REPORTS

Ultrasound Guidance of Thrombolytic Therapy in Pulseless Electrical Activity: A Case Report

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ABSTRACT

A young man presented to the emergency room in extremis and deteriorated into a state of pulseless electrical activity. Bedside echocardiography by emergency medicine physicians was crucial to the clinical decision to implement thrombolytic therapy for suspected massive pulmonary embolus.

INTRODUCTION

Pulmonary embolism (PE), a leading cause of death in the United States resulting in 200,000 deaths per year, can rapidly progress to fatality.[1,2] Ten percent of patients with PE will die within the first hour of the event.[1]

The clinical presentation of PE ranges from nonspecific symptoms to severe shock and even death, primarily secondary to right ventricular failure.[2,3] Myocardial infarction (MI), arrhythmias, and heart block secondary to PE have also been proposed as causes of death in massive PE.[2]

Historically, treatment of PE has consisted of anticoagulation with unfractionated intravenous heparin. Heparin prevents further clot progression by interfering with the intrinsic coagulation pathway, but does not actively remove the thrombus. It is important to stress that heparin, while decreasing long term morbidity and mortality from recurrent emboli, does not improve pulmonary perfusion or right ventricular hemodynamics in the acute setting.[3-7]

Two advances have led to significant improvement in the classification and treatment of pulmonary embolism. Echocardiographic assessment of the right ventricle is utilized to risk stratify patients, while thrombolytic therapy produces active blood clot lysis in the pulmonary arteries. We present a case of massive PE in which bedside echocardiography and thrombolytics played a pivotal role in the patient's survival.

CASE

Paramedics responded to the home of a 33-year-old male with difficulty breathing. The brother heard a loud noise in the adjacent room where the patient was "lying on the floor gasping for air." Vital signs were blood pressure (BP) 50/palpation, heart rate (HR) 134, respiratory rate (RR) 28, and oxygen saturation (SaO2), 87 % on room air. His lungs were clear, the heart rhythm was sinus tachycardia, and his skin was cool and moist. The patient was rapidly transported to the hospital, with slightly improved vital signs (BP 72/palp, Sao2 98%) on 15L non-rebreather mask. He repeatedly remarked in short sentences that he was going to pass out.

On physical exam the patient was an alert, diaphoretic and cyanotic young male in severe respiratory distress. He denied medications or illicit drug use. The family stated he had no medical conditions other than a traumatic fracture of his ankle two months prior in a sky diving accident. Vital signs were BP 60/40, pulse

90, RR 30s, temperature 96.4, and SaO2 unable to obtain. He was tachypneic with clear lungs and no wheeze or rales. He was tachycardic, without a murmur or JVD. Radial and pedal pulses were diminished. There was 1+ pedal edema bilaterally with no calf erythema or tenderness.

ED Course: The treating physician suspected a pulmonary embolus and ordered an IV heparin bolus followed by heparin infusion. A stat CXR, EKG, and labs were ordered. The patient was intubated for respiratory failure using etomidate and succinylcholine under RSI protocol. Endotracheal tube placement was visually confirmed; Sa02 was 90% with manual ventilation and equal breath sounds. The patient rapidly deteriorated into pulseless electrical activity (PEA) and Advanced Cardiac Life Support was initiated immediately. A bedside subxiphoid view echocardiogram was performed using the emergency department's ultrasound machine, revealing negligible wall motion and right ventricular dilatation without left ventricular enlargement (Figure 1). The high clinical suspicion for PE in this patient, coupled with echocardiographic evidence of right ventricular (RV) dysfunction prompted the use of thrombolytics. Tissue plasminogen activator was front-end loaded (15 mg bolus, followed by 50 mg over 30 minutes, then 35 mg over 60 minutes) within minutes of the PEA. In less than five minutes, the patient regained peripheral pulses with a systolic blood pressure of 96; forty minutes later his BP was 140/115, HR 140, SaO2 97% on mechanical ventilation.

Hospital course: The patient was admitted to the ICU. Pulmonary angiogram demonstrated moderate sized PE in the left 2nd order branches and in the right upper lobe peripheral branches. Minor nasopharyngeal bleeding complicated the hospital course. The patient was successfully extubated and discharged from the hospital with no neurological deficits.

Discussion

Echocardiography has changed physicians' approach to the classification and diagnosis of PE. A patient with a PE can be placed in one of three categories: "small"—hemodynamically (HD) stable with no evidence of RV dysfunction; "submassive"—HD stable but echocardiographic evidence of RV dysfunction; and "massive"—RV failure, shock, HD unstable.[1,5,6,8] Patients with small PEs generally do well with anticoagulation alone.[6] Patients with submassive PE have an increased risk of mortality compared to patients with normal echocardiograms.[6,7,9,10] The early use of thrombolytics in this group can substantially improve and even reverse RV dysfunction.[4,6,11] Whether this physiologic effect translates into an improvement in morbidity and mortality is unclear.[6,11,12]

