Pulmonary Embolism

Carlos Baleeiro, MD

February 8, 2020
Conflict of interest

Disclosure

• I have no financial or professional conflicts regarding this presentation.
Venous Thromboembolism

Objectives

• Review options and duration of therapy for uncomplicated low risk pulmonary embolism.
• Review therapy options for massive unstable pulmonary embolism.
• Discuss controversies on the therapy for submassive pulmonary embolism.
  • Pulmonary embolism response team.
**Background**

**Significance of VTE**

- The overall incidence of pulmonary embolism (PE) is approximately 112 cases per 100,000. Deaths from PE account for at least 100,000 deaths per year in the United States.
- The pathogenesis of PE is similar to that of deep venous thrombosis. Most emboli arise from lower extremity proximal veins (iliac, femoral, and popliteal). However, they may also originate in right heart, inferior vena cava or the pelvic veins, and in the renal and upper extremity veins.
  - Henceforth we will use VTE – PE interchangeably.
  - For the purposes of this discussion, we will be focusing on thromboembolic disease only (no fat or air embolism).
- PE has a wide variety of presenting features, ranging from no symptoms to shock or sudden death. The most common presenting symptom is dyspnea followed by chest pain, cough, and symptoms of deep venous thrombosis.
Background
Significance of VTE

- PE can be classified according to hemodynamic stability (hemodynamically unstable or stable), temporal pattern of presentation (acute, subacute, or chronic), anatomic location (saddle, lobar, segmental, subsegmental), and presence or absence of symptoms (symptomatic or asymptomatic). Patients with hemodynamically unstable PE, defined as a systolic blood pressure <90 mmHg or a drop in systolic blood pressure of ≥40 mmHg from baseline for >15 minutes, should be distinguished from patients with hemodynamically stable PE because they are more likely to die from shock in the first two hours of presentation and may therefore benefit from more aggressive treatment. (Spoiler alert).
Reviewing the guidelines
Squeezing a long text in 20 minutes

Guidelines & Resources

Pulmonary Vascular

Guidelines and Expert Panel Reports
Therapy for Pulmonary Arterial Hypertension in Adults 2018: Update of the CHEST Guideline and Expert Panel Report (Published: March 2019)

Antithrombotic Therapy for Atrial Fibrillation: CHEST Guideline and Expert Panel Report (Published: November 2018)

Antithrombotic Therapy for VTE Disease (Published: February 2016)

Antithrombotic Therapy and Prevention of Thrombosis (9th Edition), Published: February 2012

This CHEST guideline series presents recommendations for the prevention, diagnosis, and treatment of thrombosis, addressing a comprehensive list of clinical conditions, including medical, surgery, orthopedic surgery, atrial fibrillation, stroke, cardiovascular disease, pregnancy, and neonates and children.

- Executive Summary: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines
- Parenteral Anticoagulants: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines
- Antiplatelet Drugs: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines
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Pulmonary Vascular

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This keeps going...
Treatment of stable PE
Going back to basics
Virchow’s Triad

Stasis of blood flow

Endothelial injury
Hypercoagulability
Going back to basics
Virchow’s Triad

- Stasis of blood flow
- Endothelial injury
- Hypercoagulability
The Coagulation Cascade
(Hold for scared reactions)
The Coagulation Cascade
What could possibly go wrong?
The Coagulation Cascade

**Therapeutic targets**

**Contact activation (intrinsic) pathway**
- Damaged surface
  - XII
  - XIIa
  - XI
  - X
  - IX
  - IXa
  - VIIIa
  - VII
  - VIIa
  - Xa

**Tissue factor (extrinsic) pathway**
- Trauma
  - Tissue factor
  - TFPI
  - Antithrombin

**Common pathway**
- X
  - Prothrombin (II)
  - Va

**Drugs**
- Warfarin
- Rivaroxaban
- Apixaban
- Dabigatran
- LMWH
- Heparin

Protein C + Thrombomodulin

Protein S
Treatment guidelines
Hemodynamically stable PE

- PE left untreated, has an overall mortality of up to 30 percent.
- When treated with anticoagulation the prognosis is excellent.
  - Rates of PE-related mortality decreased over time, with a risk-adjusted rate of 3.3% in 2001 to 2005 and 1.8% in 2010 to 2013.  
- Anticoagulant therapy is indicated for all patients with PE in whom the risk of bleeding is low.
- Initial anticoagulant therapy should be administered as soon as possible in order to quickly achieve therapeutic anticoagulation.
- Long-term anticoagulant therapy is administered beyond the initial phase of anticoagulation for a finite period of typically three months (eg, transient VTE risk factors), or up to 6 or 12 months in some cases (eg, persisting risk factors, or unprovoked VTE).
### Treatment guidelines – ACCP recommendations

