Major Pulmonary Embolism

Kenneth E. Wood, DO[a,b,*]

KEYWORDS
- Pulmonary embolism • Shock
- Thrombolytic therapy • Embolectomy • Pathophysiology

The scope and spectrum of pulmonary embolism (PE) that are likely to challenge the Intensivist are dominantly confined to 2 scenarios; first, a patient presenting with undifferentiated shock or respiratory failure and, second, an established intensive care unit (ICU) or hospital patient who develops PE after admission. In either scenario, the diagnostic approach and therapeutic options are challenging. Differentiating PE from other life-threatening cardiopulmonary disorders can be exceedingly challenging, as logistic constraints can impair definitive diagnostic testing and the therapeutic approach can be appreciably altered given the hemorrhagic risks of the critically ill when anticoagulated or considered for thrombolytic therapy. This article will review a structured pathophysiologic approach to the diagnostic, resuscitative and management strategies related to PE in the ICU.

The incidence of PE causing, contributing, or associated with death in hospitalized patients has remained relatively constant at approximately 15% for the past 40 years. This is contemporarily illustrated by a recent autopsy case series of 600 ICU deaths where all patients received thromboprophylaxis for a reported overall PE incidence of 14.3%.[1] Of the 33 patients with clinically suspected PE, PE was confirmed at autopsy in only 39%. In 73 patients, PE was not suspected and was clinically discovered only at autopsy, where it was considered the cause of death in 45% of those patients. Recent abdominal surgery and the presence of renal failure were identified as factors associated with a higher risk of a misdiagnosis. The authors concluded that in an era dominated by thromboprophylaxis, critically ill patients remain at high risk for PE and consequent to the difficulties in establishing a diagnosis, the incidence of PE is higher than previously appreciated in critically ill patients. Contemporary estimates have suggested that PE occurs in more than 600,000 patients per year and is reported to cause or contribute to the death of between 50,000 and 200,000 patients. Large, contemporary observational studies of PE and similar registry studies have reported unexpectedly high mortality rates. In the Management Strategies and Determinants of Outcome in Acute PE (MAPPET) series, an overall 3-month mortality of 17% was reported in patients with PE and an in-hospital mortality of 31% when PE was

The author has nothing to disclose.
[a] Temple University School of Medicine, 3400 North Broad Street, Philadelphia, PA 19140, USA
[b] Geisinger Medical Center, 100 North Academy Avenue, Danville, PA 17822, USA
* Geisinger Medical Center, 100 North Academy Avenue, Danville, PA 17822.
E-mail address: kewood@geisinger.edu

Crit Care Clin 27 (2011) 885–906
doi:10.1016/j.ccc.2011.09.002 **criticalcare.theclinics.com**
0749-0704/11/$ – see front matter

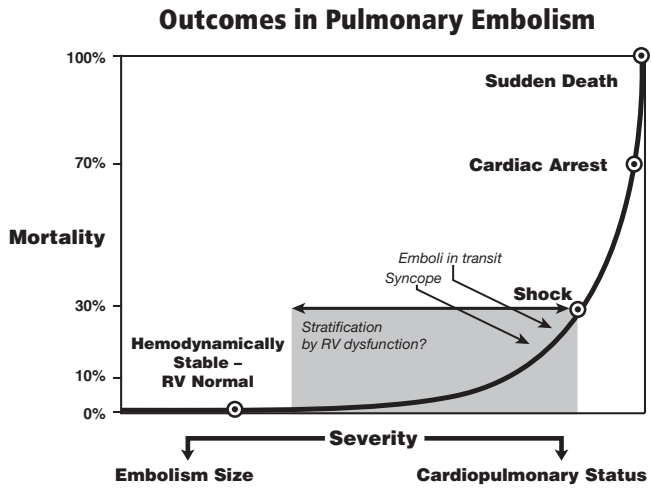

Fig. 1. Outcomes in PE. (*From* Wood KE. Major pulmonary embolism: review of a pathophysiologic approach to the golden hour of hemodynamically significant pulmonary embolism. Chest 2002;121;877–905; with permission.)

associated with hemodynamic instability. Mortality attributed to PE was 45% and 91% in the respective groups.[2] In the International Cooperative Pulmonary Embolism Registry (ICOPER), the 3-month mortality rate was 14.5% in the patients with hemodynamic stability and rose to 51.9% in those patients presenting with hemodynamic instability. Similar to the MAPPET report, PE-attributed mortality was 34% and 62.5% in the respective groups.[3]

In fatal cases of PE, it is long been recognized that approximately two-thirds of the deaths related to PE will occur within 1 hour of presentation and that anatomically massive PE will account for only half of the deaths, with the remainder attributed to nonmassive or recurrent PE. These observations have several important implications. First, using an evidence-based approach is almost impossible for the hemodynamically unstable patient, as a substantial number of these patients will die in the first hour and not undergo significant diagnostic studies. Second, it is reasonable to propose that patient outcomes from PE are related to the magnitude of the embolic obstruction against the background of the patient's underlying cardiopulmonary function. Given the dynamic interplay between a patient's underlying cardiopulmonary status and the magnitude of the embolus, similar hemodynamic presentations and clinical outcomes will manifest from an anatomically massive PE in a patient with normal cardiopulmonary function and an anatomically submassive embolus in a patient with impaired cardiopulmonary function. Third, an understanding of the pathophysiology of PE will enable the application of a physiologic risk stratification that can be used for the diagnostic evaluation and therapy of acute PE. **Fig. 1** illustrates a proposed risk stratification model defined by mortality on the ordinate and severity characterized by the cardiopulmonary status on the abscissa. Although not well appreciated, the combination of embolism size and underlying cardiopulmonary status that results in cardiac arrest is associated with a predictive mortality of 70%.[2] Given that 30% of patients with PE sustaining cardiac arrest will survive, this necessitates continued use of chest compressions to potentially mechanically fracture the PE and consideration of thrombolytic therapy or embolectomy occasionally in the absence of