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Acute Pulmonary Embolism Ignored and Under Diagnosed Killer: A Case Report

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Abstract

Background Pulmonary Embolism (PE) remains a clinically challenging diagnosis, more often ignored & missed than diagnosed & treated, with high incidence in its incidental discovery at autopsy over the past 30 years. PE should be in the top of differentials of every dyspnoea event that presents at an emergency department. We describe a case of 34- year- old man with symptoms of chest pain & dyspnoea who initially diagnosed & treated as non-

ST elevated MI but later diagnosed as acute pulmonary embolism. This case report emphasizes early diagnosis and treatment to avoid fatal outcome.

Case Summary A 34 years old normotensive & non-diabetic man presented with central crushing chest pain associated with profuse sweating for 12 hours prior to presentation to emergency department (ED). After initial assessment he was diagnosed as a case of non-ST elevated myocardial infarction (NSTEMI) with elevated Troponin-I & was shifted to coronary care unit (CCU) for further evaluation & management. His left ventricular ejection fraction (LVEF) was 60%, but dilated RA & RV with severely impaired RV function. Cardiac catheterization revealed normal epicardial coronary arteries. On the course of treatment, he was found to have a large saddle shaped thrombus in the main pulmonary trunk bi-furcation causing acute pulmonary embolism following deep vein thrombosis (DVT). The patient was treated conservatively & recovered well before discharge.

Conclusion Acute pulmonary embolism is often missed & ignored clinical entity which has fatal outcome including sudden cardiac death (SCD). This case report emphasizes development of clinical suspicion among the clinicians for early diagnosis and treatment to avoid fatal outcome.

Keywords: Acute pulmonary embolism, Acute Coronary Syndrome (ACS), NSTEMI, Saddle thrombus, venous thrombosis, sudden cardiac death (SCD).

Learning Points

- Acute pulmonary embolism (PE) is rare but can attribute to potentially fatal complication.
- It is very much challenging to diagnose.
- This clinical entity may mimic with acute coronary syndrome (ACS).
- This is more often ignored & missed than diagnosed & treated.
- Clinical presentation predicts the outcomes.

Introduction

Pulmonary embolism (PE) ranges from incidentally found from clinically unimportant occurrences to sudden cardiac death (SCD). Virchow's triad that ranges from local trauma to the vessel wall, hypercoagulability and stasis of blood leads to thrombus formation in the leg veins[1].

As thrombi form in the deep veins of lower extremities, pelvis or arms, that may dislodge and embolize to the right side of heart then to the pulmonary trunk and consequently further distal vessels with potentially fatal outcomes. The most common sources of pulmonary emboli are the pelvic veins or deep veins of the thigh[2]. Pulmonary arterial obstruction by clot causes dilatation, dysfunction, and ischemia of the right ventricle[1].

Pulmonary embolism and deep venous thrombosis are responsible for more than 250,000 hospitalizations and approximately 50,000 deaths per year in the United States. Because it is difficult to diagnose, the true incidence of pulmonary embolism is unknown, but it is estimated that approximately 650,000 cases occur annually[1]. Despite this high incidence, the diagnosis of pulmonary embolism continues to be difficult primarily because of the notorious varieties of symptoms and signs in its presentation[2].

ACS describes the range of myocardial ischemic states that includes UA, NSTEMI or STEMI. The diagnosis and classification of ACS is based on a thorough review of clinical features, including ECG findings and biochemical markers of myocardial necrosis**3**. The term MI (myocardial infarction) is used when there is evidence of myocardial necrosis in the setting of acute myocardial ischemia. STEMI is differentiated from NSTEMI by the presence of persistent ECG findings of ST segment elevation[4].

Coronary Heart Disease (CHD) is responsible for more than half of all cardiovascular incidences in individuals. During the past several years, the rates of hospitalization for MI and mortality associated with CHD have decreased. The decline in CHD mortality is partially reflective of the change in the pattern of clinical presentations of ACS[5].

