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# A Single Test, A Double Threat: Pulmonary Embolism and Coronary Stenosis Diagnosed with TRO-CT: Case Report

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**Abstract**
Pulmonary embolism (PE) and coronary stenosis are cardiovascular conditions with overlapping pathophysiological pathways, including coagulation activation, systemic inflammation, and endothelial dysfunction. This case presents an opportunity to explore the complex relationship between concurrent PE and coronary stenosis while evaluating the most effective diagnostic strategies for their simultaneous identification and management. Case presentation: A 77-year-old male with a history of ischemic stroke presented with worsening dyspnea and chest discomfort. The patient was discharged after hospitalization for non-ST-elevation acute coronary syndrome (NSTEACS) without revascularization. Discussions: This case report highlights the simultaneous occurrence of pulmonary embolism (PE) and coronary stenosis, emphasizing the diagnostic and therapeutic challenges. We utilized Triple-rule-out Computed Tomography (TRO CT) to effectively detect both coronary stenosis and pulmonary embolism in a single imaging examination, enabling simultaneous diagnosis of these conditions. TRO CT is a highly valuable diagnostic tool in cases of complex chest pain, particularly when the underlying cause is unclear and may stem from multiple etiologies. Special emphasis is placed on distinguishing between type I and type II myocardial infarction, elaborating on the different diagnostic approaches for each type and underscoring the importance of accurate identification to determine the most appropriate therapeutic strategy. Conclusions : This case report highlights the utility of Triple Rule-Out Computed Tomography (TRO CT) in simultaneously diagnosing pulmonary embolism (PE) and coronary stenosis in patients with complex chest pain.

**Keywords:** Triple Rule-Out CT, Pulmonary Embolism, Coronary Stenosis, Chest Pain

## Introduction

Chest pain is one of the most common symptoms encountered in emergency departments (EDs), accounting for approximately 5–20% of all patient visits [1]. Despite its high prevalence, the initial evaluation of chest pain remains a significant diagnostic challenge due to the broad spectrum of potential etiologies, ranging from benign musculoskeletal pain to life-threatening cardiovascular emergencies. Differentiating among these causes is crucial for timely intervention and appropriate management. However, reliance solely on patient history, early biomarkers, and electrocardiography (ECG) findings is often insufficient to determine the underlying pathology[1,2]. This diagnostic uncertainty underscores the need for advanced imaging modalities capable of simultaneously evaluating multiple potential causes of acute chest pain.

Pulmonary embolism (PE) is a critical and potentially fatal condition caused by the obstruction of the pulmonary arteries, most commonly due to thrombi originating from the deep venous system, particularly in the lower extremities[3]. As a component of venous thromboembolism (VTE), PE has an estimated incidence of 104–183 cases per 100,000 person-years in the United States, with a rising trend among older populations[4]. The clinical presentation of PE varies widely, often including dyspnea, pleuritic chest pain, tachycardia, and, in severe cases, hemodynamic instability. Given its potentially fatal consequences, rapid and accurate diagnosis of PE is paramount to improving patient outcomes.

Similarly, acute coronary syndrome (ACS), which includes unstable angina and myocardial infarction (MI), remains a leading cause of morbidity and mortality worldwide. Coronary artery disease (CAD) is frequently associated with atherosclerosis and thrombotic occlusion, leading to myocardial ischemia and infarction. Notably, PE and CAD share

overlapping pathophysiological pathways, including coagulation activation, systemic inflammation, and endothelial dysfunction, which may contribute to their simultaneous occurrence in certain patients[5,6]. The coexistence of these conditions presents a significant diagnostic and therapeutic challenge, as their clinical manifestations can be similar, leading to potential misdiagnosis or delayed treatment.

A comprehensive diagnostic approach is essential when evaluating patients with complex chest pain presentations, particularly when multiple life-threatening conditions are suspected. Triple Rule-Out Computed Tomography (TRO CT) has emerged as a valuable imaging modality in this context. TRO CT is a specialized contrast-enhanced CT angiography protocol designed to simultaneously assess for three major life-threatening causes of chest pain: PE, acute coronary syndrome (ACS), and acute aortic dissection (AAD)[7]. By enabling the detection of multiple cardiovascular pathologies in a single imaging session, TRO CT enhances diagnostic efficiency, reduces the need for multiple tests, and facilitates timely intervention.

