Surgical Management of Pulmonary Embolism

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PGY 3.85
Clinical Presentation

- 51F presented to Brookdale ED with c/o fatigue and SOB, CTA chest demonstrated bilateral large saddle pulmonary emboli. Patient was deemed poor candidate for lytic therapy given elevated INR and thrombocytopenia
- Transferred to Downstate for possible surgical embolectomy
- Vitals: 97.1/102/101/71/33/100%
- Labs: 131/2.2/89/28/18/1.17<122 5.6/2.59/16/14/48/1.7 8/2.1/2.8 9.88>13.4/38.8/82 INR 2.6
- Patient taken for OR embolectomy
Pulmonary Embolectomy and Left Pulmonary Artery Endarterectomy

- 4 units PRBC, 129 minutes cardiopulmonary bypass, 26 minutes aortic cross clamp time, 9:23 minutes of cardiac arrest
- Left pulmonary arteriotomy was made towards left PA, 20cm clot evacuated, fiber optic scope was used to evacuate further clots and left PA upper and lower endarterectomy was performed, R PA was evacuated in similar fashion
- Patient rewarmed, weaned from CPB and the chest was closed
- Patient became hypoxic in OR, and ECMO was reinstituted, and patient emerged from operating room on inotropic support.
Intraoperative Findings (i.e. the squeeze)
Post Operative Course

**POD#0**
- RV dysfunction requiring VA ECMO, intubated and sedated requiring Triple pressors + Methylene Blue injection. Hyperkalemia, lactic acidosis

**POD#2**
- Improvement in hemodynamics, dual pressors only, lactic acidosis improving, BM and Hyperkalemia resolved

**POD#3**
- Received 3/2/4/10 24 hours, shiley placed aquadex therapy begun, switched to argotraban given concern for HIT, persistent lactic acidosis, worsening BUN/CR with oliguria. Milrinone started, ECMO weaned

**POD#4**
- Triple pressors restarted, continued Oliguria, Aquadex Use
POD#5
- Received 1 platelet, started on Bicarbonate drip overnight with PRN pushes, Epinephrine drip titrated, Nitric Oxide started, CVVHD started today. VV ECMO initiated, Bronchoscopy performed.

POD#6
- Up trending pressors, continued on NO, VV ECMO, CVVHD, Bicarbonate Drip with PRN pushes, 2 units PRBC and 1 unit platelets transfused

POD#7
- Additional 2 units PRABC and 1 Unit platelets HIT negative

POD#8
- No acute events, remains on therapies

POD#9
- No acute events, remained on therapies

POD#10
- Patient converted to VA ECMO, multiple bicarbonate pushes overnight, became asystolic with ROSC, elevated potassium with asystole pronounced at 16:10
Questions?
Pulmonary Embolism

- First described by Rene Laennec in 1819
- Also first to describe its association to deep venous thrombosis
- Also the first to describe peritonitis and name cirrhosis
- Laennec was also famous for the invention of what other critical diagnostic tool?
The stethoscope

Invented when attempting to diagnose a young woman with a diseased heart in whom “percussion and application of the hand were of little avail on a great degree of fatness”

Also avoided the embarrassment of placing ones ear against the chest of a young woman
Rudolph Virchow

- Famously described the three factors predisposing towards venous thrombosis
  - Stasis, hypercoagulability and vessel wall injury
- Directly proved the pathogenesis of pulmonary embolism by recovering clot from humans and inserting them into the jugular veins of dogs
Pulmonary Embolism in the US

• 63 in 100,000 patients based on clinical/radiographic data

• 235,000 deaths in the US on autopsy

• ½ prevalence of MI and 3x more likely than CVA

• Often an autopsy finding where its role in death was unsuspected

• 3 most common cause of death after MI and cancer
Sequelae of PE

• Survivors of the acute event, 3.8% will go on to develop pulmonary hypertension

• Following development of pulmonary hypertension >50 mmHg 3 year mortality exceeds 90%
Pathophysiology in the pulmonary vasculature

