Pulmonary ThromboEmbolism

R. Pourbahador MD
Interventional Cardiologist
• The clinical picture of Pulmonary Embolism is variable & most patients suffering from acute pulmonary embolism present with one of three different clinical syndromes:
  1. Pulmonary Infarction
  2. Acute Unexplained Dyspnea
  3. Acute Corpulmonale
Pulmonary Infarction:

• A submassive embolism that completely occludes a distal branch of the pulmonary circulation.
• Pleuritic chest pain, Hemoptysis
• Rales
• Abnormal findings on CXR
Acute Unexplained Dyspnea:

• Submassive pulmonary embolism without pulmonary infarction.
• CXR is usually normal.
• ECG is usually normal.
• Pulse oxygen saturation is often depressed.
Acute Corpulmonale:

- Caused by the complete obstruction of 60 to 75% of pulmonary circulation.
- Experience shock, syncope, sudden death
- Syncope occurs in approximately 10% of patients with APE & is commonly ascribed to a massive, hemodynamically unstable APE.
• Pulmonary Emboli are potentially life threatening occurrences associated with significant morbidity & mortality.

• There are a variety of diagnostic tools that maximize our ability to detect PE & enable better prognostication.

• RV dysfunction & the release of cardiac biomarkers are associated with more adverse events.

• Patients treated with thrombolytic therapy show rapid improvement of RV function & pulmonary perfusion which may lead to a lower rate of early recurrent PE & a decrease the late sequela of chronic pulmonary hypertension.
MASSIVE PULMONARY EMBOLISM
CASE PRESENTATION
• 35 years old lady teacher
• Living with partner
• Type I DM – on Insulin, Non smoker
• OCP – 6 years
• Suddenly collapsed on doorway while preparing to leave for school.
• Possible LOC, No head injury. Partner called for the ambulance.
IN ER

• Denied any chest pain or palpitation
• No history of leg pain or swelling

O/E
Conscious, oriented
Tachypnoec R/R 36/mt  SpO2 – 84% RA
HR 128/mt SR  BP 94/56 mmHG
• Legs – No signs of DVT
• CVS- Normal heart sounds, No rub, possible systolic murmur left para sternal area
• Chest- Lungs – possible decreased air entry left lung but otherwise clear.
• Abdomen and Neuro - Unremarkable
ECHO REPORT

- Grossly dilated RV
- Severely hypo kinetic RV free wall
- RV apex contracts well
- PASP 55 mmHg
- Rest normal
CAUSES OF RV ENLARGEMENT

a. Tricuspid valve disease
b. Severe Pulmonary Regurgitation
c. ASD
d. Pulmonary HPN – Primary and Secondary
e. RV Infarction
f. Arrhythmogenic RV Dysplasia (Cardiomyopathy)
Case no. 2:

**Case presentation**

- 52 year-old man; morbidly obese (BMI: 40 kg/m²)
- Presented at ED: increasing dyspnoea over 6 weeks, now almost at rest; substernal chest discomfort
- OSA on CPAP treatment (compliant)
- COPD: ratio of forced expiratory volume in 1 second (FEV₁) to forced vital capacity (FVC), 0.59; FEV₁, 64% of the predicted value. No previous admissions because of AECOPD
- No hypertension. No history of CAD
- History of unprovoked intermediate-risk PE four months ago; on warfarin therapy
Case presentation

Clinical findings at presentation:

- BP: 110/70 mmHg; HR: 116/min; resp. rate: 28/min; SO₂: 88% on room air, increasing to 96% with supplemental oxygen (2 liters)
- Heart examination: no murmurs
- Lung examination: breath sounds diminished
- Jugular venous distention difficult to assess (due to obesity)
- Chronic venous insufficiency of lower extremities present
Case presentation (cont’d)

Laboratory

• Normal blood count
• Creatinine: 0.9 mg/dl
• GPT: 56 U/L; GOT: 123 U/L
• hsTnT: 0 pg/mL; BNP: 180 pg/mL
• INR: 2.7

Blood gas analysis

• pH: 7.45; $\text{PaCO}_2$: 32 mm Hg; $\text{PaO}_2$: 55 mm Hg; lactate normal
Case presentation (cont’d)
Case presentation (cont’d)
Qu. 1 Which is the most likely diagnosis?

1. COPD exacerbation
2. Congestive heart failure
3. Recurrent PE
4. Pulmonary hypertension
5. Panic attack
Respiratory physician

Does this patient have a COPD exacerbation?
Respiratory physician

Does this patient have COPD exacerbation?

