Case Report

# Utilization of POCUS in acute pulmonary embolism with hemodynamic instability: A case presentation

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## ABSTRACT

**Introduction:** A pulmonary embolism (PE) is a life-threatening condition requiring rapid identification and treatment. However, the non-specific symptoms associated with the acute onset of a PE make clinical diagnosis difficult. Point of care ultrasound (POCUS) is a readily available and evolving technique that allows for rapid identification of a PE.
**Case Presentation:** 57-year-old patient presented to the Emergency Department (ED) hemodynamically unstable following an acute onset of shortness of breath and syncope. Ultrasonography revealed right ventricle (RV) dysfunction, abnormal tricuspid annular plane systolic excursion (TAPSE), and intravascular thrombosis, indicating a PE. Subsequently, the computed tomography pulmonary angiogram (CTPA) showed large bilateral pulmonary emboli and the patient received tissue plasminogen activator (TPA).
**Discussion:** The dependence of EDs on CTPA to rule in a PE before initiating thrombolytics may delay life-saving treatment. This case demonstrates the valuable addition of POCUS to the diagnostic protocol for PEs and reduces the waiting period in hemodynamically unstable patients to initiate empiric reperfusion therapy.
**Conclusion:** This case report demonstrates the benefits of POCUS in clinical decision making and highlights the advantages of its utility in the ED.

## INTRODUCTION

A PE is an emergent condition associated with high rates of morbidity and mortality. Severe right ventricle outflow tract (RVOT) obstruction can develop which leads to obstructive shock thus, time to initiation of anticoagulant can be critical.[1,2] The diagnosis of a PE can be difficult as the signs and symptoms are often non-specific. Such symptoms as dyspnea, chest pain and syncope can also be attributed to a number of conditions including acute coronary syndrome, pneumothorax, or cardiac tamponade.[3] Previously the diagnosis of a PE required a CTPA however, prolonged wait times, institutional access to CT, and inability to obtain imaging at various times throughout the day highlighted the need for alternative imaging modalities.[3] Additionally, relative or absolute contraindications to CTs including contrast allergy, chronic kidney disease, and pregnancy also supported the need for alternative imaging methods.[3] According to the 2019 European Society of Cardiology (ESC) Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism, it is now recommended that for those patients with suspected PE who are hemodynamically unstable, the diagnosis should be made with echocardiography to prevent delay in the initiation of thrombolytics.[3] POCUS is a readily available, rapidly evolving technique whose usage has many advantages in the emergency department including being readily available and non-invasive while reducing patient exposure to radiation. POCUS allows for rapid assessment and multi-organ visualization to aid in clinical decision making in hemodynamically unstable patients to rule in/out other potential conditions, as rapid treatment decreases mortality and incorrect administration of TPA can be harmful as well.
This case demonstrates the usefulness of POCUS during assessment of a hemodynamically unstable patient presenting with non-specific symptoms and illustrates the various findings on multi-system ultrasonography that are associated with a PE.

## CASE PRESENTATION

A 57-year-old-patient presented to our ED via Emergency Medical Services (EMS) following a syncopal episode. EMS reported the patient was walking up the staircase at their home when they experienced a brief syncopal episode, not resulting in any head injury or trauma, preceded by acute onset of shortness of breath. The patient was reported to be very agitated when EMS arrived at the home and the patient indicated that they were experiencing some chest pain. On arrival, at the ED, a rapid assessment was performed. Their airway was patent with increased work of breathing at a respiratory rate of 50 breaths per minute. Their O2 saturation was 89% on a non-rebreather at 15L O2. Their color appeared normal. Their blood pressure was 80/64 mmHg and heart rate 126 bpm. They were somewhat confused and vague in providing a history. Their temperature was recorded at 33.7°C. When fully exposed it became apparent that they were wearing an Air-Boot on their left leg. Immediate investigation and management measures were instituted including, blood draw for cardiac and potentially septic protocols. Intravenous (IV) crystalloid bolus was given with no response therefore vasopressor support was begun with norepinephrine.

Review of the patient's electronic health record revealed no allergies, no medications, no chronic medical conditions. Past medical history included an admission five weeks prior for open reduction and internal fixation (ORIF) of a distal left tibial fracture. On physical examination, auscultation of the lungs revealed decreased breath sounds bilaterally with no adventitious sounds appreciated. The heart sounds appeared to be normal without any murmurs noted. The abdomen was soft, non-tender. Subsequent reassessments revealed that the patient's blood pressure dropped to 68/50 mmHg. Additionally, a Bair Hugger was placed on the patient to improve core body temperature.