There has been a substantial reduction in the incidence of STEMI and a subsequent increase in the incidence of NSTEMI[6]. The research team believes that there is room for more improvement in the prevention and management of ACS.

cTnI is 100% tissue-specific for the myocardium. cTnI has shown to be a very sensitive and specific marker for AMI[7-10]. The early release kinetics for cTnI is similar to those of CK- MB**11**. Although cTnI is 100% tissue specific for the myocardium, there are also some causes of non-cardiac increase of cardiac Troponin-I. The followings are the non-cardiac causes those can increase cTnI[12]:

- Pulmonary embolus
- Myocarditis
- Cardiac contusion
- Congestive heart failure
- Chemotherapy (Adriamycin, 5-fluorouracil)
- Cardioversion or radiofrequency ablation
- Septic shock
- Extreme endurance athletics
- Renal failure

We present the case of a patient with pulmonary embolism presented with features of acute coronary syndrome, later treated as a case of non- ST elevated MI with normal coronaries and ultimately found with a large saddle thrombus in main pulmonary artery causing acute pulmonary embolism. This case report emphasizes development of clinical suspicion among the clinicians for early diagnosis and treatment to avoid fatal outcome.

Timeline

Days Events

Day 1: Acute non-ST segment elevation myocardial infarction. Initial management with anti-ischemic & low molecular weight heparin.

Other baseline investigations were done. Prepared for revascularization due to persistent chest pain & sub normal oxygen saturation.

Coronary angiogram revealed normal epicardial coronary arteries.

Day 2: Still on-going chest pain with failure to maintain adequate saturation. Immediate detail transthoracic echocardiogram done which grew the suspicion of acute pulmonary embolism. CT pulmonary angiogram was done which revealed large saddle shaped thrombus in main pulmonary artery bi-furcation. Immediate starting of unfractionated heparin infusion with monitoring of APTT.

Day 3: Substantial clinical improvement observed & heparin transfusion kept ongoing with monitoring of APTT.

Day 4: Clinical improvement got accelerated & heparin transfusion kept ongoing with monitoring of APTT.

Day 5: Patients repeat echocardiography was done which revealed improvement of right heart function. APTT is desired level. As overall improvement of the patient observed & he was discharged from the hospital with oral anticoagulation with NOAC.

Case presentation

Clinical Course

A 34 years old normotensive & non-diabetic man presented with central crushing chest pain associated with profuse sweating for 12 hours prior to presentation to emergency department (ED). He also complained of acute shortness of breath for 01 hour on the day of admission. On examination, he looked quite unwell and diaphoretic. His Pulse was 120/min & regular, BP was 150/90. His JVP was distended. On cardiac auscultation, his pulmonary component of S_2 was soft. Chest auscultation revealed vesicular breath sound with fine bibasilar crackles. He was initially treated with anti-ischemic & pain killers with high flow oxygen to maintain satisfactory oxygen saturation. All the initial labs were sent including Covid RT- PCR.

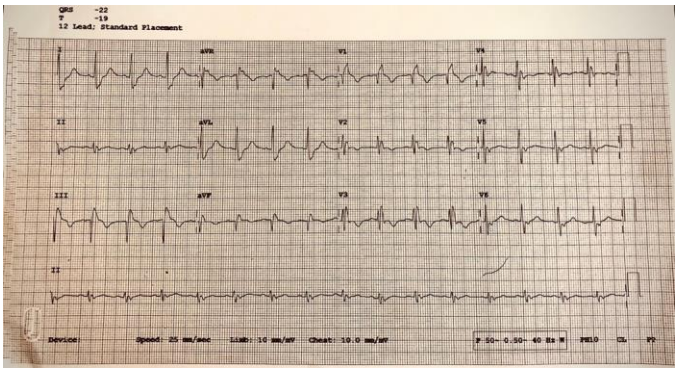

Figure 1: 12-lead electrocardiogram showing RBBB with $S_1Q_3T_3$.

He was diagnosed as a case of non-ST elevated MI with acute left ventricular failure & was immediately transferred to coronary care unit. A decision of conservative treatment was taken considering all the clinical conditions of the patient. Then he was started with low molecular weight heparin (LMWH). But despite given treatment, the patient's clinical condition did not improve. Meanwhile his Covid RT-PCR report came & it was found to be negative. His initial lab parameters showed Hb: 13.3 gm/dl, S. Creatinine: 1.08 mg/dl, Na+: 143 mmol/L, K+: 3.9 mmol/L, CRP: 4.56 mg/dl, TLC: 9.73 x 109/L, Platelet: 229 x 109/L, High-sensitive Troponin-I: 1176 ng/L, D-dimer: 4982 µg/L. He complained of persistent chest pain & shortness of breath. His oxygen saturation was maintained with high flow oxygen. His chest X-ray showed cardiomegaly, fullness of pulmonary bay with congested lung fields.

