PBLD – Table #5

Intraoperative Pulmonary Embolus

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Objectives

1. Determine patients who are at risk for perioperative pulmonary embolism
2. Form a differential diagnosis for intraoperative hypoxemia and confirm the diagnosis of PE
3. Management of intraoperative PE
4. Prevention of PE

Stem Case – Key Questions

A 14 y.o. male presented for resection of a posterior fossa tumor. He had 1 month of headaches and leaning to the right side when walking. Head CT demonstrated a 4 cm posterior fossa mass with ventriculomegaly. He was otherwise healthy and had no past surgical history. He was not taking any medications. Preop vital signs were stable and the patient did not demonstrate signs/sx of increased ICP. He underwent a smooth IV induction and easy intubation. He was positioned prone and anesthesia was maintained with isoflurane, remifentanil infusion, and vecuronium. A few short episodes of HTN and tachycardia were noted during brainstem manipulation. The resection was otherwise uneventful. Upon returning the patient to the supine position, there was a sudden decrease in ETCO2 (37 to 18). O2 sat remained 100% on FiO2 of 1. Cyanosis of the head and trunk were noted. The patient rapidly became hypotensive with SBP in the 50s.

What is the differential diagnosis of a sudden decrease in ETCO2?

What actions would you take immediately to stabilize this patient?

Are there any studies you would perform to confirm a diagnosis?

The position of the ETT was confirmed with auscultation of bilateral breath sounds. The patient was reintubated regardless to rule out the possibility of any kind of obstruction without improvement. An ABG was sent: 7.06/74/399/-10, lactate 4, PCV 40. The hypotension was treated with multiple boluses of epinephrine. CPR was initiated for persistent hypotension and bradycardia. The patient was defibrillated x1 for VT. The diagnosis of PE was suggested, and cardiothoracic surgery was emergently consulted. The patient was deemed too unstable for transport for a CT angiogram. A TTE was performed.
How is the diagnosis of PE confirmed?

What are the echocardiographic findings of acute PE?

TTE revealed RV hypokinesis, RA dilation and bowing of the atrial septum into the LA, and pulmonary hypertension.

What is the pathophysiology of circulatory failure and shock due to massive PE?

What is the treatment of acute PE?

The patient underwent emergent thoracic exploration. CPB was initiated, and a large R PA thrombus and small L PA thrombus were discovered. Thrombectomy was performed, the patient was separated from CPB, and remained stable on milrinone and norepinephrine infusions. An immediate post-op head CT was performed demonstrating a small IVH in the lateral ventrical. The pt was transferred to the PICU where he continued on pressor support and required multiple transfusions for DIC. His neuro exam declined over the next 24 hours, and an ICP monitor revealed ICPs in the 80s. A F/U head CT demonstrated a new large hematoma within the surgical cavity with mass effect in the posterior fossa and superior transtentorial and tonsillar herniation. Findings were also consistent with anoxic brain injury. The family elected to withdraw care 2 days post-op.

What patients are at increased risk for perioperative DVT/PE?

What measures can be taken to prevent perioperative PE?

PBLD Discussion

While pulmonary embolism is a well-described phenomenon in adult literature, there is less information available about pediatric patients, and therefore the diagnosis and management in this population is mostly based upon adult experience. A Canadian registry from 1990 reported an incidence of DVT/PE in patients age 1 mo. to 18 years of 0.07/10,000. Data from Australia reports a rate of 8/17,500 in 1 mo. to 13 y.o., and a Dutch study 0.05/10,000 (1). Results from the National Hospital Discharge Survey of U.S. hospitals published in 2004 found a diagnosis of PE in 0.9/100,000 children/year between 1979-
2001, 4.2 DVT/100,000, and 4.9 PE and/or DVT/100,000/year. The rate of diagnosis did not change over any 10 year period studied (2). However at least one retrospective study noted an increase in the recognized number of DVTs, which are on a continuum with PE, in their pediatric population from 0.3 to 28.8/10,000 children between 1992 and 2005 (3). Increased incidence of pediatric DVT/PE may be due to advances in care of patients with prematurity, congenital heart disease, and malignancies. In the adult population, 200,000 patients die from PE each year, making it the third leading cause of death (1).

