PAD, Diseases of the Aorta, Venous Thromboembolism, and Key Issues in Cardiothoracic Trauma

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Keep on Growing
Lower Extremity Peripheral Arterial Disease

Risk Factors for PAD

- Cigarette smoking
  - 80% of PAD patients
- Diabetes
- Dyslipidemia
- Hypertension
- Hyperhomocysteinemia
  - 30-40% PAD patients
- Increased C reactive protein
- Age
Medical Conditions and Symptoms for High Risk

- CAD
- Carotid / cerebrovascular disease
- Renal artery stenosis

- Leg pain (claudication or rest)
- Abnormal pulse assessment

Assessment: History

- Walking impairment
- Classic claudication
- Rest pain (recumbent position)
- Poor healing of lower extremity wounds
- Abdominal discomfort after eating
- Family history of 1st degree relative with AAA
Physical Assessment

- Bilateral arm pressure
- Carotid pulse assessment
- Palpation of abdomen
  - Pulsatile mass
- Auscultation for bruits
- Peripheral pulse assessment (0 to 3)
- Inspection of feet / nails
  - Distal hair loss
  - Hypertrophic nails

Ankle Brachial Index

- Most objective measure of lower extremity PAD
- Does not correlate strongly with walking impairment
- Systolic in brachial arteries, posterior tibial, and dorsalis pedis arteries
  - Supine resting position for 10 minutes
- Ankle pressure should be 10 to 15mmHg higher than brachial pressure
- Normal ABI > 1.0
Indications for Resting ABI

- Exertional leg symptoms
- Non healing wounds
- $\geq$ 70 years
- $\geq$ 50 years with history of smoking or diabetes

Ankle Brachial Index

- Above 0.9 = Normal
- 0.71 – 0.9 = Mild disease
- 0.41 – 0.7 = Moderate disease
- < 0.40 = Severe disease
- Patients with ABI > 0.5 are not likely to progress to critical limb ischemia

- Toe Brachial Index
  - Non compressible arteries
- Exercise Ankle Brachial Index
  - Normal ABI
Other Diagnostic Studies

- Continuous Wave Doppler Ultrasound
  - Velocity waveforms and systolic blood pressure at sequential segments
  - Location and severity of disease, quantitative improvement
- Lower extremity duplex ultrasound
  - Peak systolic velocity ratios
  - Location and degree of stenosis
  - Follow up after surgical revascularization
  - Aneurysms and dissections
- Abdominal ultrasound
  - Aortic and abdominal aneurysms
- MRA / CTA
  - Aortic and abdominal aneurysms
  - Lower extremity PAD
- Contrast angiography
  - Gold standard
  - Contrast induced nephropathy

Claudication

- Definition
  - Ischemic pain during exercise
  - Specific leg muscle groups
  - Relieved in 2 to 3 minutes
  - Reproducible, stable
- Prevalence
  - Most common symptom
  - Symptoms only in 20% of patients with PAD
- Location
  - Gastrocnemius muscle
  - Relation of pain to location of disease
Claudication: Differential Diagnosis

- Lumbar disc disease (sciatica)
- Spinal stenosis
- Osteoarthritis
- Muscle pain
- Neuropathy
- Chronic compartment syndrome
- Venous obstructive disease

Critical Limb Ischemic (CLI)

**Definition**

- Limb pain at rest or night
  - Pain requiring narcotics
  - Pain particularly in forefoot or toes
- Ulcers or gangrene
  - Painful and tender to touch
  - Pain particularly in forefoot or toes
- Impending limb loss
Risk Factors for Critical Limb Ischemia

- ABI < 0.4 in non diabetic patients
- Diabetic patients
- Chronic renal failure
- Low cardiac output state
- Infection, injury or skin breakdown of affected extremity

Signs and Symptoms of Critical Limb Ischemia

- Pain occurring at night or rest
  - Forefoot or toes
- Pain requiring narcotics
- Arterial ulcers or gangrene
  - Painful / tender to touch
  - Open (infection / cellulitis)
    - Specialized wound care
  - Diabetics / immunocompromised
    - Systemic antibiotics
- Impending limb loss due to compromised flow
- Subclinical signs
Other Signs of Chronic Limb Ischemia

- Dependent redness
- Pallor on elevation
- Decreased capillary refill
- Shiny skin / subcutaneous tissue loss
- Trophic skin changes
- Loss of hair
- Calf atrophy
- Potential tissue loss

Critical Limb Ischemia from Atherosclerotic Emboli

- Post endovascular procedure
- Systemic fatigue or systemic muscle discomfort
- Increased creatinine
- Bilateral limb symptoms
- Skin discoloration
  - Red non-blanched network pattern
Acute Limb Ischemia

Causes

- Thrombosis with plaque rupture
- Thrombosis of bypass graft
- Thromboembolization from aneurysm
- Arterial trauma or dissection
- Vasospasm
- Compartment syndrome
- Hypercoagulability
Acute Limb Ischemia

Signs and Symptoms

- Pain
- Paralysis
- Parathesias
- Pulselessness
- Pallor
- Polar
Treatment Goals for Lower Extremity PAD

- Reduce risk of ischemic events (MI and stroke).
- Relieve symptoms of claudication and increase functional capacity and quality of life.
- Salvage limb and reduce mortality if CLI is present.
- Reduce risk, identify, and treat all other forms of vascular disease.