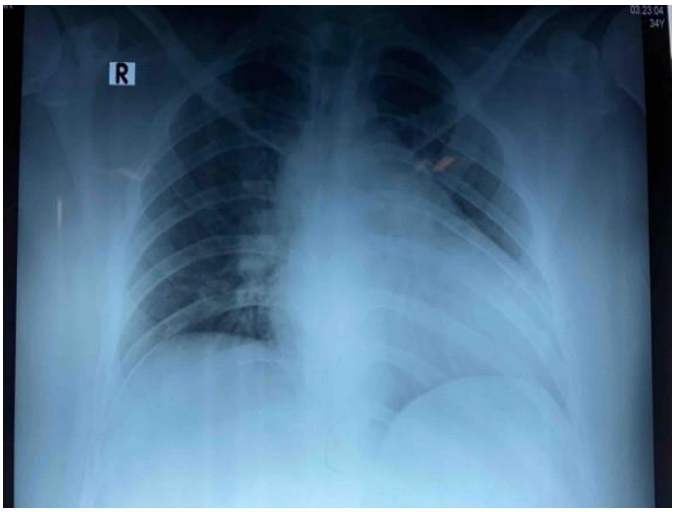

Figure 2: Chest X-ray showed cardiomegaly, fullness of pulmonary bay with congested lung fields.

Then a coronary angiogram (CAG) decision was made & the patient was taken to the cath-lab. Surprisingly, his CAG report revealed normal epicardial coronary arteries.

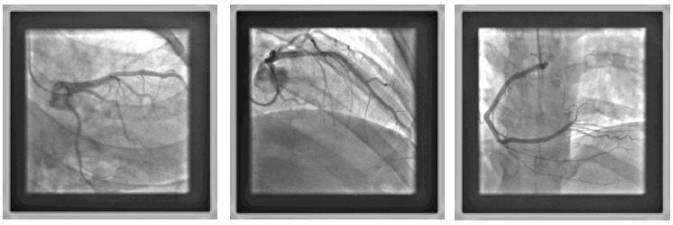

Figure 3: Coronary angiogram (CAG) showed normal epicardial coronary arteries.

Echocardiographic Studies

Dilated RA & RV. The septum is echogenic & jerky in motion. RV free wall is akinetic. Good LV systolic function with LVEF: 60%. Severe RV dysfunction with TAPSE: 09 mm. MPA & its branches were not dilated but there was haziness near the bi-furcation of MPA. These finding were reproduced by repeated echocardiographic scans. His echocardiographic findings are consistent with acute pulmonary embolism. There were features of mild pulmonary artery hypertension suggested by mild tricuspid regurgitation with PASP: 43 mmHg.

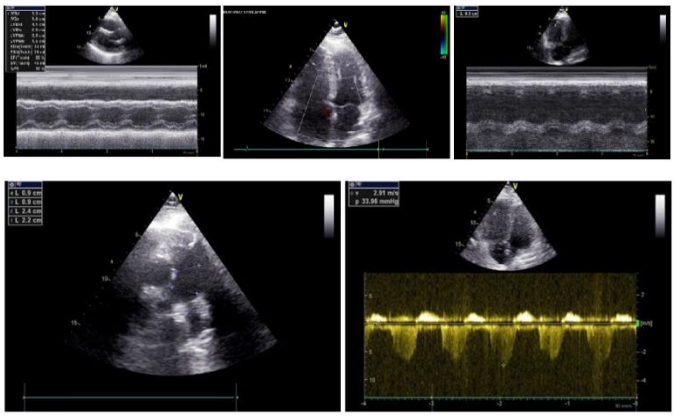

Figure 4: Transthoracic echocardiogram showing the features of acute pulmonary embolism.

