Pulmonary Embolism/Aortic Dissection/AAA

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PULMONARY EMBOLISM/AORTIC DISSECTION/AAA

Pulmonary Embolism

I. EPIDEMIOLOGY

A. 500,000+ cases annually in U.S.

B. 200,000 deaths (usually diagnosed at autopsy)

C. Diagnosed mortality = 2-8%

D. Undiagnosed mortality = 30%

II. ETIOLOGY

A. Lower extremity DVT
   1. Popliteal, ileofemoral
   2. Pelvic
   3. Calf vein a significant cause of PE, often propagate to popliteal, ileofemoral

B. Upper extremity DVT
   1. Subclavian, axillary
   2. Majority are catheter related
   3. Emboli have equal risk of mortality as LE clots

C. Superficial thrombophlebitis

III. RISK FACTORS FOR THROMBUS FORMATION

A. Virchow’s triad: venous stasis, vessel wall injury and hypercoagulability

B. Acquired
   1. Immobilization (e.g., hospitalized patients)
   2. Surgery within 3 months (esp. orthopedic and neurosurgery)
   3. Malignancy (esp. pancreatic, lung, stomach, colon, ovary)
   4. Pregnancy (esp. post-partum)
   5. Oral contraceptives/hormone replacements
   6. Obesity
   7. Trauma to lower extremities/pelvis
   8. Previous PE/DVT
9. CHF
10. Smoking
11. Long distance travel
12. Antiphospholipid antibody
13. Lupus anticoagulant

C. Inherited

1. Factor V Leiden mutation
2. Antithrombin III deficiency
3. Protein C deficiency
4. Protein S deficiency
5. Hyperhomocysteinemia

IV. PRESENTATION OF PE

A. Classic triad of dyspnea, chest pain and hemoptysis < 20%

B. Symptoms

1. Dyspnea - 73%
2. Pleuritic chest pain - 66%
3. Cough - 37%
4. Hemoptyis - 13%
5. Syncope - 13%
6. New onset wheezing

C. Signs

1. Tachypnea (>16/min) - 70%
2. Rales - 51%
3. Tachycardia - 30%
4. Accentuated P2 - 23%
5. Fever (usually low-grade) - 14%
6. JVD
7. Parasternal lift

V. DIFFERENTIAL DIAGNOSIS

A. MI/angina

B. Pericarditis

C. Aortic dissection

D. CHF
E. Asthma

F. COPD

G. Pneumonia

H. Pneumothorax

I. Costochondritis

J. Pleurisy

VI. CLINICAL RULES FOR PREDICTING THE PROBABILITY OF PE: (WELLS CRITERIA)

A. Clinical signs and symptoms of DVT 3 pts.

B. Alternative Dx. less likely than PE 3 pts.

C. HR > 100 1.5 pts.

D. Immobilization or surgery in the previous 4 wks 1.5 pts.

E. Previous DVT or PE 1.5 pts.

F. Hemoptysis 1 pt.

G. Cancer 1 pt.

1. Clinical probability
   a. Low < 2
   b. Intermediate 2-6
   c. High > 6

VII. DIAGNOSIS

A. ABG

1. 20% of patients with PE have normal $pO_2 (> 85)$
2. A-a gradient 150- $(1.25 \times pCO_2)$
   a. Normal range = 13-20
   b. 6% with PE have normal gradient

B. EKG

1. Abnormal in 70% of PE patients
2. Sinus tachycardia
3. Non-specific ST changes (esp. inverted T waves V1-V4)
4. S1Q3T3
5. RAD
6. RBBB
7. Cor pulmonale (tall, peaked P waves in lead III)
8. New onset atrial fibrillation

C. CXR

1. Abnormal in 70% of PE patients
2. Atelectasis/infiltrate (esp. > 24 after onset of symptoms)
3. Pleural effusion
4. **Hampton’s hump** is rare (pulmonary infarction-triangular pleural based infiltrate with apex toward hilum)
5. **Westermark’s sign** is rare (sharp vessel cutoff)
6. Elevated hemidiaphragm

D. D-Dimer

1. Degradation product of cross linked fibrin
2. Non-specific esp. in post op, malignancy, increasing age, infection, pregnancy
3. Latex agglutination (e.g. SimpliRED) sensitivity only 50-60%
4. ELISA (90-95% sensitive)