An electrocardiogram (EKG) was completed during the patient's admission five weeks prior which was normal at that time. The EKG obtained on arrival showed sinus tachycardia and a new Right Bundle Branch Block (Figure 1).

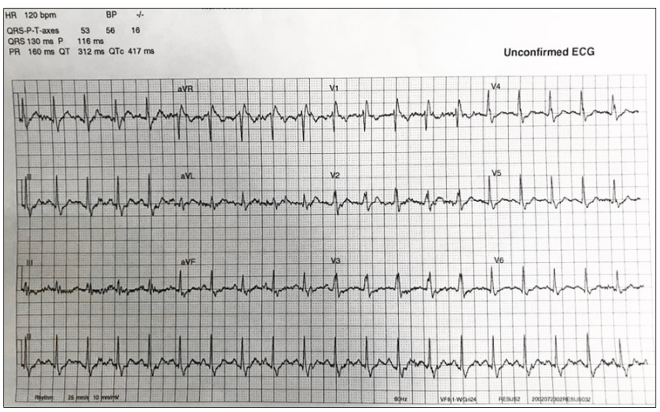


***Figure 1.*** *EKG showing a new right bundle branch block demonstrated by RR' in V1 and V2.*

Our differential diagnosis was somewhat broad at this time owing to the patient's vague history, non-specific symptoms, and hemodynamic instability. The differential included various cardiac and lung pathologies including acute coronary syndrome, pulmonary embolism, cardiac tamponade, pneumothorax, pneumonia, and septic shock.

The laboratory results were significant for leukocytosis (Table 1). Additionally, the patient was determined to have a metabolic acidosis with an anion gap of 24.7 which was not compensated. Thus, an additional respiratory alkalosis was present. His chest x-ray was compared to a previous one, completed five weeks prior which was normal at that time. There were no infiltrates, pneumothorax, or effusions seen on the chest x-ray however, the superior vena cava (SVC) was newly enlarged (Figure 2). The tachycardia, and hypotension, in the presence of leukocytosis was concerning for sepsis thus, the patient was given IV Piperacillin/ Tazobactam 3.375g. Furthermore, the new EKG changes and reduced $O_2$ saturation in the absence of ST elevation and significantly elevated troponin placed PE at the top of our differential diagnosis.

***Table 1.*** *Laboratory results and associated reference ranges.*

| | Results | Reference Range |
|---|---|---|
| ***Complete Blood Count*** | | |
| Leukocytes | 12.2 ↑ | 4.5 – 11.0 x10^9/L |
| Hemoglobin | 127 ↓ | 140 – 180 g/L |
| Platelets | 234 | 150 – 400 x10^9/L |
| ***Chemistry*** | | |
| Sodium | 143 | 135 – 145 mmol/L |
| Potassium | 3.9 | 3.5 – 5.0 mmol/L |
| Chloride | 105 | 95 – 110 mmol/L |
| Creatinine | 153 ↑ | 54 – 113 µmol/L |
| Urea | 7.0 | 3.0 – 7.0 mmol/L |
| Glucose | 15.7 ↑ | 3.5 – 7.8 mmol/L |
| Estimated Glomerular Filtration Rate (EGFR) | 41 | |
| Lactic Acid | 12.71 ↑ | 0.5 – 2.2 mmol/L |
| Troponin | 6.3 | 0 – 34.2 ng/L |
| Creatine Kinase | 41 | 30 – 200 U/L |
| ***Coagulation*** | | |
| International Normalized Ratio (INR) | 1.21 | 0.8 – 1.2 |
| Activated Partial Thromboplastin Time (APTT) | 35.1 | 30 – 45 seconds |
| D - Dimer | 13978 ↑ | 0 – 230 ng/ml |
| ***Venous Blood Gas*** | | |
| pH | 7.105 ↓ | 7.320 – 7.430 |
| PCO2 | 57.2 ↑ | 38.0 – 50.0 mmHg |
| O2 Saturation | 17.9% ↓ | 60 – 85% |
| HCO3 | 17.2 ↓ | 22 – 29 mmol/L |
| Base Excess | 10.8 | mmol/L |
| ***Arterial Blood Gas*** | | |
| pH | 7.342 ↓ | 7.350 – 7.450 |
| PCO2 | 23.7 ↓ | 35 – 45 mmHg |
| PO2 | 119 | 83 – 108 mmHg |
| HCO3 | 13.3 ↓ | 21 – 28 mmol/L |
| Base excess | 13.2 ↑ | -2.0 – +2.0 mmol/L |
| O2 Saturation | 99.1% | 95 – 99% |