This case report presents a rare instance of concurrent PE and significant coronary stenosis diagnosed using TRO CT in a patient presenting with acute chest pain and dyspnea. The case highlights the critical role of TRO CT in expediting diagnosis and guiding appropriate treatment strategies. Furthermore, this report underscores the importance of considering dual pathology in patients with complex cardiovascular presentations and the need for a multidisciplinary approach in managing such cases.

**Case Presentation**

A 77-year-old male with a history of ischemic stroke was referred to the hospital with progressively worsening shortness of breath that had developed one day before admission. The dyspnea was aggravated by physical activity and relieved by rest. The patient denied paroxysmal nocturnal dyspnea and orthopnea. He also reported chest discomfort (VAS 4) accompanied by cold sweats. There was no history of cough or hemoptysis. One day before admission, he had been discharged after a five-day hospitalization for non-ST-elevation acute coronary syndrome (NSTEACS) without undergoing revascularization. Before this episode, the patient had been physically active and had no history of prior surgeries.

On physical examination, his vital signs were within normal limits, with no signs of hemodynamic instability. There were no signs of jugular vein distension, rales, murmurs, or lower limb edema. Other systemic examinations were unremarkable.

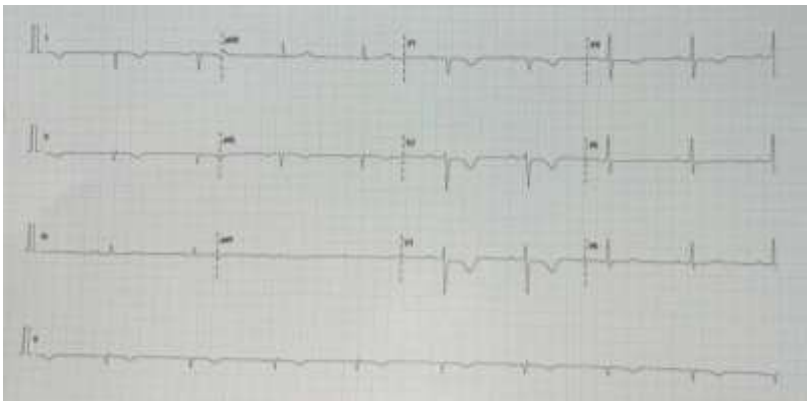

**Figure 1.** Electrocardiographic (ECG) findings demonstrating an S1Q3T3 pattern, characterized by a deep S wave in lead I, a Q wave in lead III, and T-wave inversion in lead III, which is suggestive of pulmonary embolism (PE).

Electrocardiography (ECG) revealed a deep S wave in lead I, a Q wave in lead III, and T-wave inversion in lead III, forming an S1Q3T3 pattern as shown in [figure 1]. Additional T-wave inversions were observed in leads V1–V6, I, and II. A chest X-ray showed Palla's sign, indicating enlargement of the right descending pulmonary artery branch, without increased bronchopulmonary markings. Laboratory investigations showed elevated high-sensitivity troponin I and D-dimer.

Transthoracic echocardiography (TTE) demonstrated right ventricular dilation with reduced right ventricular function (. There was no evidence of McConnell's sign or intracardiac thrombus. Left ventricular wall motion abnormalities were noted in the inferior and posterior regions. Lower limb Doppler ultrasound revealed a thrombus in the femoral vein (FV) with partial compressibility.