Risk Reduction

- Smoking Cessation
- LDL-C < 100 mg/dL or < 70 mg/dL
- BP < 130/80 for diabetics or patients with renal disease
  - Beta blockers are not contraindicated; do not impact walking capacity
  - Potential for any antihypertensive to exacerbate symptoms
- HgbA1C < 7%
Diabetic Foot Care

- Proper foot wear
- Routine care from podiatrist
- Daily foot inspection
- Skin cleansing and topical moisturizers to prevent dryness and fissures
- Skin lesions addressed urgently

Treatment for Claudication

- Supervised exercise
  - Increased speed, distance and duration of walking.
  - Decreased claudication per each distance.
  - 100-200% improvement in walking is expected over 3 to 6 months.
  - Increase in routine activities of daily living.
  - Increase in quality of life
  - Improvements with supervised exercise exceed the improvements with medication therapy.
Components of Supervised Exercise

- Treadmill or track walking
- 45 to 60 minutes per session
  - Exercise 35 to 50 minutes
- Minimum of 12 weeks
- Periods of exercise and rest
- Goal: Induce claudication in 3-5 minutes
- Resistance training as adjunct only

Treatment for Claudication

- Pharmacological Treatment
  - Cilostazol (Pletal)
    - PDE Inhibitor (inhibit platelet; vasodilator; positive lipid effects)
    - 40-60% improvement in walking
  - Pentoxifylline (Trental)
    - Decrease blood viscosity; decrease neutrophil adhesion, decrease fibrinogen
    - 20-25% improvement in walking
  - L-arginine (precursor to nitric oxide)
  - Propionyl –L-Carnitine (co-factor skeletal muscle metabolism)
  - * Ginkgo biloba (decreases viscosity / inhibits platelets)
Treatment for Claudication

- Not recommended
  - Oral prostaglandins (Iloprost)
  - Vitamin E
  - Chelation
    - Risk for hypocalcemia

Revascularization

- Goal: Improve quality of life
- Indications
- Classification of Lesions
  - Inflow disease
    - Suprainguinal vessels – limit blood flow to common femoral
  - Outflow disease
    - Stenosis from common femoral to infrapopliteal trifurcation
  - Runoff disease
    - Stenosis in trifurcation vessels to pedal arteries
Endovascular Treatment: Intermittent Claudication

- Preferred method for single stenosis in iliac lesions and femoropopliteal lesions
- Primary stenting used for iliac lesions
- Primary stenting not used in femoral, politeal, and tibial arteries
- Long term patency greatest in common iliac

Current Issues
- No role for DES
- Role in limb salvage
- Laser, cryoplasty balloon, mechanical atherectomy
Surgical Treatment: Intermittent Claudication

- Indicated for longer lesions or diffuse disease
- CAD evaluation required
- Inflow lesions are corrected before outflow lesions
- Surgery avoided in younger patients

Surgical Treatment: Intermittent Claudication

**Inflow Procedure**
- Aortobifemoral bypass
  - Bifurcated synthetic graft
  - Proximal: sewn to aorta immediately below renal arteries
  - Distal: Common femoral arteries or deep femoral arteries

**Outflow Procedure**
- Femoral-popliteal bypass
  - Vein grafts are superior to synthetic grafts
Treatment for CLI

- Goal: Avoid amputation
- Revascularization evaluation
- Endovascular treatment is first line
- Inflow lesions treated first
- Adequate flow to foot required for healing
Amputation

- Significant necrosis of the weight bearing portion of foot
- Uncorrectable flexion contracture
- Paresis of the extremity
- Refractory rest pain from ischemia
- Sepsis
- Very limited life expectancy

Other Treatments for CLI

- Antiplatelet and anticoagulation therapy
- Intravenous (or intraarterial) prostanoids (vasodilator and platelet aggregation inhibitors)
  - Prostaglandin E1 (PGE1)
  - Prostacyclin (PGI2) (Iloprost) Note: Oral Iloprost is not effective in reducing the risk of amputation or death.
- Parental pentoxifylline treatment is not useful for CLI
- Maintenance of limb in dependent position
- Treatment of infection to reduce demand
Treatment for Acute Limb Ischemia

- Systemic anticoagulation
- Catheter based thrombolysis
  - Urokinase (preferred over streptokinase)
  - Intraarterial
  - Best if ischemia < 14 days
- Mechanical thrombectomy devices
  - Option when contraindication to thrombolysis

Aneurysms
Abdominal Aortic Aneurysm

- Aortic diameter 3 cm
  - 50% or > from normal arterial diameter

Approximately 1.7% of women and 5% of men have an aortic diameter of 3.0 cm or more by the age of 65 years.

Prevalence increases by 6% per decade thereafter.

Risk factors for AAA

- **Male**
- **Family history**
- **Advanced age**
- **Cigarette smoking**
  - Polycystic kidney disease
  - Coronary artery disease, cerebral vascular disease, or peripheral arterial disease
    - Approximately 10% of patients with cerebral arterial disease *
  - Popliteal aneurysms
  - Marfan syndrome
  - Body Mass Index

*Hypertension has not been identified as a consistent risk factor.
AAA Pathophysiology

- Degeneration of media (and adventia)
  - 90% of infrarenal
  - Elastin fragmentation and degeneration are observed in aneurysm walls.

- Inflammatory process
  - AAAs demonstrate a chronic adventitial and medial inflammatory infiltrate

- "Inflammatory Aneurysms" – smokers
  - White shiny fibrotic material; adhere to structures
  - More symptomatic – increase surgical mortality

- "Infectious Aneurysms"
  - Secondary infection from existing aneurysm

Diagnosis

- Diagnostic studies
  - Most asymptomatic
  - Ultrasound
  - CTA or MRA for mapping for surgical intervention

- Physical exam
  - Pulse palpation
  - Aneurysmal palpation

10-20% with focal out pouching or blebs – though to contribute to risk for rupture.
Elements of Aortic Imaging Reports

The following table outlines specific qualitative and quantitative elements that are important to include in CT and MR reports.