- Mechanical obstruction, degree of compliance/age directly affects pulmonary vascular resistance and hemodynamics

- Serotonin, ADP, PDGF and thromboxane released by clot causes ↑↑ PVR

- Furthermore, downstream ischemia causes relative vasoconstriction
Shunting

V/Q ratio is PE is ???
0

Mucous Plug ???
infinity
Clinical Presentation

• Most PE’s clinically silent, obstruction of 50-60% prior to hemodynamic sequelae

• Tachycardia, rales, low grade fever, pleural rub, most have normal PaO2, PaCO2

• High index of suspicion necessary for diagnosis
Diagnosis

- ABG non specific or diagnostic, non specific EKG changes and CXR findings
- V/Q scans effective but logistically difficult to organize
- Pulmonary angiography the gold standard
- CTA has superseded PA angiography based on convenience, availability and lack of invasiveness
- Transthoracic echocardiogram is useful adjunct for the evaluation of right heart function
Unfortunately

There is no Patty the PE dog to aid in detection…

Figure 1.
Cliff in detection dog outfit on one of the hospital wards.
Classification

- Pulmonary embolism-no vascular compromise, no evidence of myocardial necrosis or right heart strain, <30% occlusion of PA
- Submassive PE-no evidence of hemodynamic instability, with evidence of right heart dysfunction or myocardial necrosis 30-50% occlusion
- Massive-systemic hypotension requiring inotropic support usually results in severe organ dysfunction
Treatment Options

Clinical probability of pulmonary embolism

Establish diagnosis (ECHO, CT angiogram, cardiac biomarkers)

Hemodynamically stable

No RV dysfunction

Anticoagulation contraindicated

Heparin or LMWH

Recrrent embolism

Caval filter

Coumadin

RV dysfunction

Thrombolysis

Cot fragmentation

No clot fragmentation

Thrombolysis contraindicated

ELS and/or embolectomy

Hemodynamically unstable
Who should receive what therapy

- Which patients should receive what therapy:

A. 80M incidental PE hemodynamically stable with evidence of right heart strain

B. 56M new PE, hypotensive with evidence of right heart strain

C. 63M s/p R fem pop bypass with new PE, right heart strain and hypotension requiring pressers
Anticoagulation

• Low molecular weight heparin/heparin remain the primary therapy in the hemodynamically stable “teaching point” bolus heparin on the way to CT scan if suspicion is high and risk of bleeding is low

• Anticoagulation prevents propagation, **DOES NOT** cause lysis, body’s intrinsic system causes lysis

• Monitor for HIT, switch to non heparin based anticoagulation if suspected i.e. argatroban

• Warfarin remains the gold standard for long term anticoagulation, but novel anticoagulants are acceptable, 3 months minimum treatment for provoked DVT, 6 months for unprovoked DVT/PE
Thrombolytic Therapy

- Initial studies demonstrated little benefit for thrombolytic therapy vs anticoagulation.

- Newer literature demonstrates improved mortality in patients with evidence of right heart strain and systemic hypotension. No benefit in patients with right heart strain alone.

- No evidence of benefit of systemic vs catheter directed thrombolytic therapy.
Percutaneous Embolectomy

*It Exists….*

Three Main Types:

1. Suction catheter
2. Thrombus fragmenters, i.e. impella devise
3. Rheolytic thrombectomy
Surgical Embolectomy

Indications:

1. Critical hemodynamic condition with low survival likelihood

2. Main/lobar pulmonary embolism with impaired gas exchange

3. Unstable patients with absolute contraindications to thrombolysis/anticoagulation

4. Large clot trapped in atrium or ventricle
Pulmonary Embolectomy

- Trendelenberg described in 1908, with very poor outcomes

- Dr. John Gibbon, inventor of cardiopulmonary bypass, described ECMO in a note 1931, as

  "a means to allow the surgical removal of pulmonary emboli"

- First successful pulmonary embolectomies using CPB were described by Cooley (1961) and Sharp (1962)
Technique

