Pulmonary Thromboendarterectomy in a patient with Supra-systemic Pulmonary Artery Pressures

Problem Based Learning Discussion
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OBJECTIVES

At the completion of this PBLD, the participant will be able to
1) describe the etiologies and incidence of thromboembolic disease
2) discuss the preoperative evaluation and hemodynamic considerations for Chronic Thromboembolic Pulmonary Hypertension (CTEPH).
3) describe the intraoperative anesthetic management and monitoring considerations for patients with severe supra-systemic pulmonary hypertension.
4) explain the management of patients with pulmonary hypertension going for non-cardiac surgery

CASE PRESENTATION

49 y/o woman with a history of chronic thromboembolic pulmonary hypertension is admitted in preparation for pulmonary thromboendarterectomy. Pt suffered a deep vein thrombosis (DVT) and subsequent massive pulmonary embolism (PE) several years prior, during convalescence from foot surgery. At the time of the PE she developed acute cardio respiratory failure requiring mechanical ventilation. She was placed on thrombolytic therapy followed by heparin and then Coumadin. Subsequently, a Greenfield filter was placed.
QUESTIONS

Q1. What are the most common hypercoaguable disorders?

Q2. What is the incidence of DVT & PE following lower extremity fractures without thromboprophylaxis?

Q3. How long should one be therapeutically anticoagulated with a combination of heparin and Coumadin, before resolution of clot occurs?

Q4. Which patients should get an inferior venacava (e.g. Greenfield) filter?

Q5. Do the pulmonary artery pressures return to normal after an initial PE following prompt and complete initial anticoagulation therapy?

Q6. What is the pathophysiology of pulmonary hypertension in these patients?

Q7. How many patients go on to develop Chronic Thromboembolic Pulmonary Hypertension (CTEPH)?

Q8. Why do some patients develop CTEPH and others do not?

CASE CONTINUATION

Over the past year, pt noted progressive exertional dyspnea - unable to walk one block (NYHA class III). She also noted increasing lower extremity edema. More recently, walking across the room has triggered worsening SOB.

PMH: Hx of Antiphospholipid antibody syndrome - otherwise unremarkable

Medications: Sildenafil 50 mg po TID, Lasix 40 mg po BID, Coumadin 2.5 mg po daily

Vitals: BP 105/58 HR 105 RR 19 SpO2 89% 10 L O2 by face mask

Physical Examination:
Height: 5'4 f, Weight: 185 lb, BMI: 31.8
Obese, dusky in color, lips cyanotic
Sitting upright – 3 to 5 word orthopnea - receiving O2 10L/facemask
Airway: Class II, full ROM of mandible and cervical spine
Lungs: Clear (bilateral)
Heart: regular, II/VI systolic murmur noted between scapulae
Abdomen: large, hepatomegaly
Extremities: bilateral lower extremity edema

Q9: What are the causes of cyanosis?
Labs: Electrolytes WNL, Hct 49.2

Q10: What do you think of this hematocrit?
Platelets -230
Hepatic enzymes moderately elevated
EKG - sinus at 114, right axis deviation, incomplete RBBB, inferior strain pattern
CXR - Clear lung fields (diminished vascular markings bilaterally)
Echo - EF 73%, mild left ventricular diastolic dysfunction
RV markedly enlarged and hypertrophic - mildly depressed function
RA markedly enlarged - moderate TR
TR envelop 5 m/s, PAP 100 mm Hg + CVP

Q11: What do you think about the Echo findings?
Right heart cath - RAP 18 RV 105/15, PAP 107/55 (74)
CO 3.1, CI 1.0
PVR 1738 dyne/sec/cm^5
Left heart cath - no significant CAD

Q12: What do you think about the Cath data? What are the most important numbers?
Does the RVEDP of 15 have any meaning to you? PVR > SVR what does that mean? Is the blood flowing backward? Wedge pressure of 6mmHg – What are your thoughts? Is this expected?

V/Q scan - moderate-to-large ventilation perfusion mismatches
Pulmonary angiogram - suggestive of CTEPH

Q13: What is the RV response to increased volume and pressure?

Q14: What conditions will exacerbate the baseline pulmonary hypertension?

Q15: What is the typical preparation for a patient prior to PTE?

Q16: What are your hemodynamic goals in terms of rate, rhythm, preload, afterload and contractility?

Q17: What monitoring will you plan to use in this case?

Q18: If you plan to use a PA catheter, what are the pros and cons to placing awake prior to induction vs. asleep following induction?

Q19: If a V-fib arrest should occur, how effective are external chest compressions in patients with this degree of increased pulmonary vascular resistance?

Q20: NOW, what is your plan for induction?
**Q21:** Which rescue drugs would you like to have available?

**CASE CONTINUATION**

**Induction:**
Pt brought to OR with 18G PIV and right radial A-line in addition to standard monitors Preoxygenated for 10 minutes to SpO\textsuperscript{2} 89%.

Etomidate 10 mg, fentanyl 250 mcg, midazolam 5 mg, rocuronium 100 mg, dopamine infusion at 5 mcg/kg/min

Easy mask ventilation and intubation  
ETT secured and mechanically ventilated with 0.5 MAC Isoflurane  
Pt SpO\textsuperscript{2} drops to low 80's  
BP drops from 100/60 to 70/40.