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>The location at which the aorta is abnormal.</td>
</tr>
<tr>
<td>2.</td>
<td>The maximum diameter of any dilatation, measured from the external wall of the aorta, perpendicular to the axis of flow, and the length of the aorta that is abnormal.</td>
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<tr>
<td>3.</td>
<td>For patients with presumed or documented genetic syndromes at risk for aortic root disease measurements of aortic valve, sinuses of Valsalva, sinotubular junction, and ascending aorta.</td>
</tr>
<tr>
<td>4.</td>
<td>The presence of internal filling defects consistent with thrombus or atheroma.</td>
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<tr>
<td>5.</td>
<td>The presence of intramural hematoma (IMH), penetrating atherosclerotic ulcer (PAU), and calcification.</td>
</tr>
<tr>
<td>6.</td>
<td>Extension of aortic abnormality into branch vessels, including dissection and aneurysm, and secondary evidence of end-organ injury (eg, renal or bowel hypoperfusion).</td>
</tr>
<tr>
<td>7.</td>
<td>Evidence of aortic rupture, including periaortic and mediastinal hematoma, pericardial and pleural fluid, and contrast extravasation from the aortic lumen.</td>
</tr>
<tr>
<td>8.</td>
<td>When a prior examination is available, direct image to image comparison to determine if there has been any increase in diameter.</td>
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Note: This is Table 5 in the full-text version of the TAD Guideline.
Classification of AAA

- **Infrarenal**
  - Below renal arteries
- **Juxtarenal**
  - Just below renal arteries
- **Pararenal**
  - Involving origin of renal artery (arteries)
- **Suprarenal**
  - Involving aortic segment containing visceral arteries
- **Type IV thoracoabdominal**
  - Suprarenal extending to level of diaphragm

Complications of Aneurysms

- Dissection resulting in rupture or occlusion
  - Rupture most common complication
  - Mortality associated with rupture is as high as 90%
  - Aneurysm size most important predictor for rupture
  - Other factors increasing risk for rupture include: hypertension, COPD, tobacco use, family history and female gender

- Thromboembolic ischemic events

- Compression and resulting damage to nearby anatomical structures
  - Very large or inflammatory aneurysms can compress the duodenum
  - Popliteal aneurysms can compress the popliteal veins and cause venous insufficiency
  - Very uncommon but devastating complications are aortoenteric fistula causing GI bleed or aortocaval fistula causing acute congestive heart failure
Patients with AAA have an increased risk of other cardiovascular events. In one longitudinal study, there were 110 deaths in patients with aneurysms and only 6 were due to rupture. (Newman et al., 2001)

**ADDITIONAL RISKS ASSOCIATED WITH ANEURYSMS INCLUDE DEATH FROM OTHER CARDIOVASCULAR CAUSE.**

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**Criteria for Aneurysm Repair**

- Infrarenal or juxtarenal aneurysms 5.5 cm or >
- Suprarenal > 5.5 to 6.0 cm
  - Aneurysms above the renal arteries have a higher post operative mortality and higher risk for renal insufficiency and other complications. This is the reason for the larger diameter size required for elective repair.
- Aneurysms that have demonstrated a growth spurt
Aneurysm Expansion

- Proteolytic destruction of extracellular matrix
- Atrophy of smooth-muscle cells
- Inflammation

Surgical specimens of AAA reveal inflammation, with infiltration by lymphocytes and macrophages; thinning of the media; and marked loss of elastin.

Retrospective data has provided evidence that statin therapy may slow rate of aneurysm growth and reduce risk of rupture (inhibit proteolytic destruction).

Other Treatment Considerations

- Hypertension and smoking accelerate the rate of aneurysm growth therefore smoking cessation and tight blood pressure control are important aspects of patient management.

- Beta-blocker may be used in appropriate patients to decrease rate of aneurysm expansion.

- Retrospective data demonstrates some evidence that ACE-I may reduce risk of rupture.
Treatment for Symptomatic Aneurysms

- Symptomatic aneurysms are surgically treated regardless of size
  - An evaluation for urgent surgery is done with the following presentation:
    - Back pain or abdominal pain
      - Pain is most common symptom
      - Pain is a long lasting steady pain not generally affected by movement but some relief may be obtained with knees in a bent position.
      - Abrupt, severe, worsening pain, or pain radiating toward lower extremities is sign of impending rupture.
    - Hypotension
      - Hemorrhagic shock develops rapidly
    - Pulsatile abdominal mass
      - Mass will not be pulsatile with significant hypotension

Elective Repair AAA

- Size of aneurysm
  - Aneurysm volume is a better predictor of areas of peak wall stress than aneurysm diameter

- Preoperative assessment
  - CTA or MRA

- Perioperative Management
  - Beta-blockers

- Surgical Considerations
  - Aortic cross-clamping above renal arteries
  - Mortality risk
    - Infrarenal versus Type IV
Endovascular Repair

- Designed to decrease risks associated with open repair.
- Provides option for high risk patients not candidates for open repair.


- Aorta is not cross-clamped during deployment
- Y-shaped endograft
  - Two branches for the iliac arteries
  - Main trunk for the proximal aorta
- Modular in design
  - One component for main aortic trunk and one limb of the iliac artery
  - Second component deployed in the contralateral iliac artery.
  - Third component may act as an extension of the main system if needed
Endovascular Repair

- Synthetic fabric reinforced with stent struts
- Proximal end deployed proximal to neck of aneurysm
  - Barbs are used to fixate and prevent migration
- After placement of contralateral iliac portion of stent graft – balloon inflation is used to secure the sites of anastomosis

Clinical Evaluation of Endovascular Repair

- EVAR trial 1
  - Healthy patients
  - 30 day mortality lower for endovascular
  - Mortality from aneurysms lower with endovascular
  - Repeat interventions increased with endovascular
  - No difference ALL CAUSE mortality

- The Dutch Randomized Endovascular Aneurysm Management (DREAM) trial
  - Similar results to EVAR 1

- EVAR trial 2
  - Non open repair candidates
  - No benefit of endovascular repair

- Questions comparing endovascular repair to open repair?
  - 30 day mortality
  - Aneurysm related mortality
  - Reintervention
  - All cause mortality
  - Operable versus inoperable candidates
  - Endovascular versus medical management in non operative candidates
Technical Considerations

Preprocedural 3D Imaging

Specific Anatomic Criteria

Note: Patients must meet specific anatomic criteria to be eligible for endovascular repair.