- No cough or sputum purulence
- Mild COPD
- No history of exacerbations
- Discrepancy between COPD stage and pulmonary arterial enlargement on chest X-ray
Cardiologist

Does this patient have recurrent PE?
Early recurrence

- Poor quality of anticoagulation (failure to achieve therapeutic aPTT and INR)
- Cancer

aPTT, activated partial thromboplastin time; BMI, body mass index; INR, international normalized ratio.
Case: CT pulmonary angiography
Heart Team

Does this patient have chronic thromboembolic pulmonary hypertension?
Case: echocardiography
Case: V/Q scan

Q scan

V scan
Case: Pulmonary angiogram
Case presentation (cont’d)

Hemodynamic data on right heart catheterization

• RAP: 26 mmHg
• PAP: 90/35 mmHg (mean, 57 mmHg)
• PAWP: 16 mmHg
• Cardiac output: 2.5 L/min
• PVR: 1294 dyn·sec/cm$^5$
• Pulmonary artery saturation: 43%
Heart Team

Final diagnosis

Chronic thromboembolic pulmonary hypertension
Chronic obstructive pulmonary disease (mild)
Obstructive sleep apnea
Case no. 3:

History

• A 61 year old male
  – New onset shortness of breath for 4 days.
  – He also noticed a cough, with blood tinged sputum.
  – He had no chest pain, no orthopnea or paroxysmal nocturnal dyspnea, or fever.
  – He used his albuterol inhaler several times with no improvement in his shortness of breath
• Past history:
  – Hypertension
  – Asthma
  – Remote history (10 years prior to current presentation) of left lower limb swelling that subsided after treatment for 6 months.

• Social history: Smoker 40 pack year history

• Family history: no relevant family history.

• Drug history:
  – fluticasone/Salmeterol combination inhaler twice daily,
  – albuterol inhaler as needed for shortness of breath
  – hydrochlorothiazide 12.5mg once daily
Physical Examination:

- Vital Signs:
  - HR 113/min,
  - BP 150/93,
  - respiratory rate 22/min,
  - oxygen saturation via pulse oximetry 89% on room air,
  - temperature 97.2° F (36.2 °C)
• HEENT: (Head, Eyes, Ear, Nose, Throat examination) was normal.
• JVP was not raised.
• Chest clear no wheezes, or crackles.
• Abdomen soft non tender, no palpable liver, spleen.
• Lower extremities: no edema, no other abnormalities
• PEFR (peak expiratory flow rate) was above 75% of predicted
What is your differential diagnosis? What test would you like to perform next?
Investigations

- Chest X ray → Clear Lung fields
- ECG → Sinus Tachycardia
- Basic metabolic panel (BMP) includes Na, K, HCO₃, Chloride, BUN, Creatinine was within normal limits.
- Complete blood count: Hb 15.9, WBC 7200 with normal differential, platelet count 270.
- Liver function test, and liver enzymes were within normal limits.
Arterial Blood Gas on Room Air

- pH 7.42 (normal)
- pCO2 33.2 (mildly reduced)
- pO2 55 (moderately reduced)
- Oxsat 87%
What would you like to do next?
Large thrombus
Symptoms

- Dyspnea at rest or with exertion (73%)
- Pleuritic pain (44%),
- calf or thigh pain (44%),
- calf or thigh swelling (41%),
- cough (34%),
- >2-pillow orthopnea (28%),
- wheezing (21%)

PIOPED II. Stein PD, Beemath A et al
Signs

- tachypnea (54%),
- tachycardia (24%),
- crackles (18%),
- decreased breath sounds (17%),
- Loud S2 (15%),
- Raised JVP (14%)

PIOPED II. Stein PD, Beemath A et al
D dimer

- Fibrin degradation product
- Is elevated in most patients with PE
- High negative predictive value
- i.e. useful to rule out PE in patients with low to intermediate pretest probability
ECG

- Sinus tachycardia
- Non specific ST/T changes
- Classical findings are uncommon
  - S1Q3T3 pattern,
  - RV strain,
  - new incomplete RBBB
Chest Xray

- Usually abnormal but doesn’t differentiate PE from other diagnoses

<table>
<thead>
<tr>
<th></th>
<th>PE</th>
<th>No PE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atelectasis or a pulmonary parenchymal abnormality</td>
<td>69%</td>
<td>58%</td>
</tr>
<tr>
<td>Pleural Effusion</td>
<td>47%</td>
<td>39%</td>
</tr>
<tr>
<td>Normal</td>
<td>12%</td>
<td></td>
</tr>
</tbody>
</table>

Stein et al
Chest. 1991;100(3):598
VQ Scan

• Interpreted as probability i.e.
  – Low Probability
  – Intermediate Probability
  – High Probability

• Diagnosis of PE using VQ scan requires integration of the pretest probability

• Normal VQ Scan virtually excludes PE
Management

- Anticoagulation (Acute)
  - Unfractionated Heparin
  - Low Molecular Weight Heparin
  - Fondaparinux
- Anticoagulation (Chronic)
  - Warfarin
- New agents
  - Dabigatran
  - Rivaroxaban
Thrombolysis

- Indications
  - Massive PE (SBP <90 for >15mins)
- Controversial Indication
  - Severe hypoxemia
  - Large thrombus burden
  - RV dysfunction
    - ECG, Cardiac Enzyme Elevation, Echocardiography
  - RV thrombus in transit
  - Saddle Embolus
Duration of anticoagulation

