

Point of care ultrasound in cardiac arrest: A case report of PE in PEA arrest

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Abstract

Bedside ultrasound has become an integral part of cardiac arrest resuscitation, enabling the provider to identify potentially reversible conditions in real time. In the setting of pulmonary embolism, bedside ultrasound can be used to diagnose right ventricular dilation thus indicating possible venous thromboembolism. We present a case of a patient with known metastatic cancer who presented with pulseless electrical activity. Bedside transthoracic echocardiography was used to diagnose right ventricular dilation and presumed pulmonary embolism (PE). Given this finding the patient was administered tPA, resulting in return of spontaneous circulation and eventual hospital discharge neurologically intact.

Keywords: point of care ultrasound; transthoracic echocardiogram; PEA arrest; pulmonary embolism; thrombolytics

Introduction

Pulmonary embolism (PE) is a cause of cardiac arrest that carries a high mortality rate [1, 2]. In one study, PE was found to be a major missed diagnosis in 84% of autopsies and in another, PE along with ischemic bowel were the major missed diagnosis in 89% of cases [3-6]. Given the high mortality rate associated with this diagnosis, it is imperative that there is a quick, efficient diagnostic tool that can be utilized during cardiopulmonary resuscitation (CPR) efforts. Bedside ultrasonography (US) has become a routine part of the practice of emergency medicine. This tool can be utilized to diagnosis pulmonary embolism as a cause of cardiac arrest leading to the ability to effectively treat this fatal disease [7-10]. Treatment of PE in cardiac arrest can consist of thrombolysis, anticoagulation, or thrombectomy. Thrombolysis has been successful in the setting of cardiac arrest, but there has not yet been a randomized controlled trial to determine which treatment has the most significant effect on morbidity and mortality [11-20].

Case report

A 66-year-old male with recently diagnosed stage IV gastric cancer and diabetes was transported to the emergency department with the complaint of acute shortness of breath. EMS found the patient in respiratory distress. His initial rhythm was sinus. He then went into pulseless electrical activity (PEA) arrest in route to the ED. He had return of spontaneous circulation (ROSC) after one round of advanced cardiovascular life support (ACLS). Upon arrival to the emergency department, he was again in full arrest and ROSC achieved shortly after. Bedside

transthoracic echocardiogram was performed by the Emergency Medicine resident and attending, revealing a dilated right ventricle with a large thrombus visualized within the right ventricle. Figure 1 shows the parasternal long cardiac ultrasound view. The decision at that time was made to give the patient tPA. He was transported to the ICU and was noted to be alert and following commands the following morning. He was ultimately discharged within a week of arrival neurologically intact.

Massive PE and acute myocardial infarction account for almost two thirds of all out of hospital cardiac arrests of no apparent cause [21, 22]. In those with cardiac arrest, PEA can be found in as many as 63% of cases of PE [7, 23]. During massive PE, there is a dramatically diminished return of blood to the left ventricle resulting in marked reduction of cardiac output. However, the electrical conduction of the heart is not immediately reduced, resulting in PEA as the presenting rhythm [24].

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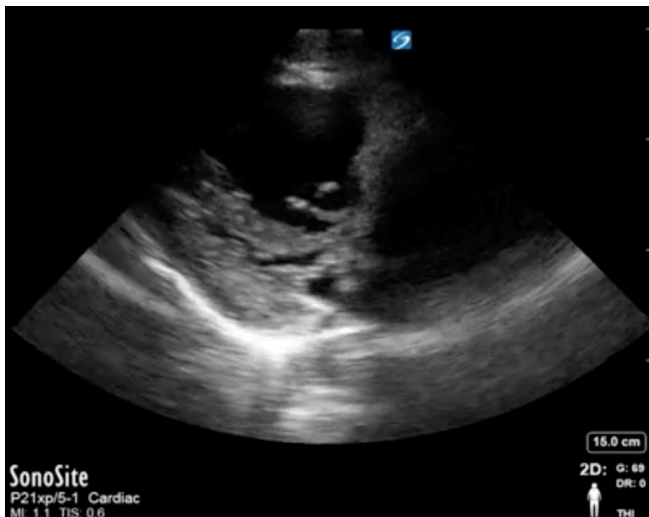


Figure 1 Parasternal long cardiac ultrasound view. This PSL cardiac view shows a dilated right ventricle concerning for right heart strain. The left ventricle is collapsed.

The primary cause of death in acute PE is right ventricular (RV) failure. When 25-30% of the pulmonary vascular bed becomes occluded by clot, pulmonary arterial pressures begin to rise in an effort to compensate for the decreased blood flow [25]. This decrease in blood flow leads to further increased pulmonary arterial pressure because of hypoxia induced vasoconstriction in the pulmonary circulation. This increased pressure results in RV dilation and increased RV wall tension. However, the right ventricle is a thin-walled structure and is not conditioned normally to compensate for pressures over 40 mmHg [26]. The increased right ventricular wall tension results in prolonged RV contraction time that can lead to leftward bowing of the interventricular septum as well as a right bundle branch block, which is often seen on echocardiogram and electrocardiogram, respectively, in acute PE [12]. The prolonged RV contraction time ultimately leads to decreased left ventricular (LV) filling and thus decreased cardiac output [27].