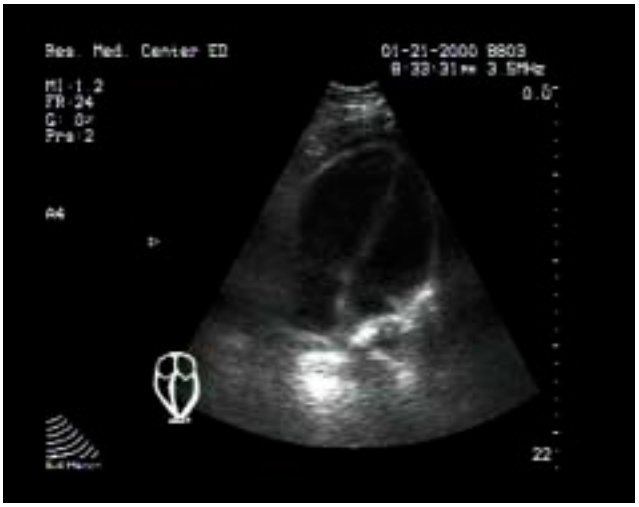

Figure 1. Subcostal four chamber view taken at time of initial cardiac arrest.

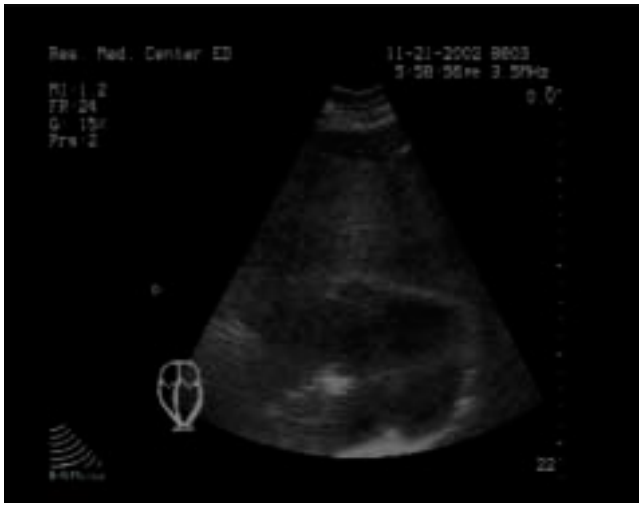

Figure 2. Apical four chamber view taken at approximately two and a half hours after cardiac arrest.

Patients with cardiogenic shock secondary to RV failure ("massive PE") present a particular challenge to the managing physician. Patients with massive PE account for a small percentage of total PEs, but have a high mortality rate (18-58%) that is even higher (65%-95%) if they require CPR.[2,6,13,14] Bedside ultrasound is often a critical imaging modality because these patients are frequently too unstable for definitive "gold standard" testing outside of the emergency department. Although it is not an effective screening tool for all cases of suspected PE, echocardiography is ideally suited for detecting the RV dysfunction secondary to massive PE.[8-10,14-17] While more definitive echocardiographic signs of RV dysfunction include hypokinesis, abnormal septal motion, and tricuspid regurgitation, a simple qualitative measurement of RV dilatation (diameter greater than or equal to LV) can be used as evidence of PE.[8,15,17]

There is a paucity of literature concerning thrombolytic therapy for massive PE, but available evidence supports its use in this setting.[6,7,11] A study comparing thrombolytics to heparin in massive PE was halted after only 8 patients; all 4 patients treated with thrombolytics and anticoagulation survived, while all 4 patients treated with anticoagulation alone died.[18] The American College of Emergency Physicians (ACEP) supports the consideration of thrombolytic therapy in confirmed massive PE. They further advocate the consideration of thrombolytics in suspected massive PE based upon bedside echocardiographic findings of RV dysfunction.[19]

Although the use of thrombolytic therapy in the setting of cardiac arrest from suspected PE was historically contraindicated, there have been no randomized studies to support this prohibition of its use. On the contrary, there have been nearly 100 reported cases of successful cardiac resuscitations in which thrombolytics were used. Bailen notes there is no evidence that thrombolysis used during CPR for PE causes an increase of hemorrhagic complications, and thus advocates thrombolysis during CPR for suspected cases of PE.[2]

Emergency physicians often care for patients who present in cardiopulmonary arrest. While PE accounts for only 2-15% of unexpected sudden deaths, there is limited, but compelling, evidence that thrombolytics can be a life saving intervention.[2] The goal would be to identify those patients whose arrest is most likely secondary to PE and would thus benefit from thrombolytics. History of present illness is essential, but often limited. Physical exam is most often non-specific in PE, and therefore objective ancillary evidence is crucial in this situation. When patients with PE present in cardiac arrest, the rhythm is usually PEA or asystole.[2] In one series, twenty-five of thirty-six consecutive patients with unexplained cardiac arrest presented in PEA.[13] Transesophageal echocardiograms were performed on all patients in PEA, fourteen of whom showed evidence (RV enlargement with no LV enlargement) consistent with PE. Nine of these fourteen patients with PEA and echocardiographic evidence of right ventricular strain had a PE, while none of the non-PEA patients had PE. Although further study is needed, it seems reasonable to conclude that, in cases of unexplained PEA and a high clinical suspicion of PE, bedside echocardiographic screening for RV dilation could be used to support the consideration of potentially life-saving thrombolysis.