- Studies suggest only 14-54% of patients meet anatomic criteria.
Endograft Leaks

- **Result in continued blood flow into the aneurismal sac.**
- **Typically occur within 1 year after surgery and are the most common reason for repeat intervention.**
- **Type I** - caused by problem with proximal or distal graft attachment sites. Can cause high pressures to build inside the aneurismal sac and can lead to rupture. Must be repaired.
- **Type II** - caused by retrograde flow from branch vessels. Very common. Majority seal spontaneously. Considered the most benign of the 4 types of leaks.
- **Type III** - caused by a defect in the actual graft. Require prompt surgical repair.
- **Type IV** - Diffuse leaks throughout the graft. These are rare.

Other Complications

- **Vascular injury – rupture of aneurysm**
- **Distal ischemia from mechanical obstruction, thrombosis, or embolism**
  - Early devices up to 10% of patients
- **Endotension**
  - No leak
  - Yet - excluded aneurismal sac continues to enlarge and remain under high pressure.
- **Endografts may also migrate if the proximal aorta expands.**
  - Graft diameters are oversized to accommodate the future expansion of the aorta.
  - Late complication.
Open Repair of AAA

- Mid-line transabdominal approach or extraperitoneal left flank incision.
- Thoracoretroperitoneal approach necessary for type IV aneurysms.
- Pararenal, suprarenal and type IV aneurysms require aortic cross-clamping above the renal arteries.

Open Repair for AAA

- Postoperative renal insufficiency is the most common complication in patients requiring aortic cross-clamping above the renal arteries.
- Type IV aneurysm repair also has a small risk for spinal cord ischemia.
- Infrarenal aneurysm repair has the lowest operative mortality.
- Type IV aneurysms have the highest operative mortality
  - (Hirsch et al., 2005).
Dacron tube graft:
- Allow ingrowth in the interstices to form a pseudoendothelial layer.
- Knitted or woven.
- Knitted: more porous, incorporate tissue well; prone to more bleeding.
- **Woven**: more impervious, most commonly used for aortic replacement.
- Typically impregnated with collagen to avoid preclotting and promote optimal healing.
Thoracic Aortic Aneurysms

- Ascending aortic aneurysms
  - Degenerative changes in medial layer of artery
- Descending aortic aneurysms
  - Usually atherosclerotic in nature

Reasonable to reduce blood pressure with beta blockers and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers to the lowest point patients can tolerate without adverse effects.

Beta blockers (must) and angiotensin receptor blockers (losartan) is reasonable for patients with Marfan syndrome, to reduce the rate of aortic dilatation unless contraindicated.

Source: ACC / AHA 2010 Guidelines
Ascending and Aortic Arch Aneurysms

**Indications for Surgical Repair Evaluation**
- Symptomatic aneurysms
- Expanding aneurysms (> .5 cm/year)
- Aneurysms 4 cm to 5 cm (with Marfan Syndrome and other genetic disorders) or > 5.5 cm (without Marfan Syndrome)
- Aneurysms between > 4.5 cm if aortic valve surgery is indicated
- Any acute Type A dissection as discussed below
- Mycotic aneurysms

Surgical Considerations
- CPB required
  - Deep hypothermic circulatory arrest (DHCA) with or without antegrade or retrograde cerebral perfusion is usually used to facilitate reanastomosis of the arch vessels

* Distal aortic arch via left thoracotomy without CPB
“Elephant Trunk Procedure

Left, Preoperative disease. Middle, Stage I with replacement of ascending aorta and arch with a Dacron graft with the distal graft sutured circumferentially to the aorta distal to the left subclavian artery and the free end of the graft (“elephant trunk”) within the descending aneurysm. Right, Completion of procedure using an endovascular stent graft attached proximally to the “elephant trunk” and the distal end secured to a Dacron graft cuff.

SOURCE: Images reprinted with permission from the Cleveland Clinic Foundation.

Descending Thoracic and Thoracoabdominal Aneurysms

**Open Surgical Indications**
- Symptomatic aneurysms (i.e. end organ ischemia)
- Aneurysms > 6.0 cm (or less if genetic etiology)
- Complicated acute Type B dissections as described below. Uncomplicated acute Type B dissection if low risk patient.

**Surgical Considerations**
- CAD evaluation
- Anticipated clamp time of less than 30 minutes may be done via "clamp and go"
- Complex or larger aneurysms either
  - left heart, partial, or
  - full cardiopulmonary bypass with hypothermic circulatory arrest.
- Neurophysiology monitoring is reasonable
- Cerebral spinal fluid drainage (focus on spinal cord perfusion pressure)
- Novel approaches to spinal cord protection
- Renal protection strategies
Hybrid Open Visceral Revascularization and Endograft Aneurysm Exclusion

Schema of TAA treated with initial left iliac artery–to–left renal artery–to–superior mesenteric artery bypass graft and subsequent placement of a thoracoabdominal endograft.

Proximal superior mesenteric artery and left renal arteries were ligated.

SMA indicates superior mesenteric artery; and TAA, thoracoabdominal aneurysm.


Source: 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the Diagnosis and Management of Patients with Thoracic Aortic Disease

**ISSUE:**

Patients are usually asymptomatic until a catastrophic event occurs. Therefore: Identifying disease in high risk patients while still stable is a priority.
Pathophysiology

- Intimal tear
- False channel

- Risk of rupture: outer wall
- Dissection – hematoma – occlusion of vessels
Risk Factors for Development of Thoracic Aortic Dissection

Conditions Associated With Increased Aortic Wall Stress

- Hypertension, particularly if uncontrolled
- Pheochromocytoma
- Cocaine or other stimulant use
- Weight lifting or other Valsalva maneuver
- Trauma
- Deceleration or torsional injury (e.g., motor vehicle crash, fall)
- Coarctation of the aorta

Note: Information on this slide is adapted from Table 9 in full-text version of TAD Guidelines

Risk Factors for Development of Thoracic Aortic Dissection (continued)

