DIAGNOSIS OF NEAR-FATAL PULMONARY EMBOLUS-IN-TRANSIT WITH FOCUSED ECHOCARDIOGRAPHY

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Abstract—Background: Among patients who die from pulmonary embolus (PE), approximately two-thirds succumb within an hour of presentation. Computed tomography can provide a definitive diagnosis but is associated with practical limitations. Echocardiography can increase diagnostic certainty of PE by visualizing signs of acute right ventricular (RV) strain. This case highlights a potentially lethal finding associated with PE and the role of clinician-performed bedside echocardiography in the timely management of this disease. Objective: To describe a case of PE-in-transit diagnosed by clinician-performed focused echocardiography. Case Report: A 78-year-old man with lymphoma presented to the Emergency Department with shortness of breath. His blood pressure was 95/53 mm Hg; his oxygen saturation was 84% on room air. A focused echocardiogram showed a highly mobile elongated mass traversing the right atrium and right ventricle, consistent with a PE-in-transit. Anticoagulation was initiated and Cardiovascular Surgery was consulted for emergent thrombectomy. Minutes after reviewing the ultrasound with the surgeons, the patient was transported to the operating room. Just before surgery, the patient had a cardiac arrest. Exploration of his heart failed to reveal thrombus; however, extensive clot burden was removed from the pulmonary arteries, with subsequent return of spontaneous circulation. Conclusion: The clinician performed a focused echocardiogram to evaluate the cause of the patient’s critical state. PE-in-transit, a rare entity associated with large PEs, was identified, which obviated the need for further diagnostic evaluation and led to immediate aggressive therapy. Increased familiarity with the uses of bedside sonography in the evaluation of shock and respiratory distress may allow clinicians to become more proficient in managing these patients. © 2013 Elsevier Inc.

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INTRODUCTION

Patients with pulmonary embolus (PE) who are in shock have a four-fold higher mortality rate than those without hemodynamic instability, and in those patients who die from PE, approximately two-thirds succumb within an hour of presentation (1–5). For this reason, PE causing hemodynamic instability needs to be identified rapidly. Computed tomography (CT) can provide a definitive diagnosis; however, obtaining a CT involves significant delays, removal of the patient from the resuscitation area, and exposure to potentially nephrotoxic intravenous contrast.
Echocardiography has the ability to significantly increase diagnostic certainty by visualizing sonographic signs of acute right ventricular strain (6,7). Board-certified echocardiologists are rarely available on a timely basis for these critically ill patients during nights and weekends. Thus, the American Society of Echocardiography and the American College of Emergency Physicians have endorsed the performance and interpretation of focused cardiac ultrasound at the bedside by clinician sonologists(8). In addition, point-of-care evaluation of the lower extremities may reveal deep vein thrombosis; a finding that strongly supports the diagnosis of PE in the appropriate clinical setting. The following case highlights an uncommon but potentially lethal finding associated with hemodynamically significant PE, and the role of bedside echocardiography in the timely management of this disease.

CASE REPORT

A 78-year-old man with non-Hodgkin’s lymphoma presented to the Emergency Department with several days of right lower-extremity swelling, shortness of breath, and chest pain. His physical examination revealed an ill-appearing man in respiratory distress. His vital signs were: blood pressure 95/53 mm Hg, heart rate 75 beats/min, respiratory rate 20 breaths/min, temperature 36.5°C, and oxygen saturation of 84% on room air. He had unilateral right lower-extremity swelling. Focused bedside ultrasonography of the right lower extremity confirmed the presence of a deep vein thrombosis. A focused cardiac ultrasound showed normal right ventricular size and systolic function, and a highly mobile elongated mass traversing the right atrium and right ventricle consistent with a PE-in-transit (Figure 1, Video 1).

Anticoagulation with intravenous heparin was initiated and Cardiovascular Surgery consultation was obtained for emergent thrombectomy. The ultrasound images were reviewed with the surgical team, and the patient was transported to the operating room <20 min after the ultrasound examination. Moments before initiation of surgery, the patient had a cardiac arrest. Surgical exploration of the heart failed to reveal thrombus, and exploration continued into the pulmonary artery where an extensive clot was discovered and removed (Figure 2). A transesophageal echocardiogram performed in the operative suite after the embolectomy revealed severe right ventricular (RV) systolic dysfunction and a right-to-left ventricular (LV) end-diastolic diameter ratio of >1, but no evidence of intracardiac thrombus (Figure 3). The patient had return of spontaneous circulation and survived to hospital discharge with completely intact neurological function.

DISCUSSION

Many applications of bedside ultrasound in the emergency evaluation of patients with undifferentiated shock and
dyspnea have been described (9–12). Readily identifiable sonographic findings can assist in differentiating hypovolemic, distributive, mechanical, and cardiogenic causes of shock. The absence of these findings serves to raise the suspicion of metabolic or toxic derangements. In the current case, the combination of hypoxia with shock was concerning for a variety of pathologies—including massive pulmonary embolism, cardiogenic shock with pulmonary edema, pneumonia with septic shock, tension pneumothorax, and massive pleural effusion.

