis of Pulmonary Embolism.” *Archives of Surgery* 91: 25-28.

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