E. VQ scan

1. Safe in pregnancy, but CT now preferable
2. High probability - PPV 85% = treat
3. Normal - 95% specific = no treatment
4. Nondiagnostic (intermediate probability/low probability) - PPV 20% - does not rule in or out PE. Must do additional studies.
5. PIOPED (Prospective Investigation of Pulmonary Embolism Diagnosis) JAMA 1990
   a. 59% of PEs were non-high prob. scans
   b. Combination of high prob. scan and clinical suspicion/low prob. scan and low clinical suspicion – PPV and NPV >95%, but most scenarios not like this
   c. Conclusion of study = pulmonary angiography for non-high prob. scans

F. CT (helical/spiral)

1. Advantages
   a. Rapid
   b. Can provide alternative diagnosis (e.g., dissection, pneumonia)
c. Newer multi-detector-row CT with thin collimation improves visualization of subsegmental vessels and improved sensitivity (90%) over 1st generation single-detector-row CT (70%) Some protocols also scan deep veins in pelvis/legs for DVT. The “triple rule-out”- PE, CAD and aortic dissection
d. Can also be used in pregnancy if necessary

2. Disadvantages
a. Contrast - renal toxicity and allergic rxns
b. Sensitivity 50-70%, specificity 90%
c. Misses distal emboli – doubtful clinical significance

G. Pulmonary angiography

1. Gold standard
2. PPV near 100%, NPV 90%
3. Mortality < 1%
4. Morbidity 5% (hematoma, contrast rxns, arrhythmia, resp. insuf.)

H. ECHO-trans-thoracic

1. Evaluates right heart strain (found in 40% of PE’s)
2. Perform on all patients with PE??

VIII. DIAGNOSTIC ALGORITHMS

A. High clinical suspicion + positive test = PE (PPV=96%)

B. Low clinical suspicion + negative test = probably not PE (NPV=96%)

C. High clinical suspicion + negative tests (e.g., D-dimer, CT, VQ) = additional tests, e.g. DVT evaluation, pulmonary angiogram

D. Low clinical suspicion and positive test = additional testing

IX. RISK STRATIFICATION

A. Low risk

B. Intermediate risk (submassive PE)

C. High risk (massive PE) increased troponin, BNP

X. TREATMENT

A. Anticoagulation
1. Heparin (unfractionated)
   a. 80 U/kg bolus and 18 U/kg/hr
   b. Monitor PTT - therapeutic at least 1.5 times control
   c. Approx. 50% of patients subtherapeutic at 24 hrs
   d. May cause heparin associated thrombocytopenia
   e. Safe in pregnancy
2. Low molecular weight heparin
   a. Enoxaparin (Lovenox) (1 mg/kg SQ bid)
   b. Fondaparinux (Arixtra) sq gd
   c. No monitoring required
   d. Less likely to induce thrombocytopenia
   e. Safer and more effective than unfractionated heparin
   f. Safe in pregnancy
3. Warfarin (Coumadin)
   a. 3-6 months if time limited risk factor (e.g. post-op, trauma, estrogen use), 6 months to life if idiopathic etiology or recurrent thromboembolism
   b. INR target 2.5
   c. Temporary hypercoagulable state for approx. 5 days
   d. Initial dose 5-10 mg po
   e. Teratogenic
4. Thrombolytic therapy
   a. For patients with “massive” PE
   b. Reduces morbidity and mortality
   c. Indications
      i. Hypotension (< 90 systolic)
      ii. Extreme hypoxemia
      iii. Right heart strain on ECHO
      iv. Use t-PA (Alteplase) 100mg over 2 hours (15mg bolus, 50 mg over 30 min and 35 mg over 60 min) or r-PA reteplase
      v. Controversial indication- “submassive PE (RV dysfunction, but normal BP)
5. Vena caval filters
   a. Indications
      i. Contraindications to anticoagulation (e.g. recent surgery, intracranial bleed, GI bleed)
      ii. Recurrent PE/DVT despite adequate anticoagulation (rare)
6. Surgical/transvenous embolectomy
   a. Massive PE with refractory hypotension despite thrombolytic therapy or if thrombolytics contraindicated

Aortic Dissection

I. INCIDENCE

   A. Laennec - first described dissecting aneurysms in 1826
B. Aortic dissection is the more accurate term

C. 2,000/yr in the U.S.

D. 3 male:1 female

E. Median age = 60 years old

F. Mortality (if untreated)
   1. 25% within one hour of event
   2. 50% at one week
   3. 90% at one year

II. RISK FACTORS

A. Hypertension
   1. 90% of patients are hypertensive on presentation or have a history of HTN

B. Congenital valvular disease
   1. Bicuspid or unicommissural aortic valve, coarctation

C. Connective tissue disease
   1. Marfan’s, Ehlers-Danlos, SLE, giant cell arteritis

D. Pregnancy – third trimester

E. Iatrogenic
   1. Cardiac catheterization, aortic clamping during surgery

III. PATHOGENESIS

A. Intimal tear with dissection of blood from the lumen into the media. Results in a true lumen and a false lumen

B. Origin
   1. 75% in proximal aorta
   2. 25% in distal aorta
IV. CLASSIFICATION

A. DeBakey: less useful
   1. Type I - begins in ascending aorta, extends distally
   2. Type II - confined to ascending aorta
   3. Type III - distal to left subclavian artery

