

**ACUTE PUMONARY EMBOLISM:
UPDATED GUIDELINES**

**MASSIVE vs. SUBMASSIVE
DIAGNOSIS & TREATMENT
NURSING IMPLICATIONS**

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Epidemiology of PE

- VTE kills more people annually than Breast Cancer^{1,2}
- Annual incidence > 500,000
- DEATHS: 60,000 to ~ 200,000 per year¹
- 11% die suddenly
- 67% not diagnosed while alive
- Undiagnosed PE has a mortality of 30%
- Diagnosed PE has mortality < 12%
- PE: 1 yr mortality of 39% in the elderly³
- DVT: 1 yr mortality of 21% in the elderly³

1. ACS Breast cancer facts and figures 2001
2. Anderson, et alia, Arch Int Med 1991
3. Kallifa WD et alia, Arch Int Med 1994; 154(8): 861-6

Epidemiology of PE

**Pulmonary embolism may be the #1
preventable cause of death in
hospitalized patients**

1. Claggett, et alia, Chest 1995; 108:312S -334S

Which one of you in here, has had a PE?

- Lungs are natural filters
- Anything on route to the left heart from the right heart will be filtered by the lungs
- We throw showers of microemboli every day
- The lungs are highly fibrinolytic – they lyse clots all the time

Sources of Emboli

Primary source:

> 90% from deep vein thrombosis (DVT) in the legs

- Sometimes the pelvic veins
- Occasionally the upper extremity veins

Non-blood clot emboli

- Air
- Tumor
- Amniotic fluid
- Fat – common after long bone Fx especially in the elderly;
 - change in LOC, thrombocytopenia, chest & neck petechiae

Predisposing Risk Factors for Clot Formation

- Virchow's Triad
 - Venous stasis
 - Hypercoagulability
 - Vessel wall injury
- Virchow is famous for elucidating the mechanism of pulmonary thromboembolism, and coining the term embolism



Predisposing Risk Factors for Clot Formation:

Stasis

- Immobility, bed rest
- Obesity
- Varicose veins
- Anesthesia
- Congestive heart failure/cor pulmonale
- Prior venous thromboembolism
- Central venous catheters - 1 in 10 cases of PE
- Pregnancy
- Air travel > 4 hours

Predisposing Risk Factors for Clot Formation:

Hypercoagulability

Congenital;

- Factor V Leiden mutation
 - most common hereditary blood coagulation disorder in the United States
 - present in 5% of Caucasians, 1.2% of African Americans
- Prothrombin G20210A mutation; 2nd most common
- Protein C & Protein S deficiency
- Antithrombin III deficiency
- Dysfibrinogenemia
- Homocysteinuria

Predisposing Risk Factors for Clot Formation:

Hypercoagulability

Acquired;

- Estrogen use, Birth control pills
- Hormonal changes of pregnancy
- Malignancy
- Thrombocytosis; rare chronic disorder characterized by the overproduction of platelets in the bone marrow
- Disseminated intravascular coagulation (DIC)
- Heparin induced thrombocytopenia (HIT)
- Antiphospholipid syndrome
- Nephrotic syndrome
- Paroxysmal nocturnal hemoglobinuria (PNH)

PE CATEGORIES

- **MASSIVE**
 - Enough clot obstruction of the PA blood flow to cause \uparrow RV afterload, then Pulm HTN
 - May cause pulmonary infarction
 - “SADDLE EMBOLUS”
- **SUBMASSIVE**
 - Embolism to one or more pulmonary segments without \uparrow in RV & PA pressures
 - Risk of chronic Pulm HTN



Figure 10-10 Pulmonary embolism. The saddle embolus, with its characteristic wedge-shaped opacity, is seen in the lower lung fields. (Revised, Adams CD, 2009). Pulmonary Embolism, p. 265. Philadelphia: Elsevier Health Sciences.

Acute PE: SIGNS & SYMPTOMS

	Massive	Submassive
Dyspnea	85%	82%
Pleuritic pain	64	85
Cough	53	52
Hemoptysis	23	40
Tachypnea	95	87
Tachycardia	48	38
Loud P2	58	45
Crackles	57	60
Phlebitis	36	26

HEMODYNAMIC EFFECTS OF PE

- RA, RV & PA pressures rise
- RV may dilate and may fail “cor pulmonale”
- Acute tricuspid regurgitation
- LV may wonder “where’s the blood?”
- Systemic hypotension may occur
- Compensatory mechanisms kick in:
 - Sympathoadrenal response, R-A-A-A Axis
 - TACHYCARDIA early, BRADYCARDIA late/ominous
 - A little more volume couldn’t hurt!

BLOOD GASES EFFECTS OF PE

- Impaired gas exchange
- ↑ alveolar dead space in clotted lung segments
- V_A / Q_C mismatching
 - R ♥ to L ♥ intrapulmonic shunting in massive PE
- Compensatory mechanisms kick in:
 - Tachypnea with alveolar hyperventilation
 - Serotonin, endothelin – raise PVR
 - Sympathoadrenal response, R-A-A-A Axis
 - A little more volume couldn't hurt!