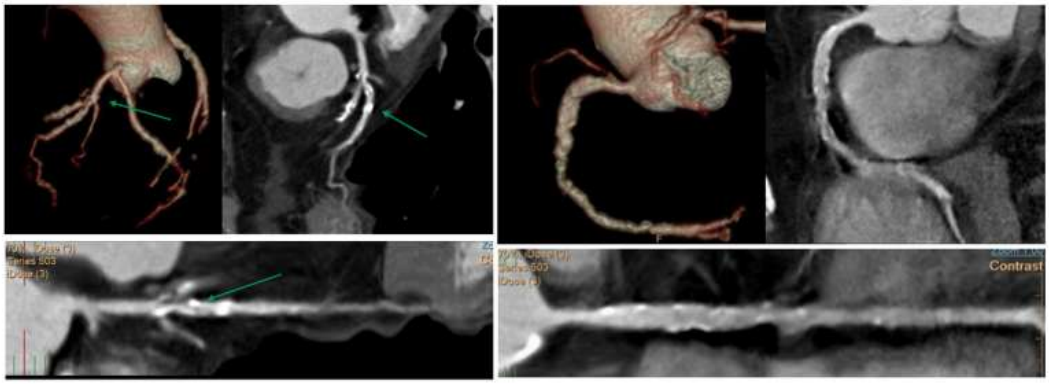

**Figure 2**. CT Coronary Angiography (CCTA) **revealed a** calcified plaque with luminal stenosis (60–70%) in the proximal left anterior descending (LAD) artery and a mixed plaque with severe stenosis (90%) at the ostium of the first diagonal (D1) branch.

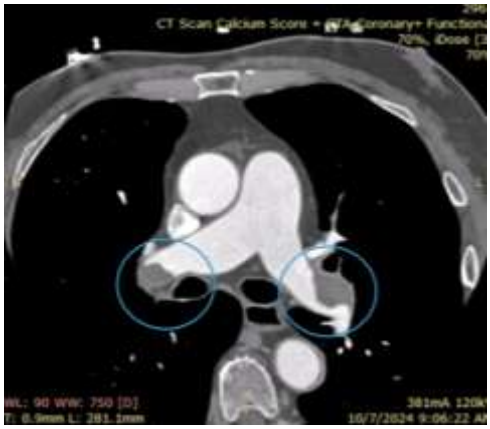

**Figure 3.** CT Pulmonary Angiography (CTPA) revealed a filling defect in the left pulmonary artery (LPA) and right pulmonary artery (RPA), indicative of pulmonary embolism (PE).

Given these findings, a Triple Rule-Out Computed Tomography (TRO CT) was performed. The scan confirmed filling defects in the left pulmonary artery (LPA), left upper lobe segmental branches, left lower lobe segmental branches, right pulmonary artery (RPA), right upper lobe segmental branches, right middle lobe segmental branches, and right lower lobe segmental branches, consistent with pulmonary embolism (PE). Additionally, significant coronary stenosis was identified, including significant stenosis (60-70%) in the proximal left anterior descending (LAD) artery and a mixed (90%) ostial LAD stenosis at the ostium of the second diagonal (D2) branch as shown in [figure 2 and 3].

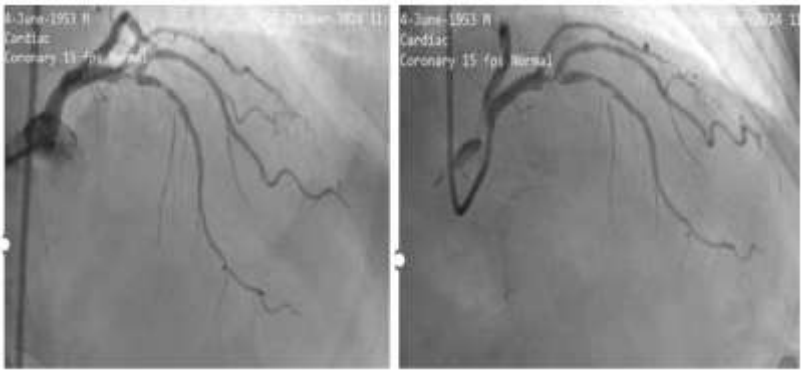

**Figure 4.** Coronary Angiography Findings shows significant stenosis in the proximal to mid left anterior descending (LAD) artery, with a maximum stenosis of 95% in the proximal LAD.

The patient was treated with subcutaneous fondaparinux 7.5 mg once daily for five days and underwent diagnostic coronary angiography (DCA), which confirmed significant stenosis (95%) in the proximal LAD and a non-significant 30% stenosis in the proximal right coronary artery (RCA) as shown in [figure 4]. Subsequently, a stent was placed in the proximal LAD. The patient showed significant clinical improvement following treatment and was discharged in stable condition after five days of hospitalization.