- Median sternotomy, CPB, occlusion of SVC & IVC.
- Longitudinal incision 1-2cm distal to pulmonary valve-occasional extension in left PA as necessary.
- Right PA counter incision occasionally used for improved exposure.
- Clot evacuated direct/fiber optic vision, with forceps/fogarty balloon catheter & suction catheters.
- Pleural entry to squeeze lungs remove clots. Repair with 6-0 prolene suture.
- IVC filter placement varies upon institution.
ECMO

- The use of ECMO has been described as both a therapeutic and supportive therapy
- Cardiopulmonary support while allowing:
  - Heparinization
  - Infusion of fibrinolytics
  - Bodies own fibrinolytic system to take action
Results

• Mortality reports in the literature vary wildly from 0-%-62%
• MI(27-64%) & ELS requirements(44-57%) are most associated with increased mortality
• 80% of survivors have normal PA pressures & exercise tolerance with no further evidence of disease
• Patient’s with continued PA obstruction > 40% demonstrate increased PA pressures
Retrospective Review

Midterm benefits of surgical pulmonary embolectomy for acute pulmonary embolus on right ventricular function

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- Retrospective review at a single tertiary referral center
- 44 patients
- 35-submassive PE, 9 massive PE requiring inotropy
- 1 in hospital mortality, embolectomy and CABG combined
- 21 patients with ECHO follow up, 12 with mid term follow up
- 21 patients demonstrated improvement in right ventricular hemodynamics
- 12 patients with mid term follow up demonstrated sustained RV, improvement with minimal evidence of valve dysfunction
Retrospective Review

Outcomes After Surgical Pulmonary Embolectomy for Acute Pulmonary Embolus: A Multi-Institutional Study

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• Retrospective Multi-Center Trial
• Poor standardization, limited chart review
• 214 patients, 176 submassive, 38 massive
• 11.7% mortality rate, 23.7% massive PE, 9.1% submassive PE
• 28 patients experienced cardiac arrest with mortality rate of 32.1%
• 3 month mortality of massive PE 58%, submassive PE 21%
Surgical pulmonary embolectomy: Should we extend its role?

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Management of acute massive pulmonary embolism: Is surgical embolectomy inferior to thrombolysis?☆

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A B S T R A C T

Background: Although current guidelines for pulmonary embolism (PE) treatment recommend surgical embolectomy when thrombolysis is contraindicated or has failed, their clinical outcomes rarely have been compared directly.

Methods: After excluding patients aged under 18 years and those with submassive or non-massive PE, 45 consecutive patients (median age, 68 years; 62% female; 31% experienced cardiac arrest before PE treatment onset; 33% had cancer diagnosis history; and 29% received extracorporeal membrane oxygenation [ECMO]) who underwent only thrombolysis (TL group; n = 19) or surgical embolectomy (SE group; n = 26, including 4 who had failed thrombolysis) for acute massive PE from 2000 to 2013 at Samsung Medical Center were enrolled to assess cardiac mortality as primary outcome.

Results: Median follow-up duration was 17.2 months. In the SE group, significantly higher proportions of patients had recent surgery and ECMO. Overall 30-day all-cause mortality rate was 24% (n = 11), without significant difference between the SE (15%) and TL (37%) groups (P = 0.098); however, cardiac mortality rate was significantly higher in the TL than SE group (Log rank P = 0.023). TL was an independent multivariate predictor of cardiac death (P = 0.03).

Conclusion: In this small retrospective single center experience, surgical embolectomy is associated with lower cardiac mortality risk than thrombolysis, which might render it first-line treatment option for acute massive PE for patients without life-limiting comorbidities.
Fig. 1. Management strategy for acute massive pulmonary embolism at Samsung Medical Center. PE: pulmonary embolism, DNR: do not resuscitate, VA ECMO: venoarterial extracorporeal membrane oxygenation.
Fig. 3. Kaplan–Meier survival curves for all-cause death.
Fig. 4. Kaplan-Meier survival curves for cardiac death.
Who should receive what therapy

- Which patients should receive what therapy:
  
  **A** 80M incidental PE hemodynamically stable with evidence of right heart strain
  
  **B** 56M new PE, hypotensive with evidence of right heart strain
  
  **C** 63M s/p R fem pop bypass with new PE, right heart strain and hypotension requiring pressers
Chronic Pulmonary Thromboembolic disease

- Without lysis, after 2 weeks, pulmonary thrombus become adherent to wall with subsequent conversion to connective/elastic tissue combined with smooth muscle cells and neutrophils.