Conditions Associated With Aortic Media Abnormalities

Genetic

- Marfan syndrome
- Ehlers-Danlos syndrome, vascular form
- Bicuspid aortic valve (including prior aortic valve replacement)
- Turner syndrome
- Loeys-Dietz syndrome
- Familial thoracic aortic aneurysm and dissection syndrome

Note: Information on this slide is adapted from Table 9 in full-text version of TAD Guidelines
Genetic Disorders

Marfan Syndrome

Ehlers-Danlos Syndrome, Vascular Form

Bicuspid Aortic Valve

Turner Syndrome

Characteristic facial features
Fold of skin
Construction of aorta
Poor breast development
Elbow deformity
Rudimentary ovaries
Gonadal streak
(mesonephric gland)
No menstruation
Risk Factors for Development of Thoracic Aortic Dissection (continued)

Conditions Associated With Aortic Media Abnormalities (continued)

Inflammatory vasculitides
- Takayasu arteritis
- Giant cell arteritis
- Behçet arteritis

Other
- Pregnancy
- Autosomal dominant polycystic kidney disease
- Chronic corticosteroid or immunosuppression agent administration
- Infections involving the aortic wall either from bacteremia or extension of adjacent infection

Note: Information on this slide is adapted from Table 9 in full-text version of TAD Guidelines

Classification of Dissections

- Acute or chronic
- **Type A Dissections:** Dissections involving the ascending aorta.
- **Type B Dissections:** Dissections involving the descending thoracic aorta. These dissections begin distal to the left subclavian artery.
Clinical Presentation

Chest or back pain with variation in upper extremity blood pressure is key assessment finding in aortic dissection. Recurrent chest or back pain can indicate extension or rupture. The presence of aortic regurgitation in the setting of chest pain is also suspicious for aortic dissection.
Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Conditions

- Marfan Syndrome
- Connective tissue disease*
- Family history of aortic disease
- Known aortic valve disease
- Recent aortic manipulation (surgical or catheter-based)
- Known thoracic aortic aneurysm
- Genetic conditions that predispose to AoD†

* Loeys-Dietz syndrome, vascular Ehlers-Danlos syndrome, Turner syndrome, or other connective tissue disease.

† Patients with mutations in genes known to predispose to thoracic aortic aneurysms and dissection, such as FBN1, TGFBR1, TGFBR2, ACTA2, and MYH11.

Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Pain Features

Chest, back, or abdominal pain features described as pain that:
- is abrupt or instantaneous in onset.
- is severe in intensity.
- has a ripping, tearing, stabbing, or sharp quality.
Estimation of Pretest Risk of Thoracic Aortic Dissection

High Risk Examination Features

- Pulse deficit
- Systolic BP limb differential > 20mm Hg
- Focal neurologic deficit
- Murmur of aortic regurgitation (new or not known to be old and in conjunction with pain)

Auscultatory Areas

1. Aortic area (2RSB)
2. Pulmonary area (LSB)
3. Epic's point (LSB)
4. Tricuspid area (LSB)
5. Mitral area (LSMCL)
Diastolic Murmurs
Aortic Regurgitation

- **Timing:** Early diastole
- **Location:** Aortic area
- **Radiation:** Toward apex
- **Configuration:** Decrescendo
- **Pitch:** High
- **Quality:** Blowing

Austin Flint Murmur

- Very severe chronic AR or acute AR
- Diastolic murmur: functional mitral stenosis
- Severe AR
  - blood flow back through the aortic valve
  - regurgitant volume presses on open anterior leaflet of mitral valve
  - moves the leaflet towards the closed position
  - functional Mitral Stenosis
- **Timing:** Mid diastolic
- **Location:** Cardiac apex
- **Configuration:** Plateau
- **Pitch:** Low pitch
- **Quality:** Rumbling
- **Intensity:** Soft
### Risk-based Diagnostic Evaluation: Patients with High Risk of TAD

Patients at *high-risk* for TAD are those that present with at least 2 high-risk features (outlined in more detail in the following slides).

The recommended course of action for high-risk TAD patients is to seek immediate surgical consultation and arrange for expedited aortic imaging.

<table>
<thead>
<tr>
<th>Expedited aortic imaging</th>
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<tbody>
<tr>
<td>• TEE (preferred if clinically unstable)</td>
</tr>
<tr>
<td>• CT scan (image entire aorta: chest to pelvis)</td>
</tr>
<tr>
<td>• MR (image entire aorta: chest to pelvis)</td>
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### Diagnosis

- **Transthoracic echocardiography and transesophageal echocardiography:** A visible intimal flap separates the true and false lumens. Echocardiography is most helpful with type A dissections. Transesophageal echocardiography is very valuable and can be performed bedside.
Diagnosis

CT: CT is particularly helpful in evaluating dissections of the thoracic aorta.

MRI: MRI is an excellent diagnostic tool but has limitation because it cannot be used emergently or in critically ill patients.
Complications of Dissection

- Aortic regurgitation from retrograde dissection involving aortic valve or from aortic dilatation.
- MI from retrograde coronary artery dissection.
- Cardiac tamponade from ascending aorta or aortic arch rupture.
- Intrapericardial rupture from descending aortic dissection ruptures into intrapericardial space – most commonly left sided.
- Retroperitoneal bleed from rupture of abdominal aorta dissection.
- Stroke from brachial artery compromise.
- Paraplegia, reduced blood flow to kidneys, bowels, and lower extremities from compromise of arterial branches.

Recommendations for Initial Management

Initial management of thoracic aortic dissection should be directed at decreasing aortic wall stress by controlling heart rate and blood pressure as follows:

- In the absence of contraindications, intravenous beta blockade should be initiated and titrated to a target heart rate of 60 beats per minute or less.
- In patients with clear contraindications to beta blockade, nondihydropyridine calcium channel–blocking agents should be used as an alternative for rate control.
c. If systolic blood pressures remain greater than 120 mm Hg after adequate heart rate control has been obtained, then angiotensin-converting enzyme inhibitors and/or other vasodilators should be administered intravenously to further reduce blood pressure that maintains adequate end-organ perfusion.

d. Beta blockers should be used cautiously in the setting of acute aortic regurgitation because they will block the compensatory tachycardia.