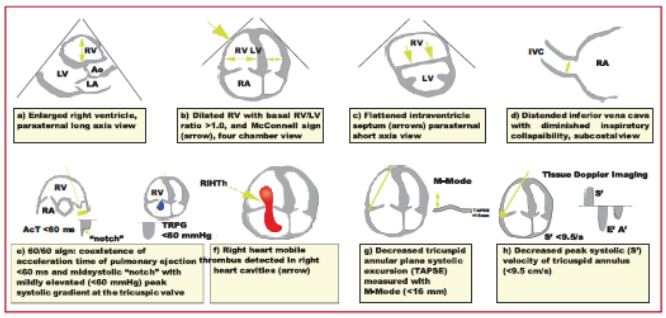

Figure 5: Graphic representation of transthoracic echocardiographic parameters in the assessment of right ventricular pressure overload. A′: peak late diastolic (during atrial contraction) velocity of tricuspid annulus by tissue Doppler imaging; AcT: right ventricular outflow Doppler acceleration time; Ao: aorta; E′: peak early diastolic velocity of tricuspid annulus by tissue Doppler imaging; IVC: inferior vena cava; LA: left atrium; LV: left ventricle; RA: right atrium; RiHTh: right heart thrombus (or thrombi); RV: right ventricle/ventricular; S′: peak systolic velocity of tricuspid annulus by tissue Doppler imaging; TAPSE: tricuspid annular plane systolic excursion; TRPG: tricuspid valve peak systolic gradient[13].

As his echocardiography suggested towards acute pulmonary embolism decision of CT pulmonary angiogram (CTPA) was made. His CTPA revealed large saddle thrombus straddles the pulmonary artery bifurcation extending into the main pulmonary arteries

partially occluding their lumens. Multiple intraluminal hypodense filling defects are also seen along the anterior and posterior segmental & sub-segmental branches of upper & lower pulmonary lobar arteries partially occluding their lumens. The visible aorta and pulmonary veins appear normal.

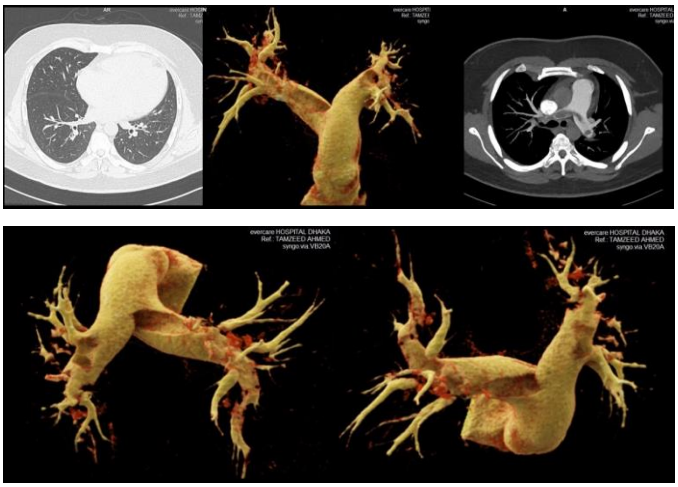

Figure 6: CT pulmonary angiography showing the saddle thrombus.

After CTPA, the family was briefed in details about the condition, treatment modalities & possible outcomes of the patient. He underwent a duplex scan of the deep venous system of both lower limbs, which was found to be a short segment echogenic partially occluding thrombus of about 18.1 x 8.9 mm in right common femoral vein resulting about 40% diameter reduction. Vascular surgeon was involved & was managed with unfractionated heparin infusion (with titration by checking APTT at regular intervals). After initial evaluation, a blood sample was taken to examine the thrombophilia panel. It was found that his levels of protein C- 55 IU/dl (70-146 IU/dl), protein S- 105 IU/dl (60- 130 IU/dl) & antithrombin- III- 75.5 µg/ml (75-125 µg/ml) were like this. It showed that his protein C level was low. Further echocardiography was performed and it revealed improved right heart function. After the recovery of the patient from the acute stage, anticoagulant therapy was given, initially in the form of enoxaparin sodium at a dose of 60 mg twice daily for seven days. In conjunction, Rivaroxaban was given at a daily dose of 30 mg.

Discussion

Pulmonary embolism is a medical emergency. It often ignored, missed rather difficult to diagnose. It differs considerably in size and number, and the underlying causes, including malignancy, trauma, and protein C or S deficiency[1]. Protein-C deficiency by plasma level alone is found in 1 in 200 to 1 in 500 persons in the general population[14,15]. However, many affected individuals remain asymptomatic throughout life. The cardinal clinical manifestation of heterozygous protein C deficiency is venous thromboembolism[16,17]. In our instance the patient presented with acute pulmonary embolism.

