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CASE REPORT

Atypical Presentation of Massive Pulmonary Embolism, a Case Report

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Abstract

The lack of pathognomonic signs and symptoms makes the diagnosis of pulmonary embolism (PE) difficult. Here, we report a case of a 42-year-old man presented to the emergency department with worsening epigastric pain, hypotension, frank bradycardia, and final diagnosis of PE. Although previous studies have indicated that abdominal pain was observed in 6.7% of patients with PE, the exact reason for abdominal pain in PE still remains unknown. Tension on the sensory nerve endings, hepatic congestion, and distention of Gilson's capsule are some of the possible mechanisms of abdominal pain in PE. We conclude that emergency physicians should pay more attention to PE, which is an important differential diagnosis of shock state. In this context, rapid ultrasound in shock (RUSH) should be considered as a vital sign that needs to be evaluated when recording the history of patients presented to the emergency department with signs and symptoms of shock.

Key words: Pulmonary embolism; ultrasonography; shock; abdominal pain; bradycardia

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Introduction:

The lack of pathognomonic signs and symptoms makes the diagnosis of pulmonary embolism (PE) difficult. A well-timed diagnosis and management of PE are necessary to decrease mortality. It has been reported that, annually, 50,000–200,000 deaths occur as a result of PE, with a mortality rate of 30% without treatment (1). Postmortem studies have revealed that up to 70% of PEs have been misdiagnosed. Moreover, 40% of the dying patients had visited a clinician for ambiguous symptoms in the weeks prior to their death (1, 2). This significant amount of misdiagnosis highlights the importance of reviewing the different possible presentations of this fatal medical condition. Here, we report a case of a patient presented to the emergency department (ED) with worsening epigastric pain, hypotension, frank bradycardia, and final diagnosis of PE.

Case report:

The patient was a 42 year-old man, admitted to the ED with chief complaint of severe abdominal pain begun from the morning. The pain was sensed prominently in epigastric area with acceleration/deceleration pattern, and also crusher/vague quality that increased after eating. The pain was not positional and had neither transition nor shifting. He had no significant past medical history or previous surgery. But should mention that was addicted to methadone and was a heavy smoker (30 pack cigarettes per year) for many years. On admission to ED, he was so agitated with 28/min respiratory rate, 68/min pulse rate, 85/65 mmHg blood pressure, 88% oxygen saturation in room air, and 37°C axillary temperature. Electrocardiogram (ECG) showed frank bradycardia that not matches with other vital signs (Figure1).

The patient was pale, had dry mucosa, presented marked epigastric tenderness on deep palpation, and had neither rebound tenderness nor guarding. The pulses in the extremities were filiform, lung examination was normal, and there were multiple red rushes (red macules) on the chest and extremity skin. Focused assessment with sonography for trauma (FAST) revealed mild, free fluid in Morison's pouch. With the impression of perforated hollow viscous organs, the emergency surgical consultant performed an upright chest X-ray (CXR) examination after primary resuscitation, but noted no specific finding. This examination was repeated after pushing 500 mL of air via nasogastric (NG) tube, but no specific observation could be found. Double contrast abdominal and pelvic spiral computed tomography (CT) scan revealed intraperitoneal infiltration with increased intestinal wall diameter and ascites. The white blood cell count was 16200/mm^3, arterial blood pH was 7.3, PCO_2 was 18.6 mmHg, HCO_3 was 11.2 mEq/L, and BE was −10.7. All other laboratory data, including serum amylase and lipase, urine analysis, hepatic and renal function tests, and coagulation profile were normal. During admission

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to ED, the patient's blood pressure was low and did not get better with fluid therapy. Hence, rapid ultrasound in shock (RUSH) examination was performed. RUSH examination showed weak heart ventricles contractibility, right ventricular (RV) strain, and a clot in the apex of RV; hence, pulmonary CT angiography of the chest was immediately performed (Figure 2). Spiral pulmonary CT angiography showed massive emboli in the distal right pulmonary artery, hypodense clot in the right atrium with increased diameter, and also a hypodense clot in the RV apex. With the diagnosis of massive pulmonary emboli, high dose of heparin was initiated and cardiac surgery intervention was accomplished. The patient was transferred to the intensive care unit (ICU), but unfortunately died.