B. Stanford: most useful
   1. Type A (Proximal) - (Type I & II) involves the ascending aorta with or without the descending aorta (75% of cases)
   2. Type B (Distal) - (Type III) dissection confined to the descending aorta (25% of cases)

V. CLINICAL PRESENTATION

A. Chest pain
   1. Present in 90% of patients (absent in 10%). Classically is tearing, ripping or stabbing in nature. Maximal intensity at onset which is sudden. May radiate to the back (interscapular) and then the mid and low back as the dissection propagates distally

B. Aortic insufficiency
   1. Common (>50% of patients)

C. CHF
   1. Secondary to aortic insufficiency

D. Myocardial infarction
   1. Uncommon
   2. Due to coronary artery occlusion (esp. right coronary artery)

E. Syncope
   1. Due to pericardial tamponade, hypotension or cardiac conduction disorders

F. Hypotension or pseudo-hypotension
   1. True hypotension secondary to free rupture of the dissection
   2. Pseudo-hypotension due to compression of the true lumen by the
false lumen resulting in reduced blood flow to the subclavian arteries with a difference in blood pressures between the arms (systolic difference of \( \geq \) 15 mm Hg is significant)

G. Neurologic

1. CVA secondary to carotid artery dissection/obstruction
2. Paraparesis or paraplegia secondary to spinal cord ischemia (spinal a. occlusion)

H. Renal

1. Renal artery occlusion may result in uncontrolled hypertension, decreased urinary output and a rising creatinine

I. Mesenteric ischemia

1. Secondary to superior mesenteric artery occlusion

VI. PHYSICAL EXAM

A. Hypertensive, or history of hypertension common

B. BP in both arms (SBP difference \( \geq \) 15 mm Hg)

C. Beck’s triad - muffled heart tones, JVD and hypotension if pericardial tamponade

D. Aortic insufficiency murmur

E. Pulmonary edema

F. CNS

G. EKG- LVH, MI, heart block

VII. DIAGNOSTIC EVALUATION

A. Value of D-dimer controversial – may be useful

B. Chest X-ray (should be taken upright)

1. Abnormal in 90% of patients (normal in 10%)
2. Mediastinal widening \( > 8 \) cm at T4 (75% of pts)
3. Indistinct aortic knob
4. “Calcium” or ring sign- (> 5mm separation of intimal calcification from outer border of aorta)
5. Dilated proximal aorta
6. Pleural cap (apical pleural effusion)
7. Left mainstem depression
8. Deviation of the tracheal or NGT to the right

C. Diagnostic information needed by the surgeons

1. Presence of dissection
2. Involvement of ascending aorta
3. Extent of dissection
4. Branch vessel involvement
5. Aortic insufficiency
6. Pericardial effusion

D. Angiography

1. 88% sensitive, 94% specific
2. Identifies branch vessels and evaluates the aortic valve
3. Disadvantages
   a. Invasive
   b. Specialized personnel required
   c. Uses contrast media
   d. May miss false lumen if clotted
   e. Patient must be moved out of the ED

E. CT

1. Noninvasive
2. 95% sensitive, 95% specific
3. Identifies other causes of mediastinal widening (e.g., tumors)
4. Disadvantages
   a. Uses contrast media
   b. Unable to access aortic valve, branch involvement
   c. Patient must be moved out of the ED

F. TEE (Trans-esophageal echo)

1. Portable (can study the patient in the ED)
2. No contrast media
3. 98% sensitive, 77% specific
4. Evaluates aortic valve, branch vessels, pericardial effusion
5. Disadvantages
   a. Operator dependence
   b. Invasive
   c. Availability
G. MRI

1. Noninvasive
2. 98% sensitive, 98% specific
3. MRI necessary for evaluation of aortic valve
4. No contrast
5. Disadvantages
   a. Availability
   b. Patient must be moved out of the ED
   c. Not for critical patients