**Discussion**

This patient was initially suspected of having non-ST-elevation myocardial infarction (NSTEMI) based on clinical symptoms, elevated high-sensitivity troponin I , and ischemic ECG findings, including T-wave inversions in leads V1–V6, I, and II (4). However, the presence of an S1Q3T3 pattern raised suspicion for pulmonary embolism (PE). While highly specific (80–90%), its low sensitivity (15–25%) necessitated further evaluation[6].

Additional imaging findings supported the PE diagnosis. Chest X-ray (CXR) revealed Palla's sign as shown in figure 2, indicating right descending pulmonary artery enlargement, present in ~19% of PE cases[7]. Transthoracic echocardiography (TTE) showed right ventricular dilation and dysfunction, consistent with PE-related right heart strain[7]. Other echocardiographic markers, including right heart thrombus (~4% of PE cases, linked to high early mortality)[3] and the 60/60 sign (pulmonary vein acceleration time <60 ms, tricuspid regurgitation velocity <3.9 m/s), further supported the diagnosis.

Lower limb Doppler ultrasound confirmed deep vein thrombosis (DVT) in the femoral vein (FV) with partial compressibility, further supporting the PE diagnosis. Given that ~70% of PE cases originate from DVT, its presence in a patient with clinical suspicion of PE is often sufficient to initiate anticoagulation therapy without waiting for confirmatory CT pulmonary angiography (CTPA)[3].

In complex chest pain cases, Triple Rule-Out Computed Tomography (TRO CT) is a valuable tool for detecting PE, acute coronary syndrome (ACS), and aortic dissection. It offers high sensitivity (86–100%) and negative predictive value (93–100%) for ACS, along with 71.4% sensitivity and 89.5% specificity for PE and 93.8% sensitivity for aortic dissection[1,8]. ECG-gated imaging enhances coronary artery visualization by minimizing cardiac motion. Although ESC guidelines do not suggest TRO CT due to the need for further risk-benefit evaluation[1,9,10], in this case, it effectively confirmed 90% ostial LAD stenosis and PE, facilitating timely intervention. TRO CT, as described by Fraunfelder and Halpern, employs a broader scan range and a biphasic contrast injection technique to optimize imaging of the coronary, pulmonary, and aortic vasculature[1,11]. However, concerns remain regarding higher radiation doses and contrast volume compared to coronary CT angiography (CCTA)[12,13].

TRO CT identified both PE and significant LAD stenosis, necessitating careful differentiation between Type I and Type II myocardial infarction (MI) to guide appropriate management[3,5,14]. Type I MI results from plaque rupture and coronary thrombosis, requiring urgent revascularization, while Type II MI results from oxygen supply-demand imbalance due to systemic factors such as PE-related right heart strain[5,15].

According to the Fourth Universal Definition of Myocardial Infarction (2018), MI is defined as acute myocardial injury with ischemic evidence, marked by troponin elevation (>99th percentile upper reference limit) with at least one of the following: ischemic symptoms, ECG changes, Q waves, imaging evidence of myocardial loss, or intracoronary thrombus on angiography[5,16]. While this patient met these criteria, distinguishing between Type I and Type II MI was challenging. The 90% LAD stenosis suggested Type I MI, but the presence of PE-induced right ventricular strain raised the possibility of Type II MI. ESC guidelines highlight this diagnostic gap, emphasizing the need for better risk stratification tools before deciding on invasive strategies[5,15]. Recent literature from ESC (2024) proposes an algorithm for MI classification, prioritizing initial troponin assessment, ischemic burden evaluation, and identification of systemic contributors to oxygen imbalance[2,17].

**Conclusion**

This case underscores the diagnostic challenges in patients presenting with simultaneous PE and ACS-like symptoms. TRO CT was instrumental in confirming both conditions, guiding the decision for anticoagulation and coronary intervention. However, caution is warranted when using TRO CT indiscriminately, given its higher radiation and contrast exposure. Future studies should refine TRO CT indications to optimize its use in complex chest pain syndromes. Additionally, differentiating Type I from Type II MI remains a clinical challenge, requiring multimodal assessment and individualized treatment strategies.

**Declaration of patient consent**

The authors certify that the patient has obtained an appropriate patient consent form. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

**Conflicts of interest**

The authors declare no conflicts of interest.

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