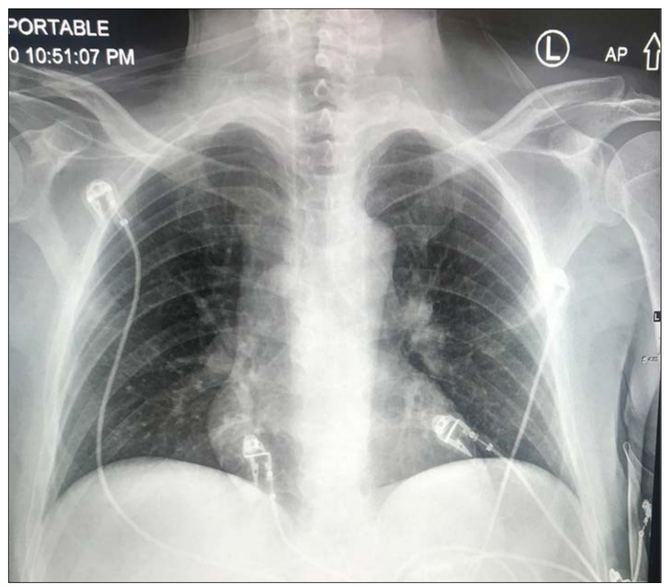


***Figure 2.*** *Anterior-posterior chest radiography exhibits an expanded SVC.*

Subsequently, the pre-test probability of a PE to determine the need for diagnostic work-up was calculated using the Wells' Criteria for PE (Figure 3). The patient received six points, placing them in the moderate risk range according to the Three Tier Model therefore we decided to order both a D-dimer and CTPA (Figure 3).[4]

| Criteria | Points |
|---|---|
| • Clinical signs/symptoms of DVT | 3 |
| • PE is #1 diagnosis OR equally likely | 3 |
| • Heart Rate >100 | 1.5 |
| • Immobilization ≥3 days OR Surgery in the previous 4 weeks | 1.5 |
| • Previous diagnosed PE or DVT | 1.5 |
| • Hemoptysis | 1 |
| • Malignancy with treatment within 6 months or palliative | 1 |

**Management**

- <2 – Low Risk: D-dimer
- 2-6 – Moderate Risk: D-dimer or CTA
- >6 – High Risk: CTA

***Figure 3.*** *Wells' Criteria for PE used to calculate the patients pre-test probability of a PE.*[4]

While awaiting the CTPA, a POCUS assessment was completed. Evaluation of the cardiovascular system revealed RV failure as RV wall hypokinesia was visualized with interventricular septal flattening. Additionally, abnormal TAPSE was visualized as the displacement of the tricuspid annulus towards the apex during systole had a value less than 16 mm, signifying abnormal systolic function. McConnell's sign was also present, described as diffuse RV free wall dysfunction with apical sparing signifying acute RV dysfunction (Figure 4).[2] The RV free wall thickness was less than 5 mm signifying an acute rather than chronic process. The tricuspid valve and RV function are best seen on the apical four chamber view (Figure 4).[2] The FAST scan was negative for free fluid in the abdomen, no pericardial effusion or abdominal aortic aneurysm were present. However, an expanded, non-compressible inferior vena cava (IVC) was seen indicating increased central venous pressure (Figure 5). Additionally, compression ultrasound examination of the left leg showed a non-compressible femoral vein with visible clot indicating a deep vein thrombosis (DVT) (Figure 6). Without the CTPA we were confident that the patient had a large PE as a result of the DVT in his left leg that formed following recent immobilization post-surgery. Thus, Internal Medicine was consulted at this time. Subsequently, the D-dimer was elevated at 13978 ng/mL and CTPA revealed large bilateral pulmonary emboli with poor perfusion of the lungs (Figure 7).

After consultation with the Internal Medicine team, Hematology, and the ICU, it was decided to initiate TPA at 50% TPA over 2 hours because of risk of further PE development with a known DVT. After an in-depth decision of the benefits and risks of TPA with both the patient and their family, the patient received the TPA in the ED and was transferred to the ICU shortly after.

His ICU course required dual vasopressor support initially but was discontinued within a day with the patient being discharged from the ICU within 24 hours.