Most children who present with PE have at least one or more risk factors for DVT/PE. These include presence of a central venous catheter, particularly in infants. Others are malignancy, cardiac anomalies, surgery, infection, trauma, oral contraceptives, obesity, congenital prothrombotic disorders, and lupus. Prothrombotic disorders include protein C and S deficiency, antithrombin III deficiency, and factor V Leiden (1,4,5). DVT/PE is uncommon in pediatric neurosurgical patients. One study looked at 462 children with brain tumors, 384 of whom underwent surgery and 78 of whom were medically managed. Only 3 patients, all adolescents, developed DVTs, and 2 of these developed PEs (6).

In an unanesthetized patient, PE may present with dyspnea, chest pain, cough, and hemoptysis. Tachypnea and tachycardia may be observed, and rales and a 4th heart sound may be appreciated. Limb swelling and pain may be noted in the case of a DVT. A massive PE may present with syncope, severe hypotension, extreme hypoxemia, PEA, and cardiac arrest. EKG findings are consistent with cor pulmonale including an S1, Q3, T3 pattern, RBBB, and R axis deviation (5). A sudden decrease in ETCO2 is consistent with massive PE, though the differential diagnosis also includes myocardial ischemia, heart failure, and pericardial tamponade, bronchospasm, pneumothorax, and ETT obstruction or displacement.

In a stable patient, the diagnosis of PE is commonly confirmed with contrast-enhanced spiral CT arteriography. Other modalities include ventilation-perfusion scans, MRI, pulmonary angiography, CT venography, and ultrasound to evaluate for DVT. CXR is primarily useful for ruling out other pulmonary processes. D-dimer test results are non-specific in the setting of infection, cancer, trauma, and other inflammatory states, and therefore of little value intraoperatively (5).

TTE and TEE are useful for direct visualization of emboli as well as demonstrating secondary signs such as pulmonary hypertension, RV hypokinesis, TR, and bowing of the interatrial septum right to left throughout the cardiac cycle (7). In addition to venous thromboembolism, acute pulmonary hypertension may develop secondary to CO2 embolism during laparoscopy, air embolism, bone cement in orthopedic surgery, protamine administration, and ischemia reperfusion syndrome after clamping the aorta. Echocardiography would certainly seem the most convenient modality for diagnosing PE in a patient too unstable for transport. One study pointed out that in the setting of massive PE and ongoing resuscitative measures, circumstances are less than ideal for a thorough echo exam. Indeed, in their series of 46 patients undergoing emergent pulmonary embolectomy, sensitivity of TEE was only 46%. They conclude
that failure of TEE to demonstrate a thromboembolus should not preclude further study or treatment and that echo might best be used as a monitor of hemodynamic status intraoperatively as opposed to a primary diagnostic tool (7). Other studies have reported a higher sensitivity and specificity for TEE, but these were performed on more stable sedated patients.

Massive PE is defined as obstruction of 50% or more of the pulmonary vascular bed. In addition to the physical obstruction of the embolus, the release of vasoactive substances causes further vasoconstriction and acute increase in PA pressure. This can lead to RV dilation and dysfunction, decreased LV filling, and ultimately circulatory collapse. RV dysfunction is in fact an independent predictor of mortality, and when associated with shock in the setting of massive PE carries a 30% in-hospital mortality rate (8). Immediate stabilization measures include control of ventilation, judicious fluid administration to avoid further RV dilation, and pressor support including norepinephrine, epinephrine, and dopamine (5,8).

Treatment of PE entails anticoagulation, IVC filter placement, thrombolysis, and embolectomy depending on the patient’s presentation and comorbidities. In the setting of PE with cardiogenic shock, thrombolytics such as t-PA are indicated, however absolute contraindications to thrombolytics include active internal bleeding and recent intracranial bleeding, clearly making them an impractical choice in the perioperative setting. Pulmonary embolectomy is therefore typically pursued in this setting, though some argue that patients that survive embolectomy would have survived with medical management alone (8). Pre-op cardiac arrest increases mortality following embolectomy by more than 50% based on adult literature (8).

Recommendations for DVT prophylaxis in infants and children are not well-defined. One recent retrospective study suggested that pediatric patients with severe medical conditions who require a prolonged ICU stay, particularly with central venous access, should be considered for prophylaxis with either unfractionated or low molecular weight heparin (3). Other studies looking at pediatric trauma patients found the incidence of DVT/PE to be too low to warrant routine prophylaxis (9). There is one report of IVC filters placed in a series of 10 adolescent patients, four following PE, six for deep venous thrombosis (DVT) when anticoagulation was contraindicated, and one inserted prophylactically (10). A more recent cross sectional study of trauma patients 17 y.o. and younger found that IVC filters were placed in 0.18% of patients and was unable to comment on their efficacy due to lack of long term follow up data (11).

References


