Setting The Stage: The Emergency Physician Algorithm For Acute PE Management

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Objectives
Location of treatment
PE classification - prognosis
Risk stratification - CHALLENGES

Four Principles – to optimal ED patient care

1. Harness the power of REASSURANCE
2. Embrace UNCERTAINTY
3. Frame your decisions around your patient’s HEALTH STATE
4. Have a definition of what constitutes a GOOD SHIFT

Case 2
58 year old male
CC: shortness of breath
History
• Achilles tendon repair 3 wk ago
• Grad onset dyspnea – now severe
• Near syncope
• Vague chest “fullness”
Exam
• 98.6 RR=32 P=111 BP 108/48 sat 92%
• Clear lungs, mild respiratory distress
• Unilateral leg edema

ADDITIONAL TESTING and risk stratification?
CXR: clear
ECG: T wave inv V1-V3, incomp RBBB

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Troponin I: 0.4
BNP: 200
CT:
Case 2

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- Vague chest "fullness"  
- Exam  
  - 98.6 RR=32 P=111 BP 108/48 sat 92%  
  - Clear lungs, mild respiratory distress  
  - Unilateral/lag edema

ADDITIONAL TESTING and risk stratification?  
- CXR: clear  
- ECG: T wave inv V1-V3, incomp RBBB  
- Troponin I: 0.4  
- BNP: 200  
- CT: large central clot ft main PA, additional clot L lower lobar pulm artery

What is the spectrum of disease?  
And how do we classify this PE......?  
How does this impact our decision making....?
High Risk:
Acute PE with shock or hypotension
SBP <90 mmHg, or SBP drop by >40 mmHg, for at least 15 min, not due to a cause other than PE

Intermediate High Risk:
Acute PE without hypotension and elevated PESI score with RV dysfunction
AND
Serology Pos

Intermediate Low Risk:
Acute PE without hypotension and elevated PESI score with or without RV dysfunction
OR
Serology Pos

Low Risk:
Acute PE with low PESI score

Massive:
Acute PE with sustained hypotension
SBP <90 mmHg for at least 15 min or requiring inotropic support, not due to a cause other than PE (arrythmias, hypovolemia, sepsis, LV dysfunction, pulselessness, profound bradycardia with shock)

Submassive:
Acute PE without systemic hypotension (SBP >90 mmHg) and
RV dysfunction
RV/LV ratio >0.9, RV dysfunction on echo, RV dilation on CT scan, Elevated BNP (90), NtproBNP (500), EKG evidence RV strain
Myocardial necrosis
Elevated TNI >0.4 ng/mL, TNT >0.1 ng/mL

Non high risk
Acute PE without clinical markers of adverse prognosis

What risk prediction tools exist?
And what outcomes do they predict…..

What is missing from PESI and PESI?

- Amount of clot has not been shown to be an independent predictor of adverse outcomes
- We have all seen:
  - Young patients with good cardiopulmonary reserve tolerate significant clot on CT yet look surprisingly well on vitals, biomarkers etc.
  - Older patients or those with poor cardiopulmonary reserve with unimpressive clot burden on CT who look surprisingly poor…..

- Optimal risk stratification includes:
  - RV strain on CT and or echo
  - Biomarkers/Troponin and less degree BNP
  - Hemodynamics
  - Work of breathing and oxygenation

Case 2
58 year old male
CC: shortness of breath

History
- Achilles tendon repair 3 wk ago
- Grad onset dyspnea – now severe
- Nearsyncope
- Long chest “tunnellness” Exam
  - 98.6 F, 12 P=111 BP 108/48 sat 92%
  - Clear lungs, mild respiratory distress
  - Unilateral leg edema

ADDITIONAL TESTING and risk stratification?
CXR: clear
ECG: T wave inv V1-V3, incompl RBBB
Troponin I: 0.4
BNP: 200
CT: large bila central dot R and L main PA
US: RV dilatation hypokinesis, septal shift

TIME TO DECIDE!!!!!!!
What do YOU want to do?
Treatment options: Submassive PE (AHA classification) or High-intermediate risk PE (ESC criteria)

- Heparin-based Anticoagulation?
  - What agent? (low molecular weight heparin vs unfractionated heparin)
- Systemic Fibrinolytics?
  - What agent, what dose, what risk, what goal?
- Catheter-based tPA?
  - Who
  - When
  - How

Or High-Intermediate risk PE (ESC criteria)

Back to our patient and options?