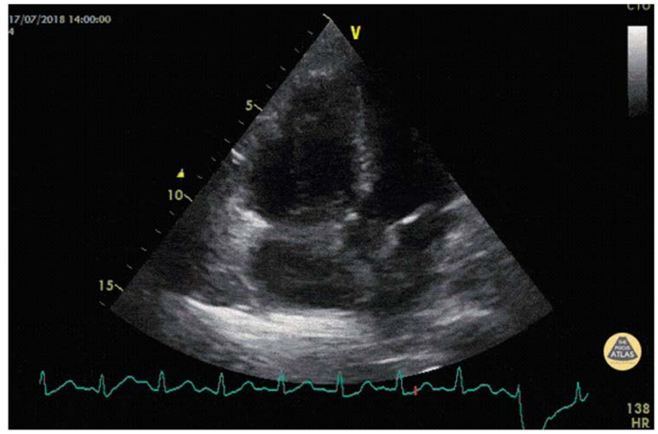


***Figure 4.*** *Transthoracic Echocardiography (POCUS) Apical 4 chamber view. Dilated RV with interventricular septum flattening and McConnell's Sign demonstrated by RV wall hypokinesis with preserved apical contractility.*[5]

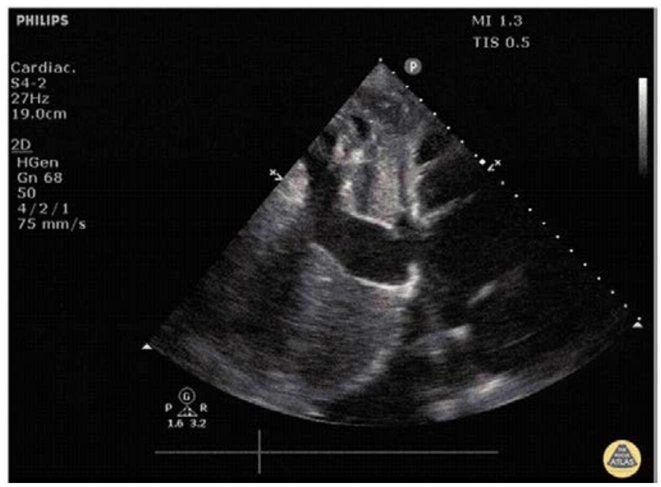


***Figure 5.*** *Sagittal view of dilated IVC.*[6]

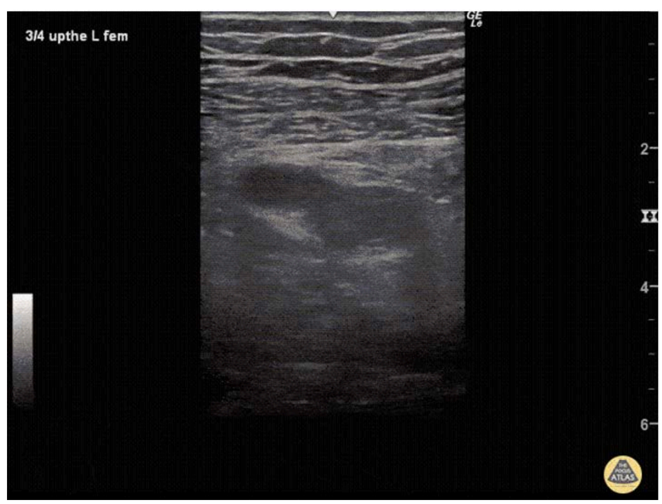


***Figure 6.*** *Transverse view of left femoral artery and femoral vein with thrombus present resulting in incomplete compressibility of the vein.*[7]

***Figure 7.*** *Transverse contrast-enhanced CT image demonstrates decreased contrast flow through right and left pulmonary arteries. Four emboli present with decreased perfusion of the lungs bilaterally.*

## DISCUSSION

Our case demonstrates the use of POCUS in a hemodynamically unstable patient who presented to the ED following an acute onset of shortness of breath and syncope. POCUS revealed signs of RV dysfunction and an intravascular thrombosis, indicating a PE as the likely cause and subsequently, the patient received the appropriate reperfusion therapy. The patient's acute dyspnea and syncope were attributed to large bilateral PEs likely originating from the left femoral DVT precipitated by recent immobilization from a tibial ORIF surgery. The PE was complicated by unstable hemodynamics as a result of obstructive shock. Obstructive shock occurs when the RVOT is obstructed and pulmonary vasculature resistance is increased. The RV experiences volume and pressure overload resulting in dysfunction which reduces left ventricle (LV) filling (preload) and thus, decreases LV outflow resulting in circulatory failure.[1] Septal bowing into the LV further reduces LV filling volume and thus output. This is the presumed mechanism of RV failure as the primary cause of mortality in severe PE as a consequence of insufficient mean arterial pressure (MAP) reducing right coronary artery (RCA) perfusion.[3,8]

