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Original Contribution

Hemodynamic instability in patients undergoing pulmonary embolectomy: institutional experience

Jeremy M. Bennett MD (Assistant Professor)[a,*], Mias Pretorius Mb, Chb, MSCI (Associate Professor)[a], Rashid M. Ahmad MD (Assistant Professor)[b], Susan S. Eagle MD (Associate Professor)[a]

[a]*Department of Anesthesiology Vanderbilt University Medical Center, Nashville, TN 37232*
[b]*Department of Cardiac Surgical Sciences Vanderbilt University Medical Center, Nashville, TN 37232*

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Abstract

Objectives: Acute pulmonary embolism is a major cause of morbidity and mortality in patients presenting for emergent cardiac surgery with overall mortality ranging from 6% to as high as 85%. While the initial focus of treatment is nonsurgical or percutaneous interventions, surgical treatment continues to be a treatment for patients with refractory thrombus burden or cardiogenic shock. Our institution regularly performs surgical pulmonary embolectomy with improved outcomes compared to current reports. We thus performed a retrospective analysis of outcomes of pulmonary embolectomy patients and anesthetic management.

Design: A retrospective review of 40 patients undergoing emergent pulmonary embolectomy over a 4 year period (2008-2012) at our institution was performed to assess for a 2nd period of critical instability.

Setting: The study was conducted at a tertiary, level 1, trauma university medical center.

Participants: The study was performed through chart review of patient hospital records.

Interventions: No interventions were performed.

Measurements Anesthetic records were reviewed along with echocardiographic records and surgical reports to assess cardiac function, need for emergent cardiopulmonary bypass, and degree of patient morbidity.

Conclusions: A total of 40 patients were studied. Hemodynamic instability occurred in 12.5% of patients at time of induction requiring emergent cardiopulmonary bypass. Another 17% of patients who remained stable following induction developed subsequent instability requiring emergent cardiopulmonary bypass during pericardial opening or manipulation which has not been previously reported. One patient died during hospitalization. Patients who required emergent bypass following induction of general anesthesia tended to receive higher doses of induction drugs than the other groups. In patients who needed emergent bypass during pericardial manipulation there were no identifiable factors suggesting that these patients remain at risk despite a stable post-induction course.

* Corresponding author. Tel.: +1 615 322 4650.
E-mail address: Jeremy.m.bennett@vanderbilt.edu (J.M. Bennett).

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1. Introduction

Anesthetic management of patients undergoing pulmonary embolectomy is challenging given the emergent nature of the procedure, acute hemodynamic instability, patient comorbidities, and experience in various centers. Surgical management of pulmonary embolism is most often considered in patients with central or branch pulmonary artery thrombus burden with cardiogenic shock. It may also be elected for patients with stable hemodynamics who demonstrate right ventricular failure on echocardiographic examination or have failed medical therapy with thrombolytics, heparin, or percutaneous embolectomy [1,2]. A recent case series of 52 patients demonstrated a 19% incidence of hemodynamic collapse during induction of general anesthesia (GA) necessitating emergent cardiopulmonary bypass (CPB) [3]. Further studies have demonstrated overall mortality from surgical intervention ranging from 6%-85% [1,2]. Given the significant morbidity of these patients, surgical manipulation and stress are likely to impact venous return, pulmonary vascular resistance (PVR), and cardiac output of these critically ill patients. Because of paucity of anesthetic-related outcomes studies for this critical patient population, we performed a retrospective study over a 4-year period of patients presenting for pulmonary embolectomy. The aim of this study is to assess the prevalence of hemodynamic instability during induction of GA as well as at any time before institution of CPB with attention toward anesthetic technique.

2. Methods

A retrospective analysis was performed on all patients who underwent urgent/emergent pulmonary embolectomy at Vanderbilt University Medical Center between January 2008 and December 2012. A waiver of consent was obtained from the Vanderbilt Human Research Protection Program for assessment of patient records. Patients who arrived to the operating room (OR) endotracheally intubated or having received active cardiopulmonary resuscitation at any time before arrival in the OR were excluded from the study. Patient's medical records were reviewed for patient demographic information including but not limited to age, sex, right ventricle (RV) and left ventricle (LV) function, comorbidities (chronic lung disease, history of smoking, cardiac disease, renal insufficiency, and anemia), body mass index, prior cardiac surgery, and postoperative morbidity. Anesthetic records were reviewed for anesthetic induction agent (etomidate, fentanyl, ketamine, and propofol), inotropic administration, ventilator parameters (ventilator mode, peak airway pressures (PAWs), and positive end-expiratory pressure), and hemodynamic changes in heart rate (HR) and central venous pressure (CVP). Preoperative radiologic and transthoracic echocardiographic information was reviewed, if available as well as all intraoperative transesophageal echocardiographic (TEE) findings to assess thrombus location and burden, RV function, and RV dilation. Right ventricular function and dilation were determined based on accepted American Society of Echocardiography standards for assessment of the RV [4]. Preoperative transthoracic echocardiographic data were available in all patients with the exception of 2 patients in the stable group and 1 patient in both the induction-unstable and delayed-unstable groups (total of 4 patients). All patients received intraarterial catheters and central venous catheters before induction of GA using local anesthesia, supplemental oxygen as supplied by a facemask, and titration of sedation as dictated by the attending anesthesiologist. The location of these catheters was in the radial artery and right internal jugular vein, respectively, for most patients. If radial arterial catheter placement was difficult or unable to be performed, a femoral arterial catheter was placed. Sedation most commonly consisted of boluses of midazolam and ketamine. After central venous catheter placement, pulmonary artery catheters were placed in all patients and advanced to 20-cm depth until separation from CPB, at which time the pulmonary artery catheter was advanced into the main pulmonary artery. All patients were prepared and surgically draped with a surgeon scrubbed and ready in the OR before induction of GA. The type of anesthetic induction drug was at the discretion of the attending anesthesiologist. Transesophageal echocardiographic was performed in all patients after tracheal intubation, where there was no concern for esophageal pathology. Hemodynamic instability requiring emergent CPB was identified as acute and persistent hypotension occurring after GA induction that did not respond to vasopressor support and required emergent initiation of CPB. If patients remained stable after induction of GA, we assessed for any other period, when hemodynamic instability developed before CPB initiation. Surgeon and anesthesia records were reviewed for all patients if instability was identified for potential description of the event.

Data are presented as median with interquartile range unless otherwise indicated. Categorical data were compared between groups using χ^2 or Fisher exact tests as appropriate. Continuous baseline data were compared using the Kruskal-Wallis test. Comparisons of CVP and HR among groups were made with the Wilcoxon signed rank test. A 2-tailed P value less than .05 was considered statistically significant. Statistical analyses were performed with the statistical package SPSS for Windows (version 21.0; IBM, New York, NY).

3. Results

The study population consisted of 46 patients of whom 6 patients were excluded for preoperative cardiopulmonary resuscitation and/or arriving tracheally intubated before the OR. A total of 40 patients were included in the study and