Recommendations for Initial Management (continued)

Vasodilator therapy should not be initiated prior to rate control so as to avoid associated reflex tachycardia that may increase aortic wall stress, leading to propagation or expansion of a thoracic aortic dissection.

Base treatment goals on highest blood pressure reading.
Treatment of Dissections

Type A
- Surgery required
- Deep hypothermic circulatory arrest
- Distal false channel not completely eliminated
- Risk for bleeding
- Replacement of aortic valve / reimplantation of coronary arteries
- Hypertension management
- Surgical considerations similar for ascending aortic aneurysms
- Surgical mortality 15-20%
Treatment of Dissections

- Type B
  - Typically treated medically
  - Higher surgical risk candidates
  - Surgical considerations similar to those for descending thoracic aneurysms
  - Surgery limited to complicated dissections:
    - Persistent pain
    - Uncontrolled hypertension
    - Evidence of expansion or rupture
    - Circulatory compromise to visceral, renal, or lower extremity vessels and resultant organ ischemia.
    - 6 to 6.5 cm diameter in chronic dissections

Aortic Dissection: Not Just A Mimic
Hypertensive Crisis

Definition

Life threatening elevation in blood pressure requiring emergency treatment (within 1 hour) to prevent end organ damage or death.

End organs:
- Heart
- Brain
- Kidneys
Signs and Symptoms

- BP > 250/150 mmHg
- Retinopathy
- Papilledema of optic disc
- Vomiting
- Severe headache
- Altered LOC
- Seizures
- S&S of heart failure

Hypertensive Encephalopathy

- BP > 250/150 mmHg
- Loss of cerebral autoregulation
  - Vasospasm
  - Ischemia
  - Increased capillary pressure
  - Cerebral edema – hemorrhage
    - Basal Ganglia
### Etiology

- Uncontrolled hypertension
- Renal dysfunction
- Preeclampsia
- Adrenergic crisis
  - Drug reactions
  - Pheochromocytoma
- Post operative complication
- Pituitary tumor
- Adrenocortical hyperfunction
- Severe Burns

### Treatment

- MAP lowered no more than 25% in first 2 hours or to 160/100 mmHg
- Nitroprusside is gold standard (contraindicated in pregnancy)
- 0.25 to 0.5 mcg/kg/min – titrate every 5 minutes
- Maximum dose 8-10 mcg/kg/min
- Immediate onset – lasts 1-5 minutes
- Thiocyanate toxicity after 48 hours or with renal insufficiency
  - Blurred vision
  - Tinnitus
  - Seizures
Treatment

Other agents
- Fenoldopam (dopaminergic agonist)
- Alpha blocking agent (or combination alpha and beta blocking agent)
- ACE inhibitors

Venous Thromboembolism
Risk Factors / Pathophysiology

- Virchow Triad
  - Venous stasis
  - Vessel wall injury
  - Hypercoagulability

- Activation of coagulation in areas of reduced flow

- Before thrombus becomes organized and adherent (5-10 days) it can extend proximally or embolize
  - Extension can damage venous valves / can cause venous outflow obstruction

- Chronic venous insufficiency can result from incomplete clot lysis and recanalization
  - Can lead to venous ulceration

Hospitalized patients incidence ranges from 20-70%

- PE from DVT is leading cause of preventable hospital mortality
  - 20% of untreated proximal DVT will result in PE
  - 10-20% of these PE will be fatal
  - Anticoagulation decreases mortality 5 to 10 fold

- Bedside assessment is not sensitive for diagnosis
  - Cannot make accurate diagnosis based on signs and symptoms
Signs and Symptoms

- Signs and symptoms related to degree of venous obstruction and inflammation of vessel wall
- Many patients asymptomatic
- Edema (unilateral)
- Reddish purple hue from venous obstruction
- Tenderness
  - Calf muscles
  - Along deep vein medial thigh
- Homan’s sign
  - Present < 1/3 patients with DVT
  - Present in 50% patient without DVT
  - Non specific
- Venous Distention
  - Patients with thrombophlebitis can have co-existing DVT
  - Fever (low grade)

Signs and Symptoms

- Clinical signs of PE
  - May be primary manifestation in small percent of patients
- Phlegmasia cerulea dolens
  - Cyanosis from massive ileofemoral venous obstruction
- Phlegmasia alba dolens
  - Painful white inflammation from massive ileofemoral venous obstruction with associated arterial vasospasm
  - Pain and white discoloration can occur with acute arterial occlusion
  - Swelling, petechiae, and distended superficial veins can be used to differentiate
Specific Risk Factors

- **General**
  - Older age
  - Immobility > 3 days
  - Pregnancy / post partum
  - Major surgery within 4 weeks
  - Long plane or car trips (> 4 hours) within 4 weeks

- **Drugs**
  - IV drug use
  - Oral contraceptives
  - Estrogen HRT

- **Medical conditions**
  - Cancer
  - Stroke
  - MI
  - HF
  - Sepsis
  - Nephrotic syndrome
  - Ulcerative colitis
  - Previous DVT

- **Trauma**
- **Vasculitis**
- **Hematologic factors that result in hypercoagulability**

Diagnosis

- **D-dimer antibodies**
- D-dimer assays are not specific to DVT
  - Can be helpful to rule out
  - Cannot be used to rule in
- Remain elevated in DVT for 7 days
- There are multiple different D-dimer assays

- **A negative D-dimer can be used to rule out in patients with low to moderate risk**
- All patients with + D-dimer or patients with moderate to high risk require duplex ultrasound
Diagnosis

- D-dimer and risk scoring are used to determine need for further diagnostic testing
- Objective testing necessary before anticoagulation
- Noninvasive studies are first line over contrast venography
- Diagnostic criteria with Duplex ultrasound: failure to compress vascular lumen (due to thrombus)
  - Overall specificity 95%
  - Most inaccurate in diagnosis of calf vein thrombosis

CT venography

- Incorporated into CT of chest for PE
- Can detect venous occlusion proximal to inguinal ligament (ileofemoral DVT)

Summary:

- Ambulatory patients: Duplex ultrasound is diagnostic test of choice
- CT venography: Best for suspected ileofemoral DVT
- Noninvasive studies, D-dimer and patient risk score are used in determining diagnosis and decision to treat
Goals of Treatment

- Prevent PE
- Prevent postphlebitic syndrome
- Reduce risk of other complications

Note: DVT of calf rarely causes significant PE.

