Vascular Emergencies: How Rapid Recognition by Hospital Based Physicians Saves Lives

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Objectives

- Recognize the varied presentation of vascular emergencies
- Appreciate/understand the need for rapid recognition
- Become familiar with options for care based upon availability of resources and stability of the patient
Vascular Emergencies to be reviewed

- Acutely ischemic extremity
- Ruptured abdominal aortic aneurysm
- Acute mesenteric ischemia
- Thoracic aortic dissection
- Pulmonary thromboembolism
Acute Limb Ischemia

- Abrupt occlusion of arterial flow to an extremity
- Largely clinical diagnosis – NOT based on doppler study or CTA
- Delay in diagnosis common – Can result in limb loss, MSOF, or death
- Often seen in patients near end of life – Judgement is the key re: appropriate level of treatment
- Either embolic or thrombotic occlusion
Acute Limb Ischemia
Acute Limb Ischemia
Embolic Occlusion: Presentation

- Symptom onset more rapid and severe than thrombotic occlusion
  - Lack of collaterals
  - Symptom onset to motor dysfunction often less than 3 hours
- Initial loss of sensation with rapid progression to motor weakness
  - Excruciating pain – aching not burning, made better by dependency
- Look for asymmetry in color, temperature, function, pulses
- Assess length of time ischemia present – and compare to severity of symptoms
- Rule of P’s
  - Pallor
  - Pain/paresthesia
  - Pulselessness
  - Poikilothermia
  - Paresis
Acute Limb Ischemia
Embolic Occlusion: Etiology

- Embolus frequently originates from the heart
  - Atrial Fibrillation – “Stopped anticoagulation for a procedure”
  - Mural thrombus in ventricle after MI
    - Akinetic wall, inflamed endocardium
  - Rarely from valvular heart disease, endocarditis, or atheroembolism
    - Embolic material much smaller and less tissue ischemic

- Paradoxical embolus
  - DVT
  - PFO, ASD, VSD

- Mural thrombus from proximal aneurysm
Acute Limb Ischemia
Thrombotic Occlusion: Presentation

- Frequently presents with paresthesia with slow progression to motor dysfunction
  - Often many collaterals present
- Important to assess history of vascular intervention
  - Arterial stents
  - Prior leg bypass surgery
- Otherwise, findings of asymmetry similar to embolic
Acute Limb Ischemia
Thrombotic Occlusion: Etiology

- Etiology varied
  - Atherosclerosis – progressive, associated with hypotensive episode
  - Prior stent/bypass graft - occlusion
  - Thrombosed popliteal aneurysm
    - Can mimic embolic occlusion with severe ischemic limb
Acute Limb Ischemia
Differential Diagnosis

- Acute nerve compression –
  - Herniated disk
  - Vertebral compression fracture
  - Spinal stenosis
  - Typically radiating pain, improved with position change

- Trauma
  - External signs of injury
  - Compartment syndrome

- DVT
  - Swollen and warm

- Musculoskeletal
  - Rheumatologic
  - Myonecrotic

- Peripheral neuropathy
  - Diabetic pain
  - Burning and stinging
Acute Limb Ischemia
Initial Management

- Call vascular early – avoid delay
- Give 1mg/kg intravenous heparin *early* – avoid secondary distal thrombosis
- Hydrate intravenously – bolus normal saline
- Determine anatomic location of occlusion by exam/doppler (hand-held)
- Type and screen
- Make clinical decision whether thrombotic or embolic
- Consider CTA if:
  - Likely thrombotic occlusion
  - Imaging *immediately* available
  - Renal function allows – *no delay*
Acute Limb Ischemia

- Why is it important to distinguish between embolic and thrombotic?
  - Window of opportunity
    - Briefly open with embolic occlusion
    - Typically much longer before irreversible damage with thrombotic occlusion
    - Thrombotic occlusion often better after anticoagulation
  - Management different
    - Embolic often easily managed with 30 minute open operation with minimal anesthetic
    - Thrombotic more complicated and often requires hybrid approach
Acute Limb Ischemia
Treatment Options: Open vs ENDO