- Heparin-based Anticoagulation?
  - What agent
- Systemic Fibrinolytics?
  - What agent, what dose
- Catheter-based treatment? (more questions than answers)
  - Who (which patients? Which doctors?)
  - When (what stage of care? And how long?)
  - How

Catheter-Directed techniques

- Infusion of lytics via dispersion catheters
- Ultrasound-assisted lytic infusion
- Mechanical thrombectomy
Patient outcome
2 years later
• Still seen in PE clinic
• Asymptomatic and exercising regularly
• Normal RV function on echo
• Was it the catheter based treatment?
• Or just being on anticoagulation alone?
• No way to know in this single case….need an RCT

Additional factors beyond Risk Prediction
• How are they doing over time? (this is among the most crucial aspects to decision making but among the least studied)
  • Cancer
  • Patient preferences and risk tolerance
    – Both for treatment outcomes AND
    – For potential adverse effects (bleeding)
  • Overall functional status
  • Primary care physician engagement (if you can locate them)
  • Intensivist engagement
  • Resources available
embrace UNCERTAINTY
Frame your decisions around your patient’s HEALTH STATE

Case 3
29 year old female
CC: chest pain

History
• Sharp lateral pleuretic CP
• Some dyspnea when walking up stairs
• No PMHx, family hx PE
• JUST started OCPs

Exam
• HR=120, BP=121/58, sat 98%
• Clear lungs, nl work of breathing
• Cardiac exam nl
• No leg edema, tenderness, asymmetry

• ECG nl
• D-dimer+
• PE+ segmental PE LL and subseg RML

Additional info
CT read by radiologist as no RV strain
- Troponin 0.03
- BNP nl
- RA sats 96%
- repeat vitals unchanged

Decisions………

Considerations

Cost to health system $$$ (11)
High adverse events
What are they getting lip? They’re just discharged in the am?

Compliance?
Follow up?
Worsening clot?
Shock?
Death?
FECAR

So why would we want to treat some at Home?

Considerations

CLINICIAN Perspective

Cost to health system $$$ (11)
High adverse events
What are they getting lip? They’re just discharged in the am?

Compliance?
Follow up?
Worsening clot?
Shock?
Death?
FECAR

Intent

Cost (patient out of pocket)
Home of patient care
“they didn’t even do anything”
Can’t work
Can’t take care of family
HOW DOES ADMIT IMPROVE HEALTH STATE?

Pain
Worsening clot?
Shock?
Death?
Bleeding
Recurrence

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ACCP 2016 Guidelines-update


"In patients with low-risk PE and whose home circumstances are adequate, we suggest treatment at home or early discharge over standard discharge (e.g. after first 5 days of treatment) (Grade 2B)."

"Suggest that patients who satisfy all of the following criteria are suitable for treatment of acute PE out of hospital:

(1) clinically stable with good cardiopulmonary reserve;
(2) no contraindications such as recent bleeding, severe renal or liver disease, or severe thrombocytopenia (i.e. < 70,000 /mm3);
(3) expected to be compliant with treatment;
(4) patient feels well enough to be treated at home.

NOTE: they do not suggest risk scoring systems HAVE to be used (PESI I or II or sPESI =0) but should augment clinical decision making.

NOTE: they don't require biomarker/echo but RV strain discourages outpt Rx.

Update from AT9 (Antithrombotic 9th edition of guidelines 2012)

Zondag W 2011 J thrombosis and Haemostasis

PE Severity Prediction: Hestia Criteria

PE Severity Prediction: 30d mortality

So how to operationalize this at my hospital?

IN ED approach

Future Questions and Directions:

Will this go the way of DVT?

RCT

Embedding decision support in EMR

Pharmacy consult in ED

Phone call to patient's outpatient pharmacy to “pre-check” pt cost

Phone call with primary MD

Importance of documenting:

- Hestia or other risk assessment
- proximal DVT or PE
- CA
- unprovoked
- male
- prior VTE
- US for DVT (+/-)

THIS IS NOT JUST GIVE A SCRIPT FOR DOAC and send home!!!!

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Ultimate driver of ED Decisions and communication

• What’s likely to improve your patient’s health state....?

• We need more research to be able to measure this

• We need more acute care / emergency care interventions that target this.

Four Principles – to optimal patient care overall

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Thank you

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