#### Grades of evidence

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Treatment guidelines – ACCP recommendations

Hemodynamically stable PE – Choice of agents

- For VTE without an associated cancer diagnosis, all direct oral anticoagulants (dabigatran, rivaroxaban, apixaban, or edoxaban) are recommended over vitamin K antagonist (VKA) therapy (all Grade 2B) and VKA therapy is recommended over low molecular weight heparin (LMWH; Grade 2C).
- For VTE associated with cancer, LMWH is recommended over VKA (Grade 2B) or any direct oral anticoagulants (all Grade 2C).
- For patients with an unprovoked proximal DVT or PE who are stopping anticoagulant therapy, the guideline suggests the use of aspirin over no aspirin to prevent recurrent VTE if there are no contraindications to aspirin therapy (Grade 2B).

Treatment guidelines – ACCP recommendations
Hemodynamically stable PE – Duration of therapy

- Anticoagulants should stop after 3 months of therapy in patients with an acute, proximal deep venous thrombosis (DVT) provoked by surgery rather than shorter or longer treatment courses (Grade 1B).
- Anticoagulants should also be stopped after 3 months in patients with a proximal DVT or pulmonary embolism (PE) provoked by a nonsurgical transient risk factor over shorter or longer courses (Grade 1B for high bleeding risk patients, Grade 2B for low or moderate bleeding risk patients).
- Anticoagulation should be given for 3 months in patients with a first unprovoked VTE and a high risk of bleeding (Grade 1B), but should be extended without a scheduled stop date in patients with a low or moderate risk of bleeding (Grade 2B).

Treatment of unstable PE
Massive pulmonary embolism

Hemodynamically unstable PE

- A small percentage of patients with PE present with hemodynamic instability or shock (approximately 8 percent; ie, “massive” PE). When patients with suspected PE present with hypotension, initial support should focus upon restoring perfusion with intravenous fluid resuscitation and vasopressor support, as well as oxygenation and, if necessary, stabilizing the airway with intubation and mechanical ventilation.

- Patients who stabilize in response to initial resuscitation efforts can be treated with systemic anticoagulation with close hemodynamic monitoring.
Massive pulmonary embolism
Hemodynamically unstable PE

- In patients with PE who are hemodynamically unstable or who become unstable due to recurrence despite anticoagulation, we suggest more aggressive therapies (ie reperfusion therapies) than anticoagulation including the following:
  - Thrombolytic therapy is indicated in most patients, provided there is no contraindication.
    - tPA 100 mg IV over 2 hours.
    - For patients with an acute PE and hypotension (massive PE), the ACCP guidelines recommend the use of thrombolytic therapy (Grade 2B), preferring systemic therapy over catheter-directed thrombolytic therapy (Grade 2C).
  - Embolectomy is appropriate for those in whom thrombolysis is either contraindicated or unsuccessful (surgical or catheter-based).
**Massive pulmonary embolism**

**Hemodynamically unstable PE - Embolectomy options**

- Catheter-directed modalities may be performed with lower total dose/burden of thrombolytics:
  - Ultrasound-assisted thrombolysis
  - Rheolytic embolectomy (pressurized saline)
  - Rotational embolectomy
  - Suction embolectomy
- Surgical embolectomy:
  - The usual indication for surgical embolectomy is hemodynamic instability due to acute PE for patients in whom thrombolysis (systemic or catheter-directed) is contraindicated, and is an option in those in whom thrombolysis has failed.
  - Mortality is not higher with surgical embolectomy.
Massive pulmonary embolism
Hemodynamically unstable PE- Catheter embolectomy

- US-assisted thrombolysis allows for a lower total dose of tPA.
- Good efficacy and low morbidity.
  - Improved RV function.
  - Increased risk of bleeding compared to anticoagulation alone.