- **Open**
  - **Advantages**
    - Better option for embolic occlusion
      - Can be done with local anesthesia and small incision
    - Reconstruction and/or hybrid procedures can be performed
    - Limited bolus thrombolysis can be used
    - Fasciotomy can be done
  - **Disadvantages**
    - Difficult to clear small vessels of thrombus
    - Limits future thrombolytic use
    - May require deeper anesthetic than patient can tolerate
    - Incision(s) involved
Acute Limb Ischemia
Treatment Options: Open vs ENDO

- ENDO
  - Advantages
    - Minimal anesthetic required
    - Minimally invasive
    - Good imaging in cath lab
    - Ability to clear small vessels of clot – especially in thrombotic occlusion
  - Disadvantages
    - Longer time to clear obstruction
    - Availability of resources
    - Contrast exposure – allergies and renal function
    - Bleeding with thrombolysis
    - Unable to perform fasciotomy
Acute Limb Ischemia
Treatment Options: Open

- Balloon catheter-based
  - Embolectomy
  - Thrombectomy
  - Bypass

- Hybrid
  - Open combined with endo techniques
    - Angiojet
    - Drug–coated balloon angioplasty of residual stenosis
    - Pulse thrombolysis
    - Mechanical fragmentation/aspiration
Acute Limb Ischemia
Treatment Options: ENDO

- **Thrombolysis**
  - Contralateral groin stick
  - Up-and-over technique
  - Needs overnight ICU stay
  - Monitor fibrinogen and CBC’s
  - Multi side port catheter - McNamara®
  - Interval imaging evaluation and catheter advancement

- **Mechanical fragmentation-aspiration**

- **Drug coated balloons, stents, stent grafts**
Acute Limb Ischemia Adjunct Treatments and Outcomes

- Vigorous hydration and HCO3 – administration
  - Reduces incidence of acute kidney injury from myoglobinuria
  - Reduces hyperkalemia immediately following reperfusion, arrhythmia

- Fasciotomy
  - Depending on severity of ischemia/length of time before reperfusion
  - Best done at initial procedure
  - Avoids foot drop/compartment syndrome
  - At least anterior compartment release
  - Often done with small incision
  - Leave Nylon sutures in place to tie down later
Acute Limb Ischemia
Conclusions

- **Call EARLY** – Window of opportunity can close abruptly
- Diagnosis and etiology can be determined by you at the bedside
- Early administration of bolus heparin reduces limb loss
- Do not need CTA prior to calling
- Hand held doppler rather than formal doppler lab evaluation to save time
- Decisions regarding open vs endo approach
  - Based upon the availability of resources
    - Interventional cardiologist
    - Modern vascular surgeon
  - Based on etiology of ischemia
Ruptured Abdominal Aortic Aneurysm (AAA)

- 10th leading cause of death in men older than 55
- Less than half reach the hospital alive
- Overall mortality 70-90%
- Smoking – most strongly associated risk factor
- Women rupture at smaller diameter than men
- First degree relatives of women with ruptured AAA - > 50% have AAA (Black Widow Syndrome)
- Ruptured aneurysms do not “dissect”- they bleed!
Ruptured Abdominal Aortic Aneurysm (AAA) Profile Risk

- Related to aneurysm size, 5 year risk:
  - 4-4.9 cm – 5-8%
  - 5-5.5 cm – 20%
  - 5.5-6.0 cm – 30%
  - >6.0 cm – 50%

- Higher in women at same AAA size
- Higher in familial cases - > 2 first degree relatives
- Higher in AAA with asymmetric bulge
- Higher in AAA with rapid expansion - > 1 cm/year
Ruptured Abdominal Aortic Aneurysm Rupture Risk

- Role of intraluminal thrombus
  - **NOT** protective
  - Does not reduce pressure on wall
  - Produces localized hypoxia
    - Neovascularization and thinning
  - Metal metallo proteinases (MMP’s)
    - Degrade extracellular matrix
    - Seen in high levels in thrombus from AAA
  - 2013 Dutch study – Thrombus thickness correlated with levels of MMP and rupture risk
Abdominal Aortic Aneurysm (AAA)
Asymmetric Bulge and Intraluminal Thrombus
Ruptured Abdominal Aortic Aneurysm (AAA)
Signs and Symptoms

- Acute onset of back and abdominal pain
- Pain may radiate to scrotum/hip – easily confused with kidney stone
- Diaphoretic, pale, tachycardic patient
- Must exclude AAA rupture before considering other diagnosis – back pain, renal colic, etc.
- First physician seeing patient – Missed the diagnosis > 50% of the time!
Ruptured Abdominal Aortic Aneurysm Diagnostic Evaluation