BLOOD GASES EFFECTS OF PE

- Key ABG abnormalities:
 - HYPOXEMIA ↓ PaO₂
 - HYPOCAPNIA ↓ PaCO₂
 - WIDENED A-a Gradient
- (A – a DO₂ = Alveolar – arterial Oxygen difference
N: 10 -20 mmHg)

CATEGORIES OF SHOCK

- OBSTRUCTIVE SHOCK:
 - MASSIVE PULMONARY EMBOLISM;
 - RV outflow tract obstructed by clot → cor pulmonale,
 - LV “sees” ↓ volume, ↑ NV, blue from nipple line up,
 - death due to RHF

Who is Dan Blocker?

ACUTE PE: Diagnosis

- Clinical signs & symptoms
- Lab data (enzymes, Troponin I, D-dimer), AGBs
- EKG; RV strain, T wave inversion
- Chest X-Ray
- VQ scan
- Pulmonary angiography / digital subtraction angio
- Helical CT scan (spiral)
- MRI
- Intravascular ultrasound

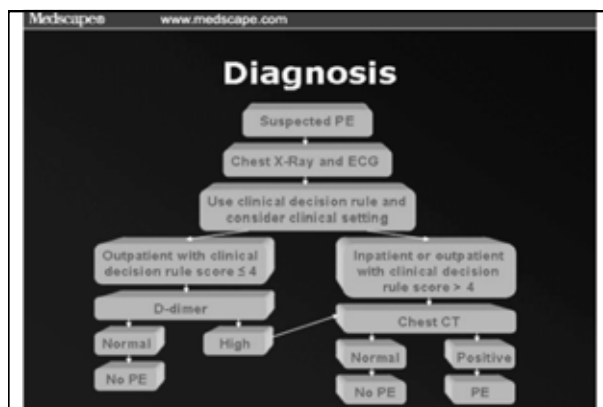
Medscape www.medscape.com

Diagnosis

Clinical Decision Rule for Evaluation of Patients with Suspected PE

Variable	Points
Clinical symptoms and signs of DVT	3.0
Alternative diagnosis less likely than PE	3.0
Heart rate greater than 100 beats per minute	1.5
Recent immobilization or surgery	1.5
Previous venous thromboembolism	1.5
Hemoptysis	1.0
Malignancy	1.0

"PE unlikely" ≤ 4 points
 "PE likely" > 4 points



ACUTE PE: Treatment

- The best treatment for PE is NOT to get one!
 - Prophylaxis
 - Early mobilization
 - Elastic stockings
 - Pneumatic compression devices
 - Heparin
 - LMWH – Enoxaparin
 - Warfarin

Rationale and Basis of Prophylaxis *At-Risk Hospitalized Patients*

- According to the Seventh ACCP Conference on Antithrombotic Therapy, the rationale of prophylaxis is based on:
 - High prevalence of VTE among hospitalized patients
 - Clinically silent nature of the disease in the majority of patients
 - Morbidity
 - Costs
 - Mortality
 - Unreliable clinical diagnosis



Geerts WH, et al. Chest 2004;126:338S-400S.

Compliance with Chest Consensus Guidelines

	Moderate Risk (2 risk factors)	High Risk (3-4 risk factors)	Highest Risk (5 or more risk factors)
Total (2002)	9/157 (6%)	43/157 (27%)	105/157 (67%)
Prophylaxis guidelines followed	7/9 (78%)	28/43 (65%)	32/105 (30%)
Prophylaxis guidelines not followed	2/9 (22%)	15/43 (35%)	73/105 (70%)

Geerts WH, et al. Chest 2004;126:338S-400S.

Why is Prophylaxis Underused in At-Risk Patients?
Potential Reasons

- Clinicians unaware of the level of VTE risk
- Not a 'one specialty' responsibility
- Heterogeneous population
- Perceived difficulties in risk assessment
- Perceived bleeding risk
- Perceived fall risk / morbidity
- Admission diagnosis is focus
- Few studies of prophylaxis

APE Treatment: Thrombolysis, Interventional Radiology, and Surgical Interventions

- Thrombolysis;
 - t-PA
 - Streptokinase
 - Urokinase
- Radiological Interventions;
 - Clot disruption with catheters
 - Transvenous catheter embolectomy
- Surgical procedures;
 - Pulmonary embolectomy
 - Thromboendarterectomy

Diagnosis-Summary

- History and physical examination
- Then, one, two, three approach to diagnosis or exclusion of PE:
 1. Clinical decision score
 2. The D-dimer test
 3. Chest CT

The Joint Commission Benchmark states:

- “DVT is one of the most common preventable causes of deaths in hospitals”
- “Only 30% to 40% of patients who should be treated to prevent DVT actually receive such treatment”
- “The use of proven and effective DVT prevention methods could save the lives of many patients”

SUMMARY / NURSING IMPLICATIONS

- NURSES MUST:
 - Identify high risk patients
 - Plan for prophylaxis
 - If none ordered, SUGGEST THE NEED FOR
PROPHYLAXIS to the primary
 - Reference the CHEST Guidelines on prophylaxis
Geerts WH, et al. Chest 2004;126:338S-400S.
 - Treat your patient with as much prophylaxis as your hospital protocols may allow
 - Change / add / modify existing protocols to reflect CHEST Guidelines



Your time on Earth has been extended – go thank your Nurse and your Doctor!