VIII. INITIAL MANAGEMENT

A. Pain management

1. IV morphine

B. Decrease DP/DT (pressure wave over time)

1. B-blocker initially until heart rate 60-70 or SBP < 120 Hg
   a. Esmolol (Brevibloc) 500mcg/kg bolus, 50-200 mcg/kg/min infusion (nice due to rapid onset and only has a 5 minute half-life)
   b. Labetalol (Normodyne, Trandate) 20 - 80 mg IV q 10-15 min (also has alpha affects as well)
   c. Propranolol (Inderal) 0.5-1 mg IV q 5 min (difficult to titrate)
   d. Metoprolol (Lopressor) 5 mg IV q 5 min
2. Vasodilator after B-blockade (to avoid reflex tachycardia) until SBP < 120 mm Hg
   a. Nitroprusside (Nipride) 0.5 mcg/kg/min to start, increase q 5 min
   b. Cyanide toxicity if prolonged use
   c. Rapid onset and offset
   d. Nicardipine

IX. SUBSEQUENT MANAGEMENT

A. Type A dissection

1. Surgical repair (mortality averages 10%)

B. Type B dissection

1. Medical management (80% survival rate)
   2. Continued anti-hypertensive and B-blockade therapy
   3. Surgery considered if:
a. Aortic rupture  
b. Aortic branch compromise  
c. Persistent pain  
d. Marfan’s syndrome  
e. Disease extension despite medical therapy  

### AAA (Abdominal Aortic Aneurysm)  

#### I. INCIDENCE  

A. 1951 - Dubost in Paris: first successful repair of AAA  
B. 4 male: 1 female  
C. Peak incidence = 70 years old  
D. 98% infrarenal (50% also involve the iliac arteries)  
E. 33% of cases initially misdiagnosed  

#### II. RISK FACTORS  

A. Hypertension  
B. Smoking  
C. COPD  
D. Diabetes  
E. Hyperlipidemia  
F. Connective tissue diseases (e.g., Marfan’s, Ehlers-Danlos syndrome)  

#### III. PATHOPHYSIOLOGY/NATURAL HISTORY  

A. Atherosclerosis with loss of elastin and collagen in the aortic wall  
B. Normal aortic diameter = 2 cm.  
C. Uncommon rupture if < 5 cm (usual size for elective repair)  
D. Approximately 30% of aneurysms > 5 cm rupture within 5 yrs.
IV. CLINICAL PRESENTATION

A. Abdominal pain or tenderness (8 - 40%)

B. Back/flank pain
   1. Due to retroperitoneal, ureteral irritation

C. Pulsatile mass (50 - 90%)
   1. Left upper quadrant location

D. Abdominal distension
   1. From retroperitoneal or intraperitoneal blood

E. Abdominal bruit (3 - 8%)

F. “Blue toe syndrome” (5%)
   1. Atheromatous debris embolizes distally

V. DIFFERENTIAL DIAGNOSIS

A. Renal colic

B. Mesenteric ischemia

C. Diverticulitis

D. Cholecystitis

E. Pancreatitis

F. Musculoskeletal back pain

G. PUD w perforation

H. GI bleed (R/O aortoenteric fistula)

VI. DIAGNOSTIC EVALUATION

A. KUB/ x-table lateral (suggests diagnosis in 90% of patients)
   1. Calcified aortic wall seen in 60% of patients with AAA
   2. Loss of renal/psoas shadow in 75% of patients with AAA
3. Order to R/O other disorders, e.g. perforation, obstruction- not to diagnose AAA

B. Ultrasonography

1. Primarily used to screen asymptomatic patients and to follow the size of AAA
2. Increasingly used in the ED for symptomatic patients to detect the presence of an AAA
3. Difficult to differentiate ruptured vs. intact aneurysm
4. Obesity, intestinal gas may interfere with imaging
5. Portable (can study pt. in the ED)

C. CT

1. Ideal study to evaluate symptomatic, but hemodynamically stable patients
2. Evaluates extent of aneurysm (e.g. iliac artery involvement)
3. Evaluates both size and leakage
4. Does not require contrast media
5. Patient must be moved out of the ED

D. Angiography

1. May miss AAA because of mural thrombi
2. Identifies branch organ involvement (e.g. iliofemoral, renal, mesenteric vessels)
3. Uses contrast media
4. Patient must be moved out of the ED
5. Rarely used now

E. MRI

1. Equivalent to US and CT in assessing aortic diameter
2. Superior to US and CT at assessing branch vessel involvement
3. MRI angiography provides similar resolution to conventional angiography without the use of contrast media
4. Patient must be moved out of the ED
5. Not for critical patients
6. Not always available

VII. MANAGEMENT

A. Asymptomatic patient

1. < 4 cm AAA - repeat US q 6 months
2. ≥ 4 cm AAA - elective repair (some surgeons wait until 5 cm)
3. Option for elective repair includes transfemoral intraluminal graft
4. Implantation in high risk surgical patients
   a. 30 day mortality for surgery – 3%
   b. 30 day mortality for graft – 0.5%
   c. 2 yr mortality after either – 6%