- If clinical suspicion in emergency department – bedside ultrasound by ER physician
  - Rapidly evaluate aortic diameter
  - Can reliably exclude RAAA if no aneurysm seen
    - Imaging quality often an issue – may still need CT scan

- If clinically suspect RAAA in patient on floor
  - Call Radiology
    - Need STAT CT abdomen/pelvis with and without contrast
    - Try to minimize contrast volume
    - Tell them what you are looking for - will need thin cuts to plan EVAR
    - Patient must be monitored with RN and personnel present in CT scan

- If patient very unstable with abdominal and/or back pain – call vascular surgeon, and initiate “pathway”
Ruptured Abdominal Aortic Aneurysm (AAA) Ultrasound Image
Ruptured Abdominal Aortic Aneurysm (AAA)
CT with Ruptured AAA
Ruptured Abdominal Aortic Aneurysm (AAA)
Ruptured Abdominal Aortic Aneurysm
Treatment

- Pre-op/intra-op “needs”:
  - Type and cross – may need O negative blood
  - Two large bore IV’s
  - Prep and drape while awake
  - Local/MAC anesthesia for groin access
  - Cell saver available
  - C-arm and capable technician
  - Bookwalter/Omni retractor and assistants

- Repair method – As of 2015 - > 60% EVAR
Ruptured Abdominal Aortic Aneurysm
Permissive Hypotension

Fluid resuscitation should be kept to a minimum

- Keep systolic BP at level to maintain consciousness and prevent ST depression
- Aggressive crystalloid use may cause:
  - Increase in retroperitoneal bleeding/tear size in aorta
  - Hypothermia
- Over 5 liter crystalloid resuscitation is a predictor of increased mortality
Ruptured Abdominal Aortic Aneurysm
Method of Repair: EVAR

• Endovascular aneurysm repair (EVAR)
  • Preferred method if anatomy suitable
  • Femoral access under local anesthetics/sedation
  • Exchange wires and put up compliant large aortic occlusion balloon and 12 Fr sheath to buttress balloon
  • Contralateral access with marker angio catheter and quickly plan/perform case with balloon inflated
  • Use aortic balloon for suprarenal occlusion and deflate/remove just before main device deployed
• Measurements needed:
  o Neck length and diameter
  o Neck angulation
  o Presence of thrombus in neck
  o Iliac access issues
  o Landing zone
Ruptured Abdominal Aortic Aneurysm (AAA)
Ruptured Abdominal Aortic Aneurysm
EVAR
Ruptured Abdominal Aortic Aneurysm Method of Repair: Open Surgical Technique (OST)

- Transperitoneal vs retroperitoneal approach to aorta
- Aneurysm sac opened with interposition grafting
- Use of cell saver minimizes blood product need
- In very unstable patient avoids time needed for EVAR setup
- Most common method of repair until 2010
- Recent data show higher mortality and morbidity than EVAR
- Most commonly used because of unsuitable anatomy for EVAR
  - Female
  - Larger aneurysm sac size
  - Advanced age
Ruptured Abdominal Aortic Aneurysm
OST
Ruptured Abdominal Aortic Aneurysm
OST
Ruptured Abdominal Aortic Aneurysm (AAA) Abdominal Compartment Syndrome

- Seen after EVAR
  - Continued ooze from lumbar arteries and IMA into unopened retroperitoneum
  - Need Foley catheter with pressure monitor
  - Bladder pressure = Intraabdominal pressure
    - Bladder pressures > 20-25 make diagnosis

- Results in tense distended abdomen
  - Difficulty with ventilation
  - Compression of IVC/renal veins
    - Decreased cardiac output/venous return
    - Decreased renal function

- Decompressive laparotomy and mesh closure - treatment
- Mortality > 60%
Ruptured Abdominal Aortic Aneurysm (AAA)

- **Mortality**
  - EVAR – 30%, OST – 50%
  - Some selection bias against OST
  - Must have a standardized pathway for RAAA patients
    - Rapid recognition/CTA in ER
    - Rapid mobilization of personnel and equipment
    - Rapid transfer to equipped OR
  - Every minute delay from recognition to procedure start = 1% mortality
Ruptured Abdominal Aortic Aneurysm (AAA) Conclusions