**Q22:** How would you treat the drop in SpO\textsuperscript{2} and BP?

Dopamine increased to 10 mcg/kg/min and epinephrine infusion started at 0.3 mcg/kg/min. **Why not phenylephrine?**

Surgeons notified and rushed to OR, rapid surgical prep and surgery started

SpO\textsuperscript{2} continues to decline to 69% while cardiopulmonary bypass (CPB) established  
SBP continues to progressively decline to 60's after initial response to epinephrine and dopamine to 90's  
TEE performed, CPB established

**Q23:** What would you expect to see on TEE examination?

**CASE CONTINUATION**

Surgery proceeded without complication. A right pulmonary embolectomy was performed, followed by right and left pulmonary endarterectomy under profound hypothermia and circulatory arrest.

**Q24:** Upon separation from cardiopulmonary bypass large amounts of dark blood emanate from the endotracheal tube. What is the most likely cause for this, and how will you proceed?

**DISCUSSION**

**Chronic thromboembolic pulmonary hypertension (CTEPH)** is obstruction of the major pulmonary arteries resulting in pulmonary hypertension. The incidence of CTEPH is underappreciated and as a result is currently an under-treated phenomenon.
**Pulmonary Thromboendarterectomy (PTE)** is the treatment of choice for patients with chronic, thromboembolic pulmonary hypertension. PTE is the surgical technique whereby surgeons remove organized thrombi along with a thin lining of intima while leaving the media intact. The surgery provides immediate and permanent relief of the pulmonary hypertension associated with the sequel of unresolved pulmonary thromboembolic disease.

In the United States alone, around 900,000 people will develop blood clots in the lungs or major veins annually: 400,000 cases have contributed to non-fatal DVT, 200,000 cases of non-fatal PE and 300,000 cases of fatal venous thromboembolism (VTE).

Approximately 10% of will die within one hour due to cardiovascular collapse or respiratory failure. Approximately 3.8% of patients suffering a PE will develop CTEPH. (The vast majority of patients do not develop the disease.) Unfortunately, many patients with CTEPH have no clear history of an acute thromboembolic event. It is likely an under-diagnosed process.

Those with clear thromboembolic event subsequently enter into “honeymoon phase” with progressive remodeling of unobstructed vasculature. CTEPH can develop in the absence of documented recurrent thromboemboli.

**The most common causes of hypercoaguable disorders include the following:**

**A. Inherited hypercoaguable conditions**

- Factor V Leiden (the most common)
- Prothrombin gene mutation
- Elevated levels of fibrinogen
- Deficiencies of natural proteins that prevent clotting (anticoagulant proteins)
- Antithrombin
- Protein C and protein S
- “Sticky” platelets
- Abnormal fibrinolytic system

**B. Acquired hypercoaguable conditions**

- Cancer
- Recent trauma or surgery
- Pregnancy and exogenous estrogen use
- Hormone replacement therapy
- Immobility for any reason (i.e. illness, surgery, prolonged airplane travel)
- Heparin-induced thrombocytopenia
- Antiphospholipid antibody syndrome
- Previous deep vein thrombosis or pulmonary embolism
- Myeloproliferative disorders such as polycythemia vera or essential thrombocytosis
PTE patients usually present with progressive dyspnea on exertion, particularly in young individuals. Often there is a history of documented pulmonary embolism (PE). Often patients have risk factors for DVT. Findings on physical examination of these patients are often consistent with right heart failure. These findings include peripheral edema, hepatomegaly and jugular venous distention. Precordial examination often reveals right ventricular heave and a unique murmur. This systolic sound results from turbulent flow moving through partially obstructed pulmonary vessels. Patients with atrial septal defects may present with cyanosis. Hepatomegaly and ascities develop late in the disease. Liver function tests may be abnormal reflecting hepatic congestion.

EKG findings show evidence of right ventricular hypertrophy with strain. CXR often reveal clear lung fields with prominent hilar regions. Enlarged right atrium and right ventricle are commonly noted. Arterial blood gas may reveal increased A-a gradient which often worsens with exercise, while the V/Q scan findings are consistent with moderate-to-large ventilation perfusion mismatches. Interestingly, signs of pulmonary hypertension and right ventricular failure on echocardiography often represent the first major clues in the diagnosis of CTEPH.

Right heart catheterization demonstrates the severity of pulmonary hypertension. Typically patients have PVR > 300 dyne/sec/cm$^5$ with mean PA pressures > 25 mmHg. Dilated right ventricle (RV), global RV dysfunction and RV compression of left ventricle (LV) with bowing of the interventricular septum toward LV during systole are common findings on echocardiography. Tricuspid regurgitation is often noted while thrombus may be seen in the main PA and/or right PA.

Pulmonary angiography usually shows irregular arterial contours with abrupt cut-off or narrowing of vessels, pulmonary artery webs and bands (organization of the thromboembolic material in the vessel lumen with subsequent scar formation), pouch defects, obstruction of lobar or segmental arteries at their point of origin.