Discussion:

A well-timed diagnosis and management of PE are necessary with the aim of decreasing mortality. Because of the lack of pathognomonic signs or symptoms, the diagnosis of PE remains vague. The classic symptoms of dyspnea, tachypnea, and decreased oxygen saturation may occur in up to 92% of cases. However, because of numerous atypical presentations, physicians must focus on other clinical features to include or exclude them as possible diagnosis. Although previous studies have demonstrated that abdominal pain was observed in 6.7% of patients with PE (1, 3). The exact reason for abdominal pain in PE still remains unknown. Tension on sensory nerve endings, infarctions within the microvasculature of the mesentery, hepatic congestion, distention of Gilson's capsule, or diaphragmatic pleurisy resulting from pulmonary infarction are some of the possible reasons for abdominal pain in PE, which have neither been proved nor refuted (1, 4). In contrast, syncope and bradyarrhythmia may cause parasympathetic reflex, because simultaneous slowing of the sinus rate with concomitant arteriovenous block is a common presentation of increased vagal tone. Such reflex may be a mechanism for syncope in patients with PE (5). In addition, bradycardia is also a side effect of methadone hydrochloride (6). Therefore, addiction to methadone, pump failure owing to massive emboli, and activation of vagal tone may explain the absence of tachycardia that we expected in our case. Bedside ultrasound allows direct visualization of pathology or abnormal physiological states, and thus has become an essential and useful component in the evaluation of hypotensive patients via RUSH protocol, which was clearly the key point in the diagnosis of our patient (7).

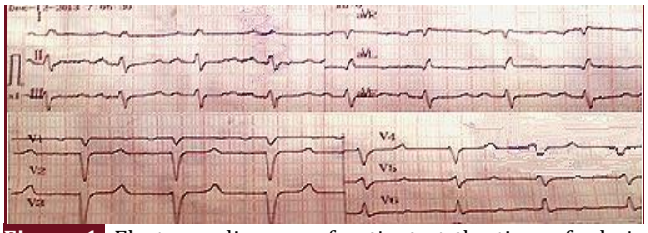

Figure 1: Electrocardiogram of patient at the time of admission. ⇧

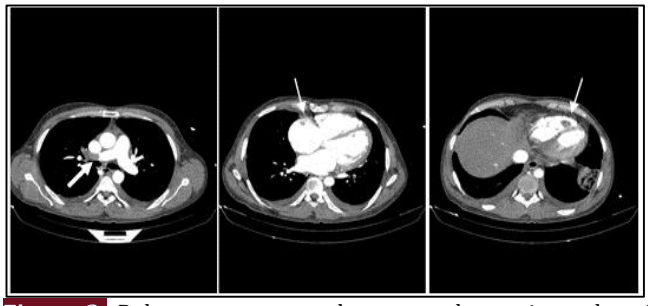

Figure 2: Pulmonary computed tomography angiography of the patient. ⇧

Conclusion:

PE is an important differential diagnosis of shock state, and hence, emergency physicians should pay more attention to it. In this context, RUSH should be considered as one of the vital signs (such as blood pressure or temperature), which needs to be evaluated when recording the history of patients presented to ED with signs and symptoms of shock.

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References:

1. Gantner J, Keffeler JE, Derr C. Pulmonary embolism: An abdominal pain masquerader. J Emerg Trauma Shock. 2013;6 (4):280-2.
2. Stein PD, Beemath A, Matta F, et al. Clinical characteristics of patients with acute pulmonary embolism: data from PIOPED II. Am J Med. 2007;120(10):871-9.
3. Courtney DM, Kline JA. Identification of prearrest clinical factors associated with outpatient fatal pulmonary embolism. Acad Emerg Med. 2001;8(12):1136-42.
4. Stein PD, Terrin ML, Hales CA, et al. Clinical, laboratory, roentgenographic, and electrocardiographic findings in patients with acute pulmonary embolism and no pre-existing cardiac or pulmonary disease. CHEST Journal. 1991;100 (3):598-603.
5. Arthur W, Kaye G. The pathophysiology of common causes of syncope. Postgrad Med J. 2000;76(902):750-3.
6. Ehret GB, Desmeules JA, Broers B. Methadone-associated long QT syndrome: improving pharmacotherapy for dependence on illegal opioids and lessons learned for pharmacology. Expert Opin on Drug Saf. 2007;6(3):289-303.
7. Perera P, Mailhot T, Riley D, Mandavia D. The RUSH exam: Rapid Ultrasound in SHock in the evaluation of the critically lll. Emerg Med Clin North Am. 2010;28(1):29-56.