- Suspect ruptured aneurysm in all patients with acute back and abdominal pain with signs of shock
- Improved CT availability should shorten time to diagnosis/treatment
- Ruptured aneurysm pathway very helpful in expediting treatment and reducing mortality
- Permissive hypotension can create better outcome
- EVAR is modern method of choice, but need resources and trained personnel available at all times
Beware of Invisible Cows

Most of the Mauna Kea access road below Hale Pohaku is open cattle range, and the cows frequently cross the road. Dark colored cows are often invisible in darkness and/or fog. Use extreme caution and drive very slowly in this open range.
Acute Mesenteric Ischemia (AMI)

- Sudden cessation of blood flow to intestines
- Occurs more commonly in women, elderly, critically ill
- Always involves SMA (superior mesenteric artery)
- Diagnosis delayed or missed by >50% of first evaluations
- Bowel metabolism high
  - oxygen needs great
  - rapid loss of viability with ↓ flow
- Like endocardium, inner lining of bowel (mucosa) affected first, therefore, physical findings delayed
- Overall-all cause mortality 75%
Acute Mesenteric Ischemia (AMI)
Differential Diagnosis

- Pancreatitis
  - Like mesenteric Ischemia, more pain/less physical findings
  - Usually less acute onset
  - Pain more commonly in back
  - Often prior history of same illness, gallstones, alcohol
  - Elevated lipase

- Ruptured aneurysm
  - Similar, severe abdominal pain-also pain often in back
  - Shock-like picture early, pulsatile mass felt

- Perforated bowel
  - Often similar acute onset
  - Peritonitis findings early, leukocytosis/fever
  - Free-air/abscess on imaging studies
Acute Mesenteric Ischemia (AMI) Differential Diagnosis (cont.)

- Diverticulitis
  - Longer time-frame of presentation
  - Typically localized discomfort
  - Not as ill initially

- Bowel obstruction
  - Nausea/vomiting more prominent
  - Less pain
  - Distended abdomen
Acute Mesenteric Ischemia (AMI)

Etiology of Ischemia

- Embolic - 55%
- Thrombotic – 30%
- Non-occlusive – 15%
Acute Mesenteric Ischemia (AMI)
Dead Bowel
Acute Mesenteric Ischemia (AMI) Embolic Etiology

- **Presentation**
  - Use very acute onset severe abdominal pain
  - Often sudden bowel evacuation without pain relief, hyperactive bowel sounds
  - Few/no findings on physical exam
  - Tachycardia with hypotension

- **Emboli lodge 5-6 cm down SMA at taper**
  - Often spares middle colic/jejunal branches
    - Different from thrombotic occlusion of origin

- **Source of clot**
  - Atria → atrial fibrillation
    - “I stopped my thinner for my operation”
  - Mural thrombus
    - Recent MI, severe CHF
  - Endocarditis
    - Vegetations
Acute Mesenteric Ischemia (AMI)
Thrombotic Etiology

- **Presentation**
  - Acute worsening of chronic pain
  - Often pre-existing history weight loss/postprandial pain
  - Frequently initiated by hypotensive episode
- Often loss of last open vessel (SMA) in patient with IMA/celiac obstruction
- Often a history of prior SMA stenting/bypass
- Thrombus develops 2° low flow state
  - Mucosa dies first – malabsorption/diarrhea develop
Acute Mesenteric Ischemia (AMI) Non-Occlusive Etiology

- **Presentation**
  - Often critically ill in ICU with unexplained acidosis/hypotension
  - Worsening abdominal pain in severe CHF

- **Elevated catecholamines with mesenteric vasoconstriction**
  - Intermittent reperfusion-release free radicals-worsen ischemia
Acute Mesenteric Ischemia (AMI) Diagnostic Aids

- **Clinical suspicion**
  - You have to consider the possibility

- **Duplex ultrasound**
  - Better with chronic ischemic-bowel gas/imaging difficulty

- **CTA – provides wealth of information**
  - Macrovascular anatomy
  - Bowel wall thickening – “thumb-printing”
  - Bowel wall enhancement in “late phase”
  - Avoid oral contrast agent – use oral water
  - Let Radiology know what you are looking for - call
Acute Mesenteric Ischemia (AMI) Embolic SMA Occlusion
Acute Mesenteric Ischemia (AMI)
Acute Mesenteric Ischemia (AMI)
Diagnostic Aids (cont.)