https://www.bostonscientific.com/
### Contra-indications to thrombolytics

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<td><strong>Patient history</strong></td>
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<td>Ischemic stroke or severe head trauma in the previous three months</td>
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<tr>
<td>Previous intracranial hemorrhage</td>
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<tr>
<td>Intra-axial intracranial neoplasm</td>
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<tr>
<td>Gastrointestinal malignancy or hemorrhage in the previous 21 days</td>
</tr>
<tr>
<td>Intracranial or intraspinal surgery within the prior three months</td>
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<tr>
<td><strong>Clinical</strong></td>
</tr>
<tr>
<td>Symptoms suggestive of subarachnoid hemorrhage</td>
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<tr>
<td>Persistent blood pressure elevation (systolic ≥185 mmHg or diastolic ≥110 mmHg)</td>
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<tr>
<td>Active internal bleeding</td>
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<tr>
<td>Presentation consistent with infective endocarditis</td>
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<tr>
<td>Stroke known or suspected to be associated with aortic arch dissection</td>
</tr>
<tr>
<td>Acute bleeding diathesis, including but not limited to conditions defined under 'Hematologic'</td>
</tr>
<tr>
<td><strong>Hematologic</strong></td>
</tr>
<tr>
<td>Platelet count &lt;100,000/mm³</td>
</tr>
<tr>
<td>Current anticoagulant use with an INR &gt;1.7 or PT &gt;15 seconds or aPTT &gt;40 seconds or PT &gt;15 seconds</td>
</tr>
<tr>
<td>Therapeutic doses of low molecular weight heparin received within 24 hours (eg, to treat VTE and ACS); this exclusion does not apply to prophylactic doses (eg, to prevent VTE)</td>
</tr>
<tr>
<td>Current use of a direct thrombin inhibitor or direct factor Xa inhibitor with evidence of anticoagulant effect by laboratory tests such as aPTT, INR, ECT, TT, or appropriate factor Xa activity assays</td>
</tr>
<tr>
<td><strong>Head CT</strong></td>
</tr>
<tr>
<td>Evidence of hemorrhage</td>
</tr>
<tr>
<td>Extensive regions of obvious hypodensity consistent with irreversible injury</td>
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</table>
Intermediate risk PE
Submassive PE
How big is too big?

- The management of intermediate risk patients is less clear. Patients with high clot burden but initial hemodynamic stability, stable patients with RV dysfunction and high BNP have a higher risk of complications and chronic thromboembolic complications than low risk patients.

- Balancing risk/benefit:
  - Systemic thrombolytic therapy increases the risk of major bleeding: 9.2% versus 3.4% compared to anticoagulation alone (with 1.5% vs 0.2% ICH).
    - Chatterjee et al JAMA. 2014 Jun;311(23):2414-21
  - Catheter-directed embolectomy with fibrinolysis has a lower risk of severe bleeding than systemic tPA but still a higher risk than anticoagulation alone.
# Submassive PE