Initial laboratory investigations were non-specific. We attributed the metabolic acidosis to hyperlactatemia as elevated levels are seen in poor tissue perfusion during shock. In addition to metabolic acidosis, a mixed disorder with respiratory alkalosis appeared, likely a result of hyperventilation as the patient presented with a respiratory rate of 50 breaths per minute. The leukocytosis was initially treated as sepsis however, in the setting of a severe acute PE, it was likely a stress response, as was the elevated glucose level.

A PE is a clinical emergency requiring rapid identification. However, the associated non-specific clinical symptoms and need to rule in/out a PE before receiving reperfusion therapy may delay the administration of life saving treatment. Additionally, dependence on CTPA may prolong time to diagnosis, therefore, POCUS examination should be used to decrease the need for CTPA in hemodynamically unstable patients with suspected PE. The signs of RV dysfunction displayed on cardiac POCUS have a high predictive value for PE. These include RV enlargement and dysfunction as shown by the McConnell's sign and decreased TAPSE. In addition to the RV free wall hypokinesis with normal apical motion ("LV apical pull"), paradoxical interventricular septum motion may also be observed. Furthermore, dilation of the IVC occurs in the presence of restricted venous return to the right heart.[1,2,3] Tricuspid regurgitation and raised RV pressures are other ultrasonic characteristics of PE associated with acute RV dysfunction as well. In addition to the cardiac and venous ultrasonographic evidence previously discussed, lung POCUS can demonstrate subpleural infarcts as well.[9] Observational studies have found that POCUS provided an empiric diagnosis rapidly compared to longer times to diagnosis and thus, treatment with standard imaging modalities.[2,10,11]

The RUSH exam was developed to reduce mortality in shock by utilizing POCUS for rapid, non-invasive hemodynamic assessment in undifferentiated shock providing a more accurate diagnosis and initiation of appropriate treatment earlier. The RUSH exam is divided into three main parts. The first is the pump which assesses the cardiac status through echocardiography. The second is termed the tank which includes assessment of the IVC, lungs, and abdominal cavities including the right upper quadrant (RUQ), left upper quadrant (LUQ), and pelvis. The third part is titled the pipes which looks at a number of the major arteries and veins in the body. The exam is not always performed in this order as the presence of RV dilation with septal flattening during step one often signifies an increase in pulmonary artery pressure likely due to a PE, as described above, thus the RUSH exam supports moving directly to DVT assessment typically performed in part 3.[11]

The identification of new RV dilation with dysfunction on ultrasonography can provide a rapid presumptive diagnosis for the initiation of empiric thrombolytic therapy, supported by a study completed by Dresden, S. et al.[2] Additionally, Zhu, R. and Ma, X., reported a multiorgan ultrasound examination exhibiting PE-related signs can increase the diagnostic sensitivity up to 90%.[1] Subsequently, the absence of these signs, increase the negative predictive value to rule out PE to 95%.[1] Additionally, previous studies are discussed which demonstrated combining the pre-test probability Wells' score with multi-organ ultrasonography and a d-dimer, greatly reduces the need for CTPA in the diagnosis of a PE.[1]

The presences of a proximal DVT on compression ultrasonography, in addition to signs of RV dysfunction on echocardiography, is considered sufficient to warrant reperfusion therapy, discussed in the 2019 ESC Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism as the majority of PE are derived from a lower limb DVT.[3]

## CONCLUSION

As demonstrated in this case, POCUS is a valuable addition to the diagnostic protocol for PE and current practice guidelines for the initiation of empiric thrombolytics. Identification of interventricular septal flattening, decreased TAPSE, and the McConnell's sign are signs of RV dysfunction displayed on cardiac POCUS that have a high predictive value for PE aiding in the rapid diagnoses in hemodynamically unstable patients and expediting life saving treatments. This new and rapidly evolving technique retains considerable utility for rapid clinical decision making in EDs.

## CONFLICT OF INTEREST

The authors listed above certify that there are no financial or personal conflicts of interest affiliated with the subject matter discussed in this manuscript that could bias their work.


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## DISCLOSURE

Some images in the article have been taken from The POCUS Atlas and have been referenced appropriately below. A copy of their license can be found at: https://creativecommons.org/licenses/by-nc/4.0/.

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