Pulmonary Embolism

Current Role of Catheter Treatment Options and Operative Thrombectomy

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KEYWORDS

• Acute pulmonary embolism • Surgical pulmonary embolectomy
• Catheter thrombectomy • Thrombolysis • Submassive pulmonary embolism

KEY POINTS

- Acute pulmonary embolism continues to have an incredibly high mortality; more than double that seen with breast cancer.
- Anticoagulation and infrequent use of systemic thrombolysis or surgery has been the mainstay treatment options for decades.
- Less invasive techniques with more targeted therapeutic modalities have become increasingly used in the contemporary management with potentially less overall risk to the patient.
- Surgical embolectomy, although more invasive, still serves a critical and undeniable role in the effective management of this diverse patient population.

INTRODUCTION

The contemporary management of acute thrombotic pulmonary embolism (PE) has evolved significantly since the landmark clinical trial reported in *Lancet* by Barritt and Jordan[1] in 1960 established anticoagulation as the foundation of medical treatment. Even to this day, acute PE remains a devastating disease with an incredibly high prevalence, especially among hospitalized patients. It is estimated that PE accounts for at least 100,000 deaths in the United States annually[2]; however, the actual number is challenging to fully estimate, because patients with sudden death more commonly have their demise attributed to underlying cardiac disease rather than a thromboembolic cause. In Europe, of the 300,000 annual deaths attributed to PE, only 7% were diagnosed antemortem, with the remainder of victims being diagnosed at the time of death or postmortem.[3] These incredible statistics are only made more

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impressive when it is realized that annual PE mortality rates in the United States are more than double that seen with annual breast cancer mortality,[4] the latter a highly visible disease with widespread general public awareness because of an effective media campaign.

Over the past three decades, the overall understanding of venous thromboembolic disease has grown exponentially; but the treatment paradigm has changed little compared with other disease states associated with high prevalence and mortality, such as cancer, cardiovascular disease, and stroke. Undoubtedly, the use of anticoagulation and even systemic thrombolysis for a patient with massive PE has made meaningful impact on overall mortality, but it remains a binary approach to a much more complicated disease. The understanding of the natural history of PE has been greatly informed by several prospective studies and international registries. The International Cooperative Pulmonary Embolism Registry (ICOPER) followed 2454 patients over Europe and North America with the diagnosis of acute PE and found a surprisingly high mortality rate of more than 15% for all-comers at 90 days.[5] This registry is important because it exposed the fact that not all of the mortality was driven simply from patients presenting with cardiogenic shock, the so-called "massive PE" patients. Rather, this category comprised only around 4.5% of the entire patient population with the remainder 95.5% being "nonmassive PE" patients.[6] Clearly, there was great variability in the majority group, which historically was only being treated with unfractionated heparin and eventually a vitamin K antagonist. Since the late 1990s when ICOPER was published, the clinical evaluation of PE has evolved to more fully risk-stratify patients into subsets based on a host of clinical, epidemiologic, and radiographic criteria. The overall goal is to harmonize the intensity of therapy with the prognostic risk of the disease. Not all pulmonary emboli are created equally.

PATHOPHYSIOLOGY

Notwithstanding the complex cause of venous thromboembolic disease, the sequelae of PE should be viewed as a disease of the right ventricle (RV). More specifically, the sudden strain imposed on the right side of the heart from thrombotic outflow obstruction, referred to as acute cor pulmonale, sets off a complex chain of compensatory mechanisms that eventually fail if the burden is too great. The pulmonary circulation is normally a low-pressure, high-flow circuit powered by the RV, which itself has a limited ability to increase wall tension and stress. In the normal state, the nonpreconditioned, thin-walled RV cannot generate a mean pulmonary artery (PA) pressure greater than 40 mm Hg. When 30% to 50% of the total cross-sectional pulmonary arterial bed becomes occluded by thromboemboli, the PA pressures begin to increase, setting off a chain of neurohumoral compensation pathways aimed at overcoming the pressure demands.[7] In addition to the significant RV stain, acute PE is a disease of gas exchange primarily mediated through severe oxygen supply/demand mismatch from extensive dead space ventilation. These consequences lead to an unraveling of RV and left ventricular (LV) function, with evolving RV ischemia, increased deoxygenation, decreased RV cardiac output, resultant poor total cardiac output, decreased systemic blood pressure, and ultimately ensuing cardiovascular collapse and death.[8,9] Understanding the complex pathophysiology is critical in risk-stratifying patients to an individualized treatment algorithm that appropriately addresses their real-time needs. For example, if a patient has already evolved to cardiogenic shock with severe hypoxemia, they may be considered for more urgent operative treatment or placement on extracorporeal membrane oxygenation rather