Heparin (UFH / LMWH)

- **Heparin**
  - Prevents extension of thrombus
  - Reduces incidence of fatal and non fatal PE
  - No effect on pre-existing non adherant thrombus
  - Does not significantly reduce risk of post phlebitic syndrome
  - Primarily caused by initial thrombus

- **LMWH has advantages over UFH**
  - Better bioavailability
  - Less monitoring
  - Lower incidence of HIT
  - Lower incidence of osteoporosis

Full anticoagulation may not be indicated in calf DVT.
Fondaparinux

- Synthetic Factor Xa inhibitor (Fondaparinux (Arixtra))
  - Daily SQ injection – fixed dose for most body weights
  - Effective and as safe as BID weight based enoxaprin
  - Also cleared renally (contraindicated in several renal impairment)
    - Creatinine clearance < 30 mL/min
  - No therapeutic monitoring required
  - No reported HIT
  - No specific antidote

Fibrinolytic Therapy

- Advantage
  - Prevention of PE
  - Restoration of normal venous circulation
  - Preservation of venous valves
  - Prevention postphlebitic syndrome

- Limitations
  - Does not prevent clot propagation, rethrombosis, or potential embolization
  - Not effective once thrombus is adherent and organized
  - If clot is large, the drug may not be able to penetrate entire clot
  - Most patients have contraindications

Only indicated in massive ileofemoral thrombosis with vascular compromise.

Heparin and oral anticoagulation always follow fibrinolytic therapy
Catheter Directed Thrombolysis

- Delivers higher drug concentration at the site with lower total dose compared to systemic administration
- Indicated in acute ileofemoral DVT
  - Adequate recannulization less likely in this location with anticoagulation alone
  - Collateral flow is limited
  - Ileofemoral DVT has higher rate of post thrombotic syndrome than other locations

Thrombectomy is an option in massive ileofemoral thrombosis with vascular compromise when thrombolysis is contraindicated.

Wafarin

- Warfarin is initiated at 5 mg daily
- Overlapped with anticoagulation therapy x 5 days until INR is therapeutic
- Treatment of 3 to 6 months for 1st episode
- Treatment of minimum of one year for recurrent episodes
Inferior Vena Cava Filter

- Greenfield filter
  - Enhances thrombolysis of trapped emboli
  - 98% long term patency
  - 4% recurrent PE
  - Can increase later risk of DVT
- Retrievable filters are also available

- Indications
  - Contraindication to anticoagulation
  - Failed anticoagulation

- Filters can be placed under transabdominal or intravascular ultrasound

Other Treatments

Graded Compression Stockings

- Post thrombotic syndrome affects up to 50% of DVT patients after 2 years
- High risk
  - Elderly
  - Recurrent ipsilateral DVT
- Graduated compression stockings (ankle pressure 30-40 mmHg)
  - Reduces risk of post thrombotic syndrome by 50%

Ambulation

- Early ambulation (day 2 after initiation of outpatient anticoagulation) is indicated
- Early ambulation is only indicated when done in conjunction with wearing graduated compression stockings
- Early ambulation results in a decrease in swelling and pain
- May also result in a decrease in post thrombotic syndrome
Inpatient versus Outpatient Therapy

- Most patients with proximal vein DVT can be safely managed with outpatient anticoagulation
- Isolated calf vein DVT may only be treated with NSAIDs or ASA with follow up
- Patients with risk factors need to be re-evaluated at one week.
- Patients with calf DVT (not on anticoagulation) need to re-evaluated in one week to assure DVT has not advanced into the proximal venous system

- Patients needing inpatient treatment
  - Suspected or actual PE
  - Ileofemoral DVT
  - CV or pulmonary comorbidities
  - Familial coagulopathies or bleeding disorders
  - Pregnancy
  - Morbid obesity
  - Renal failure
  - Contraindication to anticoagulation
  - Homeless or other social issues of non compliance

Primary Prevention

- Prophylaxis in all high risk patients
  - One large trial: 40 mg daily enoxaparin achieved 63% reduction in DVT in medical patients (Cohen, et al., 2001)

- Women’s Health Study: Vitamin E supplementation reduced the risk in women
Complications

- PE or other systemic embolization despite anticoagulation
- Soft tissue injury from very large thrombus
- Bleeding complications from anticoagulation
- Chronic venous insufficiency
- Post phlebitic syndrome
  - Pain
  - Edema
  - No new thrombus

Cardiac Trauma

- Blunt
  - Cardiac Contusion
    - S & S as ischemia / injury
    - ECG changes may be delayed
    - Dysrhythmias
  - Severe
    - Delayed free wall rupture
    - VSD
    - Ventricular aneurysm
  - Cardiac Rupture
    - Right ventricle most common
    - Surgery required
  - Valvular Injury
    - Aortic valve most affected
    - Aortic regurgitation / CHF

- Blunt / Penetrating
  - Tamponade
    - * See tamponade notes discussed in CV pathophysiology class with inflammatory disease.
Closed (Simple) Pneumothorax