- Angiography
  - Best if done for thrombotic or non-occlusive
  - Use of thrombolysis, PTA/stenting if thrombotic and symptoms less than 12°
  - Intra-arterial vasodilators
Acute Mesenteric Obstruction
Acute Mesenteric Ischemia (AMI) 
Treatment: Embolic

- Anticoagulation – initial treatment for all etiologies
- Embolic occlusion only SMA involved
  - Laparotomy and SMA embolectomy
  - Bowel resection as needed
  - Use doppler or fluorescein for assessing viability
  - Consider second – look laparotomy 24 – 48 hours later
Acute Mesenteric Ischemia (AMI)  
Treatment: Thrombotic

- **Angiogram**
  - Try to cross occluded SMA with wire → TPA, +/- stent origin, old stent, old bypass
  - Can attempt direct SMA “stick” and retrograde crossing/PTA/stenting in OR
  - Still will need laparotomy to assess bowel viability

- **Bypass grafting**
  - Vein less likely to become infected
  - Prosthetic bypass less likely to kink
Acute Mesenteric Ischemia (AMI)

Treatment: Nonocclusive

- Vigorous volume resuscitation as tolerated
- Avoid Beta Blockers and Digoxin
- Angiogram
  - Catheter in SMA – Papaverine 30mg/hr.
- Surgical exploration
  - Only if acidosis/symptoms persist
- Use heparin via another route
Acute Mesenteric Ischemia (AMI)

Conclusions

- Diagnosis often delayed – must be considered in differential diagnosis
- Give heparin **EARLY**
- Use CTA as first diagnostic tool
- Mortality 30% if treatment before 4 hours from symptoms onset
- Mortality 90% if treatment > 12 hours out
- Involve vascular, general surgery early
Acute Aortic Dissection (AAD)

Pathology

- Intimal tear, with arterialized blood coursing into aortic wall and separating the layers
- Not at all the same as a ruptured aneurysm – often confused
- Origin of flap/tear
  - Ascending aorta 65%
  - Descending aorta 25% - Near subclavian origin
Intimal tear With Blood in Medial Layer of Aorta
Acute Aortic Dissection (AAD) Classification System

- Based on location of artery tear
  - Stanford A – 60% of total –
    - originates in ascending aorta
  - Stanford B – older patients
    - Originates in descending thoracic aorta
- Tear location determines treatment modality and urgency
- Acute vs. Chronic
  - Acute – within first 2 weeks of symptom onset
    - >70% patients who die do so in first 2 weeks
  - Chronic - > 2 weeks of symptom onset
Acute Aortic Dissection (AAD) Predisposing Factors

- Hypertension – severe - > 75% patients
- Bicuspid aortic valve
- Marfan’s syndrome, Ehlers-Danlos syndrome
- Pregnancy with pre-eclampsia
- Cocaine/methamphetamine abuse
Acute Aortic Dissection (AAD) Presentation

- Chest or back pain seen in 95% of patients
  - Described often as “tearing” – severe in quality – “worst pain ever”
  - Exclude MI with EKG/enzymes
  - Chest pain in type A; Back/abdominal pain in type B

- Syncope
  - ~ 10% of patients
    - Often with tamponade or brachiocephalic obstruction

- Refractory hypertension
  - More common in Type B (70%) than A (20%)
Acute Aortic Dissection (AAD)  
Risks if Untreated/Delayed Treatment

- **Type A**
  - Dissection across aortic-pericardial junction will lead to tamponade with rupture into pericardium
  - Dissection across ostia of coronaries can lead to acute compressive coronary occlusion
  - Dissection across aortic annulus may result in loss of valvular support and acute/severe aortic insufficiency
  - Acute compression of brachiocephalic origin may result in occlusion/stroke
Acute Aortic Dissection (AAD)
Acute Aortic Dissection (AAD)
Risks of Untreated/Delayed Treatment Type B

- **Malperfusion**
  - Spinal Cord - paralysis
  - Renal – acute renal failure/flank pain
  - Mesenteric – acute bowel ischemia
  - Lower extremity – cold leg