B. Symptomatic, hemodynamically **stable** patient
   1. CT to confirm diagnosis
   2. 2 large bore IVs
   3. Type & cross 8 units PRBCs
   4. Urgent surgical consultation

C. Symptomatic, hemodynamically **unstable** patient
   1. Emergent surgical consultation
   2. Straight to operating room or consider ED ultrasound
   3. 2 large bore IVs
   4. Crystalloid, blood transfusions (resuscitate to SBP 85-95 mm Hg)

VIII. OPERATIVE MORTALITY

A. Elective repair - 4%

B. After rupture
   1. Not hypotensive - 20%
   2. Hypotensive responds to volume - 40%
   3. Hypotensive, incomplete response to volume - 60%
   4. Hypotensive, no urinary output - 80%

C. Median mortality rate after rupture – 45%
PULMONARY EMBOLISM/AORTIC DISSECTION/AAA

PEARLS

Pulmonary Embolism

1. Multiple inherited and acquired risk factors for venous thromboembolism.

2. Upper extremity DVT are often secondary to catheters.

3. Classic PE triad of dyspnea, chest pain and hemoptysis < 20% of patients.

4. Most common EKG changes are tachycardia and non-specific ST changes.

5. Classic EKG finding is S1E3T3.

6. Although the CXR is usually abnormal, the findings are non-specific.

7. ECHO is useful in looking for right heart strain.

8. ELISA D-dimer is much more sensitive than the latex agglutination test.

9. Massive PE is an indication for fibrinolysis.

10. New generations spiral CT has replaced VQ scanning. It can miss distal subsegmental emboli. Doubtful conical significance.

11. High clinical suspicion + abnormal test (e.g. CT or VQ scan) = treat.

12. Low clinical suspicion + normal test (e.g. D-dimer) = no treatment.

13. All other combinations require additional testing (e.g. lower extremity duplex, pulmonary angiogram).

14. Pulmonary angiography remains the gold standard for the diagnosis, but is underutilized.

15. LMWH is replacing heparin as the anticoagulant of choice.
Aortic Dissection

1. Occurs with greatest frequency age 60 years; 3:1 M:F.

2. Untreated mortality: 25% - 1 hour, 50% - 1 week, 90% - 1 year.


4. Pathogenesis: intimal tear with dissection through media resulting in 2 channels

5. Location: 65% ascending, 10% transverse arch, 25% descending (distal).

6. Clinical findings: chest pain, syncope, AI, CHF, MI, hypotension with diminished peripheral pulses, focal neurologic deficits, renal artery obstruction.

7. Classification: Stanford Type A-ascending (75%) usually managed surgically; Stanford Type B-distal (25%) usually managed medically.

8. CXR findings: dilated ascending aorta, obliterated knob, calcium “ring” sign (displaced intimal calcification), mediastinal widening, pleural cap, size disparity between ascending and descending.

9. Angio: demonstrates intimal flap between true and false lumen, narrowing of true lumen by false channel, extent of dissection, involvement of branch vessels, AI. Disadvantages: may fail to detect flap, may fail to opacify false lumen or may simultaneously opacify both channels, uses contrast media.

10. CT: similar accuracy as angio. Demonstrates intimal flap; defines true and false channels; reveals fluid in pleural/pericardial spaces, widening of aorta. Sensitivity and specificity = 100%. Drawback: uses contrast media.


12. Initial management: Eliminate pain (morphine.) and decrease sheer forces on aortic wall by decreasing velocity of LV contraction and systolic BP. Beta-blockade – propranolol, metoprolol, labetalol. Vasodilator - use after beta-blockade: nitroprusside or nicardipine
13. Subsequent management - Type A: surgical. Type B: medical (80% survival vs 50% survival with surgery) unless unable to halt progression of dissection.

AAA

1. Peak incidence 70 year old, 4:1 M:F.


3. Pathogenesis: localized dilation resulting from weakening of vessel wall; 98% infrarenal. Rare rupture if less than 5 cm.


5. Dx: plain radiographs - suggests dx 90%; ultrasound - detects and sizes aneurysm but may not distinguish intact from ruptured; CT - useful for symptomatic but “stable” patient. Predicts size and extent of aneurysm and presence of free fluid; angio - details arterial anatomy but defines only lumen of aneurysm.


7. Mortality: elective repair 4%; after rupture 20-80%.
REFERENCES


18. Marill KA. Serum D-dimer is a sensitive test for the detection of acute aortic dissection: A pooled meta-analysis.