- Even with recanalization, excessive inflammatory and fibrotic changes at small arteriolar level causing pulmonary hypertension leading to progressively increased PH.

- Increased PH, with relatively smaller levels of obstruction as compared to acute emboli.

- Incidence is hard to predict, estimated 100,000 patients that might benefit from surgical intervention.

- 5 year survival of 30% for PA pressure >40 mmHg and 10% >50mm Hg.
Presentation & Diagnosis

- Exercise intolerance (#1), hemoptysis, chest pain, large jugular venous pulse wave (large A wave)

- Diagnosis one of exclusion, normal CXR, normal pulmonary function testing. Signs of right heart strain on ECHO,

- V/Q testing is “the test” for r/o chronic PE. Mismatched segmental defects, “mottling” of perfusion scan

- Right heart cath provides important hemodynamic information

- CTA/angiography-gold standard for delineating anatomy
Pulmonary Endarterectomy

- First described in 1960 by Allison, however he did not preform a true endarterectomy.
- Technique popularized by Dr. Stuart Jamieson at UCSD. Largest single center surgical volume.
- More than 3,200 pulmonary endarterectomies at UCSD
Surgical Candidate Criteria

Reason to perform

- Hemodynamic-prevent/improve the hemodynamics of RV hypertrophy secondary to pulmonary hypertension
- Respiratory-reduce quantity of lung that is ventilated and not perfused
- Prophylactic-prevent worsening RV dysfunction, clot progression and small vascular changes in remaining healthy lung

Typical patient characteristics

- Pulmonary SVR 800-1200
- PH at supra-systemic levels
- RV dysfunction
- Patient symptoms, decreased exercise tolerance
Surgical Technique

- Median sternotomy, CPB initiated with deep cooling to 20°C and dissection of complete PA from ascending aorta
- Incision in right PA, at level of lower lobe branch, heart is then arrested and circulatory arrest initiated
- Loose Debris evacuated, microtome knife is used to develop the endarterectomy plane, which is carried through the dissection removing lobar segments as well
- 20 cm segments typically removed
- Process repeated on right side, and sites closed primarily
- PFO repaired if present, patient rewarmed, hemodynamically optimized
Intraoperative classification

- Type I disease
  - Fresh thrombus noted (main and lobar arteries)
Intraoperative classification

• Type II disease
  • Intimal thickening/fibrosis with/out organized thrombus proximal to segmental arteries
  • Thickened intima and “webs” noted in the main vessels
Intraoperative classification

- Type III disease
  - Fibrosis, intima thickening with/out organized thrombus in segmental & sub segmental segments, most embolic process resorbed
Intraoperative classification

- Type IV disease
  - Microscopic vascular disease without obvious thromboembolic disease
  - Worst prognosis
Results

• Sustained reduction in PVR, Pulmonary artery systolic and diastolic pressure, Increased CO and resolution of tricuspid valve dysfunction

• Clinical improvement in NYHA heart failure status from 4/3 to 2/1

• Echocardiographic evidence of right heart hypertrophy/dilatation and tricuspid regurgitation resolution

• Mortality of 1.5%, stroke 0.2%, bleeding requiring reoperation 2.8%, transfusion 33.9%, lung reperfusion injury in 15%

• Largest risk factor for mortality is having Type 4 disease
Future Directions

- PE response team are a new paradigm for the management of PE in the acute settings.
- Dedicated PE response team with further activation of a multi-disciplinary team to evaluate optimal treatment options.
- Expanded indications for embolectomy.
References