The risk of serious bleeding complications, especially intracranial hemorrhage (ICH), accompanies the use of thrombolytics. Data compiled from five studies showed an ICH rate of 1.9% (6/312) in patients given thrombolytics for PE. Of note, no patient under the age of 50 suffered an ICH. When combined with 455 additional patients from other case series, the frequency of ICH for PE thrombolysis patients dropped to 1.2%.[20] When considering thrombolysis, clinicians should seek out any known absolute contraindications, but should also weigh the relatively low rate of ICH against the extremely high mortality from massive PE.

We report on the successful use of TPA in a case of massive PE that swiftly deteriorated to PEA. Bedside echocardiography reinforced the clinical suspicion of PE and was the determinant factor in the decision to use thrombolytics. Aggressive treatment with thrombolytic therapy was very likely the key intervention that facilitated a successful resuscitation with normal neurologic outcome. Fear of adverse outcomes from bleeding and lack of a definitive

diagnosis are likely the factors that dissuade physicians from using thrombolytics in suspected massive PE. Rates of ICH, however, are relatively low compared to the mortality rates of massive PE. Bedside echocardiography can be a powerful diagnostic tool in cases of HD instability or cardiac arrest from suspected massive PE and could potentially trigger life-saving intervention.

REFERENCES

1. Haughey M. Pulmonary embolism: recent advances in diagnosis and treatment modalities. Part I—epidemiology, risk factors, and pathophysiology. *Emergency Medicine Reports* 2004;25(11):125-136.
2. Bailen MR et al Thrombolysis during cardiopulmonary resuscitation in fulminant pulmonary embolism: a review. *Crit Care Med* 2001;29(11):2211-2219.
3. Kessler CM. Acute pulmonary embolism treated with thrombolytic agents: current status of tPA and future implications for emergency medicine. *Ann Emerg Med* 1988;17:1216-1220.
4. Agnelli G et al. Thrombolysis vs. heparin in the treatment of pulmonary embolism: a clinical outcome-based meta-analysis. *Arch Intern Med* 2002;162:2537-2541.
5. Haughey M. Pulmonary embolism: recent advances in diagnosis and treatment modalities. Part II—differential diagnosis. *Emergency Medicine Reports* 2004;25(12):137-147.
6. Konstantinides S. Should thrombolytic therapy be used in patients with pulmonary embolism? *Am J Cardiovasc Drugs* 2004;4(2):69-74.
7. Loebinger MR and JC Bradley. Thrombolysis in pulmonary embolism: are we under-using it? *QJ Med* 2004;97(6):361-364.
8. Goldhaber SZ. Echocardiography in the management of pulmonary embolism. *Ann Intern Med* 2002;136:691-700.
9. Kasper W et al. Prognostic significance of right ventricular afterload stress detected by echocardiography in patients with clinically suspected pulmonary embolism. *Heart* 1997;77:346-349.
10. Jackson RE et al. Prospective evaluation of two-dimensional transthoracic echocardiography in emergency department patients with suspected pulmonary embolism. *Academic Emergency Medicine* 2000;7(9):994-998.
11. Goldhaber SZ. A contemporary approach to thrombolytic therapy for pulmonary embolism. *Vascular Medicine* 2000;5:115-123.
12. Hamel E. Thrombolysis or heparin therapy in massive pulmonary embolism with right ventricular dilation. *Chest* 2001;120:120-125.
13. Comes KA et al. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. *Am J Med* 2000;109:351-356.
14. Le Conte P et al. Efficacy of alteplase thrombolysis for ED treatment of pulmonary embolism with shock. *Am J Emerg Med* 2003;21(5):438-440.
15. Bova C et al. Diagnostic utility of echocardiography in patients with suspected pulmonary embolism. *Am J Emerg Med* 2003;21(3):180-183.
16. Grifoni S et al. Utility of a integrated clinical, echocardiographic, and venous ultrasonographic approach for triage of patients with suspected pulmonary embolism. *Am J Cardiol* 1998;82:1230-1235.
17. Miniati M et al. Value of transthoracic echocardiography in the diagnosis of pulmonary embolism: results of a prospective study in unselected patients *Am J Med* 2001;110:528-535.
18. Jerjes-Sanchez C. Streptokinase and heparin versus heparin alone in massive pulmonary embolism: A randomized controlled trial. *J Throm Thrombolysis* 1995;2(3):227-229.
19. American College of Emergency Physicians. Clinical policy: critical issues in the evaluation and management of adult patients presenting with suspected pulmonary embolism. *Ann Emerg Med* 2003;41:257-270.
20. Kanter DS. Thrombolytic therapy for pulmonary embolism: Frequency of intracranial hemorrhage and associated risk factors. *Chest* 1997;111(5):1241-1245.