Clinical features of acute pulmonary embolism occur abruptly. Dyspnea, tachypnea, chest pain, cough, and hemoptysis may take place. In more severe cases, cyanosis, syncope and circulatory instability occur, and sometimes peripheral edema may be present. In the most severe cases sudden death may ensue [18]. About 25% of PE cases present as sudden death while 15% of all cases of sudden death area attributable to PE[19]. The classic triad of pleuritic chest pain, dyspnea, and hemoptysis is rare, and clinically apparent DVT is present in only 11% of confirmed cases of pulmonary embolism in patients without underlying cardiopulmonary disease[20].

However, the scenario of pulmonary embolism is variable and most patients suffering from acute pulmonary embolism present with one of three different clinical syndromes. These clinical syndromes are pulmonary infarction, acute unexplained dyspnea, and acute cor pulmonale. The pulmonary infarct syndrome usually sustains with a sub-massive embolism that

completely occludes a distal branch of the pulmonary circulation.

Patients with this condition present with pleuritic chest pain, hemoptysis, rales, and abnormal chest X-ray. The acute, unexplained dyspnea pattern may also be the result of sub-massive pulmonary embolism without pulmonary infarction. Chest X-ray and electrocardiogram are usually normal, but oxygen saturation is often depressed. The last one, acute cor-pulmonale syndrome is caused by the complete obstruction of 60 to 75% of pulmonary circulation. Patients with this pattern present with shock, syncope, or sudden death[21,22]. Syncope occurs in approximately 10% of patients with acute pulmonary embolism and is commonly ascribed to a massive, hemodynamically unstable acute pulmonary embolism[23]. Here our patient present with chest pain, dyspnoea and investigation revealed $S_1Q_3T_3$ in ECG, echocardiography revealed dilated RA, RV and saddle thrombus in bifurcation of main pulmonary artery.

The $S_1Q_3T_3$ sign (prominent S wave in lead I, Q wave and inverted T wave in lead III) is a sign of acute cor pulmonale (acute pressure and volume overload of the right ventricle because of pulmonary hypertension) and reflects right ventricular strain[24]. This electrocardiogram (ECG) finding is present in 15% to 25% of patients ultimately diagnosed with pulmonary emboli (PE). Our patient presented with this sign. A transthoracic or transesophageal echocardiogram (TEE) can be used to demonstrate signs of right ventricular pressure overload, and right ventricular hypokinesis and/or dilatation. While the electrocardiogram and the echocardiogram have long been used to assess PE, several computed tomographic criteria recently have been validated in the assessment of the severity of PE. This allows a single test, namely the spiral CT, to establish the diagnosis and assess the severity of PE. In addition, elevated cardiac biomarkers such as cardiac troponin and brain natriuretic peptide (BNP) have well established diagnostic and prognostic roles[25].

Heparin constitutes the cornerstone of management of PE. Thrombolysis can be lifesaving in patients with massive pulmonary embolism, cardiogenic shock, or overt hemodynamic instability. Thrombolytic agents accelerate the lysis of the PE[13].

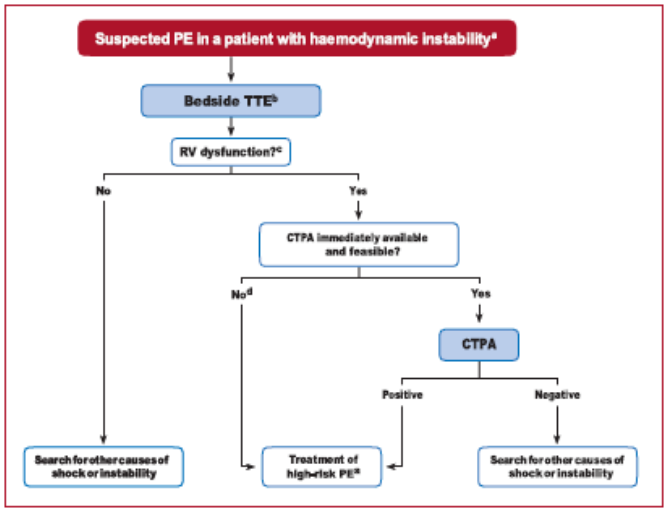

Figure 6: Diagnostic algorithm for patients with suspected high-risk pulmonary embolism presenting with hemodynamic instability[13].