The clinician performed a focused ultrasound to help determine if any of the aforementioned conditions were present. However, the presence of unilateral lower-extremity swelling, shortness of breath, and chest pain raised particular concern for pulmonary embolism. Although ultrasound is of limited utility in identifying acute PE that is not causing hemodynamic instability, readily identifiable sonographic findings are usually present with emboli large enough to obstruct the proximal pulmonary arteries, thereby leading to decreased cardiac output and ensuing hemodynamic compromise (7,13,14). The findings in massive PE include RV hypokinesis, RV-to-LV end-diastolic diameter ratio >1, a plethoric inferior vena cava without respiratory variation, McConnell’s sign (i.e., RV mid-free wall hypokinesis/akinesis with apical sparing), and an under-filled left ventricle with interventricular septal flattening or paradoxical motion (7,13–15).

The bedside echocardiogram demonstrated very subtle paradoxical motion of the interventricular septum, but failed to show the other expected findings of massive PE (Video 1). Presumably, this was because the critical mass of PE burden had not yet obstructed the pulmonary vascular bed to cause severe enough acute pulmonary hypertension. PE-in-transit, a related potentially fatal condition, was clearly identified. Anticoagulation was immediately initiated and surgical cardiothoracic intervention was pursued. Consideration was given to pharmacologic thrombolysis, but a surgical approach was ultimately selected given the patient’s unknown status of brain metastases and a readily accessible experienced surgical team.

Although all pulmonary emboli at some point pass through the right side of the heart, a PE-in-transit is diagnosed when a peripherally formed embolus is visualized floating within the right atrium or ventricle. The echocardiographic appearance of PE-in-transit is a highly mobile worm-like mass that is continuously changing shape (16,17). It is important to distinguish this entity from in situ intracardiac thrombi, which do not form in peripheral vasculature, are non-serpiginous, appear less sonographically mobile, and are not as deadly (16). The clinical presentation of PE-in-transit is similar to that of massive PE, including dyspnea, thoracic pain, syncope, hypoxia, and hypotension (17,18).

The prevalence of PE-in-transit among patients with pulmonary embolism ranges from 4–18%, with the higher end of this spectrum seen in cases with greater embolus burden (17,19,20). Alternatively, the concomitant presence of pulmonary embolism occurs in approximately 98% of patients with PE-in-transit (19). Therefore, diagnosis of PE-in-transit virtually confirms the diagnosis of PE. PE-in-transit is a highly lethal condition, with an estimated mortality of 16–45% among patients treated with anticoagulation, thrombolysis, or surgical techniques (16,17,19,21,22). This is in stark contrast to the in-hospital patient mortality of treated pulmonary embolism, which approaches 3% (23). This discrepancy in lethality is not surprising, as most cases of PE-in-transit are coupled with a great PE burden (21). Indeed, it is proposed that the pathophysiologic changes resulting from significant pulmonary arterial circulatory obstruction—namely, pulmonary hypertension, right ventricular failure, and tricuspid regurgitation—generate sufficient resistance to forward flow that the peripheral-origin embolus remains stationed in the right heart (20). Whether the right heart thromboembolism itself is responsible for the high mortality of PE-in-transit, or whether it is merely evidence of a more significant pulmonary vasculature thrombus burden and increased lethality from that, is unclear.

Recommendations for treatment of PE-in-transit are limited by a paucity of large randomized prospective studies; however, insightful information can be gleaned from existing literature. Torbicki et al. reviewed PE-in-transit cases from the International Cooperative Pulmonary Embolism Registry and found a three-fold increase in 14-day mortality among patients treated with anticoagulation who had PE-in-transit, in comparison to clinically similar patients with only pulmonary embolism (19). In a meta-analysis of existing reports of 177 PE-in-transit patients, Rose et al. reported that all patients (n = 16) who received no therapy died (21). Among patients treated with anticoagulation, thrombolysis, or surgical techniques, the mortality was lowest in patients undergoing thrombolysis (11%), followed by surgery (24%) and lastly, anticoagulation (29%) (21). In the European Cooperative Study, among patients belonging to the highest risk group, there was a trend toward lower early mortality in those treated surgically (27%), in comparison to the patients who underwent thrombolysis (40%) or anticoagulation (>60%) (16). Although further investigation regarding the optimal therapy for PE-in-transit is needed, the existing literature should prompt strong consideration of thrombolysis or surgical management rather than anticoagulation alone.

Although the distinction between PE-in-transit and massive PE in this case may not have been clinically significant, the differentiation of these conditions from other items in the differential diagnosis (e.g., pneumonia with
septic shock) was imperative. Early incorporation of focused ultrasound in the evaluation of this patient enabled the physician to rapidly confirm a diagnosis and confidently provide the appropriate—and potentially dangerous—therapies that were needed. It is likely that without the information gleaned from the ultrasound, the physician may have sought recourse to additional diagnostic testing and subjected the patient to the known associated risks (e.g., delay in diagnosis, transport outside resuscitation area).

PE-in-transit is a rare diagnosis. To our knowledge, it has previously been reported only once using point-of-care ultrasound (24). This case differs from the previous report in that the point-of-care ultrasound was the basis of immediate definitive operative treatment without additional imaging studies. Despite this expeditious care, the patient had a cardiac arrest within 20 min of the sonographic diagnosis. Had the patient not already been in the operating room, the patient might not have survived.

CONCLUSION

In this case, the clinician performed a focused echocardiogram to evaluate the cause of the patient’s critical state. PE-in-transit, a rare entity usually associated with large PEs, was identified. Early incorporation of bedside ultrasound enabled the physician to cease further diagnostic evaluation and focus all efforts on therapeutic intervention.

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REFERENCES


SUPPLEMENTARY DATA

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