- **Aneurysmal degeneration of false lumen**
  - 30-50% patients in first 4 years with medical management
  - Partial thrombosis of false lumen
    - ↑ mean pressure in false lumen
  - Late rupture in 20%
Mechanism of Malperfusion
Acute Aortic Dissection (AAD) Diagnostic Workup

- Diagnosis missed initially 25% of time
- Exclude MI with EKG and enzymes
- PA/Lateral CXR – exclude pneumonic process
- CTA
  - Sensitivity/Specificity for dissection – 90/95%
  - Ask for “Helical Dissection Protocol”
    - Identifies entry tear sites
    - True and False lumen identified
    - Orientation of dissection flap
Acute Aortic Dissection (AAD) Diagnostic Workup (cont)

- **TEE**
  - Better than CTA in ascending aorta
    - Detects entry tear sites
    - False lumen flow/patency
    - Aortic insufficiency
    - Pericardial effusions
Classic Wide Mediastinum
Acute Aortic Dissection (AAD)

Ascending Dissection

Descending Dissection
Acute Aortic Dissection (AAD) Treatment/Repair Type A

- Urgent cardiac procedure
  - Replace ascending aortic segment – replace valve as needed
  - Hypothermic circulatory arrest
  - “Glue aortoplasty” of distal anastomotic layers with “felt buttress”
  - Use of TEE to help with valve replacement decisions
  - Selected patients may benefit from endovascular approach
  - Very poor outcomes if pre-op stroke
Acute Aortic Dissection (AAD) 
Type B: Initial Treatment

- ICU with arterial line, Foley catheter
- HTN control with IV Beta Blockers –
  - Esmolol/Labetalol
  - ↓ contractility/shear forces
- Pain control with IV narcotics
Acute Aortic Dissection (AAD)
Type B: Indications for Repair

- Persistent pain despite adequate BP control
- Aneurysmal dilatation >5 cm
- End organ (spinal cord, renal, mesenteric) or limb ischemia
- Retrograde dissection into ascending aorta
- Bleeding into pleural cavity
Acute Aortic Dissection (AAD)
Type B: TEVAR

Thoracic endovascular aortic repair (TEVAR)

- May have advantages in preventing false lumen enlargement if done relatively soon after stabilization (10 days)
- Indicated for failures of medical therapy as discussed earlier
- CBAD (chronic type B aortic dissection)
  - 5 year mortality
    - TEVAR = 11%
    - Medical therapy = 20%
  - Operative mortality
    - 2%
  - Often cover left subclavian artery – May need L carotid → subclavian bypass
Acute Aortic Dissection (AAD)
TEVAR Repair and Coverage of Left Subclavian
Acute Aortic Dissection (AAD)

Conclusions

- Diagnosis and treatment plan can be based on CT imaging
- All dissections involving ascending aorta are a cardiac emergency
- Uncomplicated Type B dissections undergoing paradigm shift in treatment
- Need to exclude dissection from diagnosis in all patients with non-anginal severe chest pain
Acute Pulmonary Embolism

- Defined as a clot dislodged from lower (rarely upper) extremity venous system to pulmonary arteries
- Third most common cause of death in hospitalized patients
- Accounts for 15-20% of all post-op deaths
- Most often multiple emboli and seen in lower lobes
- In one study of people < 30 years of age in ER with only pleuritic chest pain: 25% had PE, many of which were overlooked
  - 400,000 missed diagnosis/year in U.S.
Acute Pulmonary Embolism
Presentation

- Classic triad:
  - Pleuritic chest pain
  - Hypoxemia
  - Shortness of breath

- In everyday practice, presentation quite varied
  - Hemodynamic collapse
  - Progressive SOB, pleurisy
  - Tachypnea/Tachycardia
  - Slight cough
Acute Pulmonary Embolism

Presentation

- Risk factors
  - Immobilization
  - Contraceptive/hormone use
  - Malignancy
  - Travel of 4 or more hours
  - Prior pulmonary emboli
Acute Pulmonary Embolism
Differential Diagnosis