In our case, the patient presented to the emergency department with complaints of chest pain & dyspnea. He was hemodynamically stable at admission and diagnosed initially as a case of non- ST elevated MI & treated accordingly. Acute PE came to the scene when his coronary angiogram came out normal. The patient did not have any features of DVT. In Bangladesh, there was a case report by Samsun Nahar et al.[26] in which the patient also did not have any features of DVT. Consequently, in both cases DVT was identified as the source of PE. In our case, CTPA and duplex study identified thrombus in pulmonary artery and right common femoral veins respectively. Treated with

unfractionated heparin infusion titrated by checking APTT at regular interval followed by low molecular weight heparin & oral NOAC (Rivaroxaban). This case is interesting & publish worthy as it emphasizes to grow suspicion inside the clinicians' mind because these are ignored, missed and thus rarely diagnosed and treated.

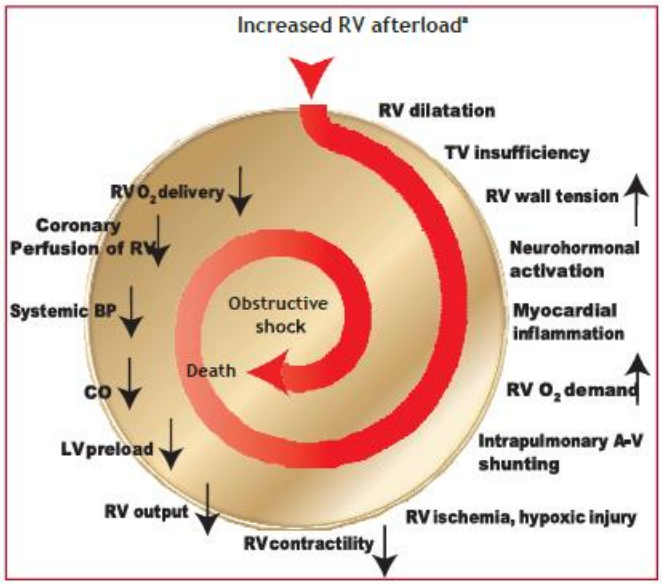

Figure 7: Key factors contributing to hemodynamic collapse & death in acute pulmonary embolism[13].

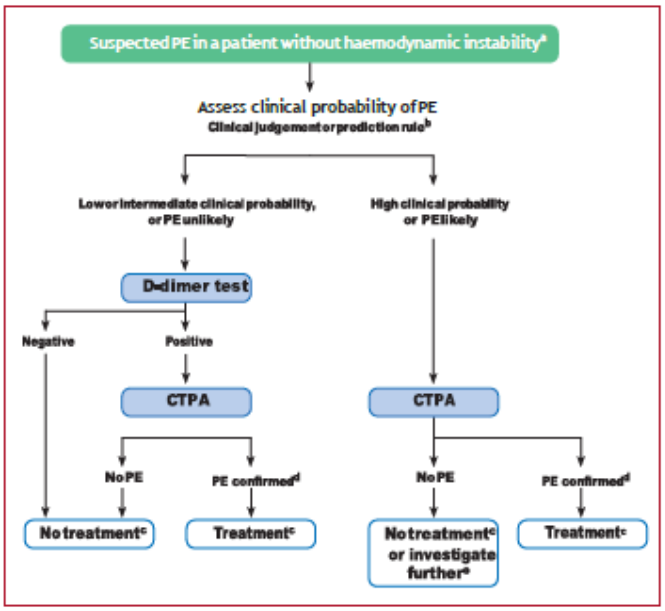

Figure 8: Diagnostic algorithm for patients with suspected high-risk pulmonary embolism presenting with hemodynamic stability[13].

Conclusion

In summary, acute pulmonary embolism is a medical emergency. It is ignored & often missed rather than diagnosed & treated. The treatment strategy should be individualized according to the patient's clinical status and hemodynamics. We inferred & emphasized on development of suspicion among the clinicians for the diagnosis & pave the treatment strategy for this clinical entity.

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