Candidates for PTE, keep in mind that this is a big case with significant risk. The patient must have functional cardiac impairment: typically NYHA class III or IV. In addition, they must have hemodynamically significant pulmonary vascular obstruction. Typically PVR > 300 dynes/sec/cm$^5$ and PA pressure > 25 mm Hg. It is not uncommon to encounter values > 1000 dynes/sec/cm$^5$ and suprasystemic pulmonary artery pressures. The patients should not have any concurrent illness that is immediate threat to life and they must desire surgery based on poor cardiorespiratory function. This is a big procedure with significant risk. The patient must be willing to accept the significant mortality risk of procedure.

**Pulmonary Thromboendarterectomy:** True endarterectomy involves stripping of the diseased intimal layer from the media. It is performed through a median sternotomy on cardiopulmonary bypass. Deep hypothermic circulatory arrest is employed during the endarterectomy to prevent bleeding from the bronchial collaterals.
**Hemodynamic consideration** for induction and the Pre-cardiopulmonary Bypass period include the following

1) Several days prior to surgery, IVC filter is placed to prevent future emboli.
2) Awake radial arterial line and large bore peripheral intravenous catheter. Hemodynamic assessment and decision making is centered on right ventricular function. Because of the right-sided pressures, the coronary blood supply to the right ventricle is at risk.
3) Maintenance of adequate systemic vascular resistance, adequate inotropic state, and normal sinus rhythm serve to preserve systemic hemodynamics as well as RT ventricular coronary perfusion.
4) Preoperative cardiac cath data are useful in determining the induction sequence. Typically avoid sedation outside the OR due to risk of hypercarbia with hypoventilation. Induction accomplished with combination of fentanyl, midazolam, etomidate and muscle relaxation. Inotropic support may be necessary during induction with very poor RT ventricular function.
5) Consider induction with inotropic support if there is evidence of impending cardiovascular decompensation

**Signs of impending decompensation include:**
- RV end diastolic pressure > 14 mm Hg
- Severe tricuspid regurgitation
- PVR > 1000 dynes/sec/cm$^5$

**Post induction monitoring:** The following are recommended

1) Internal jugular cordis and pulmonary artery catheter.
2) Femoral arterial catheter is placed because of prolonged hypothermic CPB; the radial artery catheter significantly underestimates systemic pressures in the post CPB period.
3) Intraoperative TEE is valuable in monitoring and assessing cardiac function during PTE.
4) Processed encephalogram is monitored throughout the procedure. This allows confirmation of isoelectric EEG. Cerebral oximetry is also used.
5) Temperature monitoring is measured via urinary catheter and rectal probe for core temperatures estimation. Tympanic membrane probe is used for estimation of brain temperature.
6) PAC measures blood temperature, allowing quantification of thermal gradients.

**Surgical Approach and Initiating CPB:** After median sternotomy, CBP is established with cannulation of inferior and superior vena cava and aortic cannulation. Pt is cooled to a core temperature of 20°C and a tympanic temp of 15°C with cooling blanket around the head. Circulatory arrest is achieved and sodium thiopental administered to achieve isoelectric EEG during endarterectomy of each pulmonary artery. Pt is subsequently rewarmed requiring 90-120 minutes to achieve 26.5°C. The process of separation from CPB is similar to other surgeries involving CPB. Keep in mind that end-tidal carbon
dioxide is a poor measure of ventilation adequacy in these patients both before and after CPB, because dead space ventilation is an integral part of the disease.

**Post cardiopulmonary Bypass Period:** Reperfusion pulmonary edema can occur usually presenting as frothy sputum. If frothy sputum arises, the ET tube should be suctioned and increasing amounts of PEEP applied. If frank blood, not frothy sputum, is returning from the endotracheal tube, surgical bleeding is the probable cause. If severe bleeding persists, fiberoptic bronchoscopy should be used to evaluate the source of bleeding. Lung isolation should be considered.

**Treatment options for pulmonary arterial hypertension.**

The goal for treatments in patients with PAH is to improve symptoms and quality of life. Another important objective is to lower PAP and normalize cardiac output as early in the disease process as possible before right ventricular failure ensues.

Anesthetic management of individuals with PAH undergoing cardiac or general surgery is challenging because perioperative increases in PVR readily occur and may provoke right-sided heart failure, resulting in death. (The tolerance of the RV to these sudden increases in PVR is a major concern and should always be in the forefront of providers’ considerations.)

In general, intravenous anesthetics have less effect on hypoxic pulmonary vasoconstriction, PVR, and oxygenation than do volatile agents. Nitrous oxide has been reported to increase PVR, but it is not contraindicated in these patients. Isoflurane may be beneficial by decreasing PAP and has been frequently used during noncardiac procedures. Fentanyl may be given as an adjunct or a primary anesthetic agent in these patients because it causes little myocardial depression and excellent circulatory stability.

**References:**

6. McGlothlin D, Marco TD, Preoperative Risk Assessment of Pulmonary Arterial Hypertension Patients Undergoing General Surgery. Advances in Pulmonary Hypertension_Summer 2007; 6(2)