- Pneumonic process
  - Pneumonia
  - Empyema
  - COPD Exacerbation

- Myocardial Infarction

- Congestive Heart Failure

- Pericarditis

- Sepsis
Acute Pulmonary Embolism
Clinical Scoring Systems

- Clinical scoring system is needed to better screen patients who are likely to have PE
- Modified Wells Scoring system – high, moderate, low
  - Signs/symptoms DVT
  - Heart Rate > 100
  - Immobilization ≥ 3 days or surgery within 4 weeks
  - Hemoptysis
  - Malignancy
  - Prior DVT/PE
  - PE is #1 diagnosis
    - If high risk based on above – CTA
    - If low/moderate risk – d-dimer
    - If d-dimer positive – CTA
    - If d-dimer negative – stop
Acute Pulmonary Embolism
Clinical Scoring Systems (cont)

- Various other clinical scoring systems proposed to avoid unnecessary CTA’s
  - Revised Geneva Scoring System
    - Very similar to Modified Wells Score
  - Pulmonary Embolism Rule out Criteria (PERC)
    - In low risk patients eight criteria to screen out those patients who need no further studies
Acute Pulmonary Embolism

CTA

- Gold standard for diagnosis – has replaced pulmonary angiography
- Requires IV access/contrast administration
- Determines location, volume/number vessels involved, acute vs. chronic
- Other pneumonic processes can be excluded
- Can be replaced with VQ scan if contrast allergy/renal disease
  - V/Q much lower sensitivity/specificity
Acute Pulmonary Embolism

CT Images
Acute Pulmonary Embolism Management: Stable Patient

- **Stable patients with acute PTE**
  - sBP >100
  - Minor hypoxemia
  - Sub-massive PTE on CTA

- **Better with LMWH (Lovenox®) than IV UFH**
  - Exceptions →
    - Chronic kidney disease (dosing issues)
    - Patients with possible need for intervention
    - Patients who are at risk for becoming unstable
  - Start with IV heparin for 24 to 36 hours. Once stability is assured, switch to Lovenox®
Acute Pulmonary Embolism Management: Stable Patient (cont)

- Anticoagulation
  - Patients with reversible risk factors and first event - 3 months and reassess
  - Patients with irreversible risk factors - long term therapy
  - Patients with second event – long term therapy
  - Cancer patients
    - LMWH better
Acute Pulmonary Embolism Management: At-Risk Stable Patient

- **Massive PE**
  - Involves both main pulmonary arteries on CTA
  - Often leads to hemodynamic compromise – sBP < 90
  - Evidence of right heart strain
  - Involves 5-8% of patients with PTE
  - Mortality 30-50%
  - Thrombolysis indicated if patient becomes hemodynamically unstable and no contraindication
    - ↑ risk of bleeding, especially in elderly
Acute Pulmonary Embolism Management: Concepts

Right ventricular strain
- Predictor of hemodynamic instability
- Helpful in determining patients with compromised right ventricular outflow
- EKG evidence
  - T wave inversion in precordial in V1 through V4 leads and in inferior leads (II, III, and aVF)
- Echocardiography evidence
  - Moderate to severe tricuspid regurgitation
  - Paradoxical septal wall motion towards left ventricle
  - Right ventricular and pulmonary artery dilatation
- Along with CTA evidence of massive PE, a good predictor for impending shock and in-hospital mortality
  - Thrombolysis shown to reduce mortality here yet with more bleeding complications
Acute Pulmonary Embolism Management: Unstable Patient

- Unstable patients (BP < 90) with low risk for bleeding
  - Thrombolysis - reduces mortality in PE
    - Need low risk patient
    - TPA half-life 20 minutes – should not interfere with surgical decision
  - Give TPA early to reduce chance of cardiogenic shock
  - Add IVC filter - ↓ mortality further
  - Major bleeding 2.7 times greater with TPA than with simple anticoagulation
    - Tripled in patients > 65 years of age
    - ↑ risk intracranial hemorrhage (ICH)

- Unstable patients who do not improve quickly with systemic TPA or who have a high risk for bleeding
  - Catheter directed thrombolysis
  - Catheter based fragmentation of clot with aspiration
  - Surgical embolectomy
Acute Pulmonary Embolism
Conclusion

- If in doubt, give IV heparin early
- If patient unstable and no contraindication give thrombolysis early – the earlier given, the lower the mortality
  - Can be given in the ER
- Surgical embolectomy has been suggested to lessen the incidence of post embolic pulmonary hypertension and may need to be used more often