- **First Episode**
  - Reversible $\rightarrow$ 3 months
  - Unprovoked $\rightarrow$ indefinite (if bleeding risk acceptable)

- **Recurrent PE**
  - Indefinite if risk of bleeding acceptable
Warfarin

- Started after administration of heparin (or heparin like agent)
- Adjusted dose to INR 2.0-3.0
Rivaroxaban

- Given as 15mg PO dose BID for 3 weeks then 20mg daily
- Doesn’t require monitoring
- Good safety profile (less bleeding than with warfarin)
Inferior Vena Cava Filter

- “Filter out” large emboli from the pelvis, lower extremities
- Inserted percutaneously
- Indicated for patients who have contraindications to anticoagulation
Case no. 4:

Case presentation

A 26-year-old Caucasian man with no history of disease was admitted to Gazi University Emergency Department after he had a syncopal episode in his home. The patient was in his usual good state of health until he suddenly collapsed while standing and lost consciousness for approximately five minutes. He recovered spontaneously but was extremely weak and dyspneic. He was also diaphoretic and tachypneic, but denied any associated chest pain or palpitations. No tonic-clonic activity was witnessed, and he experienced no incontinence.

The patient was a computer programmer and he had been working 18 hours a day without rest periods for a month. On admission, physical examination revealed a diaphoretic and dyspneic patient without focal neurologic findings. His heart rate was regular but tachycardic at 128 beats/minute, his blood pressure was 126/72 mmHg without orthostatic changes, and his respiratory rate was 32 breaths/minute. The room air oxygen saturation was 90%, and arterial blood gas analysis in room air revealed hypoxemia ($PO_2 = 58$ mmHg) with an elevated alveolo-arterial oxygen gradient ($A-a O_2$ gradient). Examination of his head and neck was normal. The results of chest wall examination revealed reduced breath sounds bilaterally at the lung bases. The findings of heart and abdominal examinations were unremarkable, but on examination of his legs, deep venous thrombosis (DVT) was noted in his left leg, with a positive Homans' sign in the left leg and the left calf measured 3 cm more than the right one.
Syncope, in contrast to pulmonary embolism, is relatively easy to detect, but can be a difficult symptom from which to determine the etiology. In as many as 50% of patients with syncope, no specific cause is found despite extensive evaluation. Syncope has been classified as cardiovascular (reflex and cardiac syncope), noncardiovascular (including neurologic and metabolic disorders) and unexplained [2, 6]. It occurs in approximately 10% of patients with acute pulmonary embolism and is commonly ascribed to a massive, hemodynamically unstable acute pulmonary embolism. Although the prognostic value of syncope has not been specifically addressed, it has generally been considered a poor indicator in diagnosing pulmonary embolism [7].

Syncope in the setting of pulmonary embolism can be the result of three possible mechanisms. First, greater than 50% occlusion of the pulmonary vascular tree causes right ventricular failure and impaired left ventricular filling, leading to a reduction in cardiac output, arterial hypotension, reduced cerebral blood flow, and ultimately syncope. The second mechanism of syncope associated with pulmonary embolism is the appearance of arrhythmias associated with right ventricular overload. In the third mechanism, the embolism can trigger a vasovagal reflex that leads to neurogenic syncope. However, the contribution of hypoxemia secondary to ventilation or perfusion abnormalities must also be considered and may play an important role in the development of syncope. Moreover, acute pulmonary hypertension may also lead to right-to-left flow across a patent foramen ovale, and thus exacerbate hypoxemia [8, 9].
The clinician should seek the following clues to the diagnosis of pulmonary embolism in patients who have had a syncopal episode: (a) hypotension and tachycardia or transient bradyarrhythmia; (b) acute cor pulmonale according to electrocardiogram criteria or physical examination; and (c) other signs and symptoms indicative of pulmonary embolism. The presence of any of these findings without other obvious causes of syncope should lead to further work-up, including arterial blood gas analysis, ventilation-perfusion scanning, lower extremity duplex sonogram, echocardiography, multislice computed tomography and angiography, if necessary. Although oxygen saturation levels are inadequate for screening purposes, respiratory alkalosis with hypoxia and increased A-a O₂ gradient are typically seen. However, results of blood gas analysis are normal in 10% of cases [4, 10].

In our case, the patient presented to the emergency department with complaints of dyspnea, tachypnea and tachycardia, following a syncopal episode. He had experienced immobilization for one month, hypoxemia in room air, and DVT according to the ultrasonographic results. PTE was initially considered and all of the diagnostic procedures were carried out to prove this presumptive diagnosis. Because DVT and PTE developed in this young patient with no history of any underlying diseases or disorders, he was referred for thrombophilia panel testing (including protein C or S deficiency and Factor V mutation) before treatment; however, as his long-term follow-up was performed by the Department of Pulmonary Diseases, we do not have any further detailed results from these examinations. This case is interesting because the patient did not experience a massive embolism but did develop syncope.