*Is the risk of therapy worth it?*

## Table 3. Efficacy Outcomes. *

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Tenecteplase (N = 506)</th>
<th>Placebo (N = 499)</th>
<th>Odds Ratio (95% CI)</th>
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</tr>
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<tbody>
<tr>
<td>Primary outcome — no. (%)</td>
<td>13 (2.6)</td>
<td>28 (5.6)</td>
<td>0.44 (0.23–0.87)</td>
<td>0.02</td>
</tr>
<tr>
<td>Death from any cause</td>
<td>6 (1.2)</td>
<td>9 (1.8)</td>
<td>0.65 (0.23–1.85)</td>
<td>0.42</td>
</tr>
<tr>
<td>Hemodynamic decompensation</td>
<td>8 (1.6)</td>
<td>25 (5.0)</td>
<td>0.30 (0.14–0.68)</td>
<td>0.002</td>
</tr>
<tr>
<td>Time between randomization and primary efficacy outcome — days</td>
<td>1.54±1.71</td>
<td>1.79±1.60</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Recurrent pulmonary embolism between randomization and day 7 — no. (%)</td>
<td>1 (0.2)</td>
<td>5 (1.0)</td>
<td>0.20 (0.02–1.68)</td>
<td>0.12</td>
</tr>
<tr>
<td>Fatal</td>
<td>0</td>
<td>3 (0.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonfatal</td>
<td>1 (0.2)</td>
<td>2 (0.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other in-hospital complications and procedures — no. (%)</td>
<td></td>
<td></td>
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<td>Mechanical ventilation</td>
<td>8 (1.6)</td>
<td>15 (3.0)</td>
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<td>Surgical embolectomy</td>
<td>1 (0.2)</td>
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<td>Catheter thrombus fragmentation</td>
<td>1 (0.2)</td>
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<td>Vena cava interruption</td>
<td>5 (1.0)</td>
<td>1 (0.2)</td>
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<td>Thrombolytic treatment other than study medication</td>
<td>4 (0.8)</td>
<td>23 (4.6)</td>
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<tr>
<td>Death from any cause between randomization and day 30 — no. (%)</td>
<td>12 (2.4)</td>
<td>16 (3.2)</td>
<td>0.73 (0.34–1.57)</td>
<td>0.42</td>
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<tr>
<td>Patient still hospitalized at day 30 — no. (%)</td>
<td>59 (11.7)</td>
<td>50 (10.0)</td>
<td></td>
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<tr>
<td>Rehospitalization between randomization and day 30 — no. (%)</td>
<td>22 (4.4)</td>
<td>15 (3.0)</td>
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* Plus–minus values are means ±SD. Odds ratios and P values are provided for efficacy outcomes that were prespecified in the trial protocol.*

## Submassive PE

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| Nonfatal                                                               | 1 (0.2)                | 2 (0.4)           |
| Other in-hospital complications and procedures — no. (%)               |                        |                   |
| Mechanical ventilation                                                 | 8 (1.6)                | 15 (3.0)          |
| Surgical embolectomy                                                   | 1 (0.2)                | 2 (0.4)           |
| Catheter thrombus fragmentation                                         | 1 (0.2)                | 0 (0.0)           |
| Vena cava interruption                                                 | 5 (1.0)                | 1 (0.2)           |
| Thrombolytic treatment other than study medication                     | 4 (0.8)                | 23 (4.6)          |
| Death from any cause between randomization and day 30 — no. (%)        | 12 (2.4)               | 16 (3.2)          | 0.73 (0.34–1.57)        | 0.42    |
| Patient still hospitalized at day 30 — no. (%)                         | 59 (11.7)              | 50 (10.0)         |
| Rehospitalization between randomization and day 30 — no. (%)           | 22 (4.4)               | 15 (3.0)          |

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Some of the PEITHO centers followed pts out to two years and saw no benefit of thrombolysis. Incidence of CTEPH was fortunately low in both groups.

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Diagnosis of Pulmonary Embolism

Risk Stratify:
- PESI score (see next page)
- BNP
- Troponin
- EKG

Administer Alteplase 100mg over 2 hours

Start IV Heparin with a bolus if no contra-indication

Persistent hypotension or hypoxemia?

Massive PE
- Alert Critical Care Medicine
- Contraindications to Systemic Thrombolysis?

Yes
- Low Risk - Score < 86
  - Troponin 0.4 or lower
  - (mortality <20%)
  - Continue anticoagulation.
  - Stat/urgent Echocardiogram.
  - Lower extremity Doppler ultrasound.
  - Admit to hospitalist service.

No
- High Risk – Score > 85
  - Troponin >0.4, BNP> 90
  - (mortality >20%)
  - Continue anticoagulation.
  - Stat/urgent Echocardiogram.
  - Lower extremity Doppler ultrasound.
  - Admit to critical care

Intermediate Risk
- Score 86 - 105
  - Continue anticoagulation.
  - Contact critical care and hospitalist services.

High Risk
- Score > 105
  - Consider catheter-guided therapy.
  - Admit to critical care
Summary
The different risk groups

• Stable PE:
  • Oral anticoagulation.
  • NOACs preferred for non-cancer related PE’s.
  • 3 months vs. longer courses depending on reversible risk factors.
  • IVC filters have a higher rate of DVTs and no mortality benefit
    - JACC 2014 63 (16): 1675-1683

• Unstable PE:
  • Systemic thrombolysis, catheter embolectomy or surgical embolectomy.

• Submassive PE:
  • Stratify risk.
  • Multi-disciplinary approach (PE Response Team) may help.
  • Monitoring on anticoagulation and waiting to decide on tPA/catheter does not increase morbidity or mortality.
Thank you