The diagnostic test of choice for diagnosing PE is multi-detector computer tomographic pulmonary angiography (MDCT-PA) [28-31]. MDCT-PA improved imaging techniques have increased the ability to diagnosis segmental and sub-segmental emboli and has led to a better estimation of the overall thrombus burden. These improvements have led to improved risk stratification for patients, thus allowing better to determine the most appropriate treatment options [32]. However, in the hemodynamically unstable patient, this is not an option. In our patient, knowing that he had metastatic gastric cancer heightened our suspicion of thromboembolic disease, but without ultrasound we would not have had any way of making a concrete diagnosis given his hemodynamic instability. Echocardiography, particularly in the unstable patient, can be utilized to diagnose PE [33-35]. Ultrasonographic findings suggestive of PE include: RV wall hypokinesis, RV dilation, RV apical wall sparing, right atrial enlargement, increased RV/LV ratio, pulmonary arterial hypertension, tricuspid regurgitation, underfilled hyperdynamic LV, and right heart thrombus [12, 33, 35-39]. In our patient, we began our bedside echocardiography by utilizing the parasternal long view. We visualized a dilated right

ventricle. The finding of a dilated right ventricle alone on transthoracic echocardiography (TTE) is fairly specific for PE (87%-98%) though not very sensitive for PE (31-72%) [40-43]. RV hypokinesis can be found in up to 92% of individuals with acute, large PE [44, 45]. McConnell's sign (regional wall motion abnormalities sparing the right ventricular apex), if present, have a 94% specificity for a PE [38]. Although, this was further questioned in a study looking at findings of PE, as well as RV infarction. They found that up to two thirds of individuals with RV infarction may have apical sparing [46]. On further scanning, we were able to visualize a mobile thrombus in the right ventricle.

Right heart thrombus (RHT) is not commonly seen in PE and can be found in 4-23% of cases [8, 17, 47-51]. The mortality rate in these instances is very high, up to 100% without treatment and 45% with treatment [20, 47, 51]. While there are many causes of RHT, the vast majority (up to 90%) are caused by deep venous thrombus [52]. The finding of right heart thrombus is diagnostic of PE and justifies reperfusion treatment [11, 20, 50, 53-55]. We were able to visualize a thrombus in the right ventricle during our bedside echocardiogram, thus allowing us to appropriately target our therapy towards the now definitely diagnosed cause of PEA cardiac arrest.

Therapy choices for RHT published in case reports have included anticoagulation with heparin, systemic thrombolysis, surgical embolectomy, and percutaneous embolectomy [18, 56-58]. These therapies have shown varied rates of success and mortality and there is no current consensus amongst physicians as to which therapy is best. In massive PE, thrombolysis is often the accepted method of treatment [59-62]. In a retrospective case series that evaluated 177 cases of right heart thromboembolism, the mortality post thrombolysis was significantly lower than with surgery or anticoagulation alone (11.3% vs 23.8% and 28.6% respectively) [20]. In another study, individuals with right heart thrombus treated with heparin alone had a worse prognosis [54]. The most feared complication of thrombolysis is bleeding. In one study, the overall risk of hemorrhage with the use of thrombolytics as reported as 6-20% [63]. Thrombolysis was risky in our patient with metastatic cancer, but he was also a poor surgical candidate and we decided this therapy was worth the risk given the potential benefits. The fact that our patient was able to walk out of the hospital neurologically intact, supports the evidence that thrombolysis can be a successful therapy. Our case further supports the need for additional studies, particularly randomized control trials, which would provide guidance in future treatment of RHT.

Discussion

We present a case of metastatic cancer causing PE that lead to PEA arrest. Bedside transthoracic echocardiography was employed in the work-up of this patient during CPR and right heart thrombus was visualized in real time. Given the clinical instability of the patient, the decision was made to treat with thrombolysis. The use of bedside ultrasonography is instrumental in diagnosing critical medical problems and in this case it helped to provide a favorable patient outcome. The use of thrombolytics also contributed to the rapid improvement in the overall clinical

course. Our case report is in agreement with previous case reports of patients that have survived after both the diagnosis by ultrasound of right heart thrombus and treatment with thrombolysis [50, 64-67]. This case further supports the needs for a randomized controlled trial to better determine the best treatment option for right heart thrombus during cardiac arrest [14].

Conclusion

Use of bedside echocardiography in the emergency department allowed for the timely diagnosis of massive pulmonary embolism in this patient. Routine use of thrombolytics is not a part of ACLS protocol. Without the aid of this advanced technology, this patient would not have received the treatment he needed to survive his cardiac arrest as he was too unstable for MDCT-PA. This case is evidence of the importance of use of bedside ultrasound in the diagnosis of pulmonary embolism in unstable emergency medicine patients.

Conflicts of interest

Authors declare no conflicts of interest.

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