- Air enters the intra pleural space through the lung causing partial or total collapse of the lung
  - Between visceral and parietal pleura
- **Possible etiology**
  - Primary (no underlying lung disease)
    - Blebs / bullae
    - Smoking
  - Secondary (underlying lung disease)
    - Air enters damaged alveoli
    - COPD
  - Blunt trauma (lung laceration by rib fracture)
  - Positive pressure ventilation (rupture of weak alveoli, bleb or bullous)
  - Iatrogenic – from medical procedure

**Pathophysiology**
- Disruption of normal negative intrapleural pressure
- Lung collapse
  - Decreased vital capacity
  - Decreased surface area for gas exchange
  - Acute respiratory failure (particularly secondary)

**Signs and Symptoms**
- Chest pain, dyspnea, cough, tachycardia
- Asymmetrical chest excursion, diminished to absent breath sounds on affected side, dramatic increase in peak inspiratory pressures on a mechanical ventilator

**Treatment**
- Oxygen
- Analgesics
- Observation (asymptomatic, small primary)
- Aspiration (symptomatic, small primary)
- Chest Tube Criteria
  - Secondary

Spontaneous primary pneumothorax may take 12 weeks to resolve.
Tension Pneumothorax

Pathophysiology
- Air rushes in-cannot escape pleural space
- Creates positive pressure in pleural space
- Ipsalateral lung collapse
- Mediastinal shift→contralateral lung compression→potential tearing of thoracic aorta
- Can also compress heart→decrease RV filling→shock

Tension Pneumothorax

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Similar to closed pneumothorax</td>
<td>Oxygen (100%)</td>
</tr>
<tr>
<td>If mediastinal shift:</td>
<td>Emergency decompression</td>
</tr>
<tr>
<td>- Tracheal shift away from affected side</td>
<td>Chest Tube</td>
</tr>
<tr>
<td>- JVD</td>
<td>Other as with closed pneumo</td>
</tr>
<tr>
<td>- Hypotension</td>
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</tbody>
</table>

Oxygen (100%)
Emergency decompression
Chest Tube
Other as with closed pneumo
Open Pneumothorax

Air enters the pleural space through the chest wall

**Etiology**
- Penetrating Trauma

Pathophysiology and Signs and Symptoms
- Equilibrium between intrathoracic and atmospheric pressures
- Patient condition depends on size of opening compared to trachea
- The affected lung collapses during inspiration
- May cause a tension pneumothorax
- Subcutaneous emphysema usually present

Treatment
- Similar to closed pneumothorax
- Closure of open wound with petroleum jelly gauze
  - End expiration
  - Modification for tension pneumothorax
- Chest tube and water seal drainage
Chest Tubes

First compartment allows for collection of fluid from the pleural space.
- Mediastinal chest tubes are placed when mediastinal incision is used for cardiothoracic surgery
- Pleural chest tubes are placed for hemothorax
- Standard chest tubes are 32 F
- **Assessment of chest tube patency is a key nursing function**
- Gently milk to prevent formation of clots.
- Aggressive stripping causes a negative 300 cm H₂O pressure in mediastinum and may increase bleeding.
- Assess color of drainage to determine if arterial or venous
- Dumping of blood with a position change may indicate an acute onset of bleeding (if dark in color and minimal additional drainage then note acute)
Chest Tubes Drainage

- Avoid dependent loops in the drainage tubing
- Decreased breath sounds, increased inspiratory pressures on the ventilator, or widening of the mediastinum on CXR; suspicion for undrained blood in the pleural space or in the mediastinum
- **Chest tubes should always be assessed for evidence of hemorrhage when there is a low blood pressure**
- Assess for excessive bleeding requiring surgical intervention.
  - Drainage > 1500 ml in a short period of time represents approximately 40% of the circulating volume
- Can lead to tamponade. Signs include:
  - Rising filling pressures with a decreased cardiac output and hypotension
  - CVP, PAOP, and PAD pressures that are equal
  - Sudden drop/stop in previously significant mediastinal bleeding
  - Narrowing of pulse pressure.
  - Classic pulsus paradoxus is not a sign of tamponade in the ventilated patient because positive pressure ventilation reverses the blood pressure response to respiration
  - Tachycardia, dysrhythmias, and decreased ECG voltage
  - Transthoracic or transesophageal echo used in the diagnosis

Chest Tube: Water Seal

- 2nd compartment is connected to the 1st and creates a water seal
  - Water seal allow airs to exit from the pleural space on exhalation and prevent air from entering the pleural cavity or mediastinum on inhalation
  - To maintain an adequate water seal it is important to monitor the level of water in the water seal chamber and to keep the chest drainage unit upright at all times.
  - Assess the water seal chamber for slight fluctuation (tidaling)
    - Tidaling (rising during spontaneous inspiration and falling during expiration) is normal
    - Lack of fluctuation with respiration may indicate kinking or other problems interfering with drainage.
    - May also be a good sign indicating lung re-expansion.
  - Assess for air leak by checking water seal chamber for bubbles during inspiration.
    - May bubble gently with insertion, during expiration and with a cough.
    - Continuous bubbling represents an air leak.
    - Check for system leaks by clamping before each connection (system may need to be replaced).
    - Check for leak where tube enters chest.
    - Check chest x-ray to assure last hole of chest tube is inside chest.
Chest Tubes: Suction

- 3rd compartment is connected to the 1st 2 sections and provides suction.
  - Traditional units regulate the amount of suction by the height of a column of water in the suction chamber.
  - A system valve controls the amount of suction.
  - -20 cm H₂O of suction is the typical amount applied (lower levels may be indicated for patients with friable lung tissue)
  - Goal is to have an adequate amount of suction to keep open the pleural space tissue
  - No more than -40 cm H₂O should be applied to a chest tube drainage system
  - The column of fluid limits the amount of suction. Adjust the source suction to produce only gentle bubbling in the suction control chamber.
  - Excessive external suction causes loud bubbling and also increases the evaporation of water from the suction control chamber.

Chest Tube: Other Nursing Considerations

- Assess the insertion site for subcutaneous emphysema.
- Keep unit below the level of the patient’s chest
- Do not clamp chest tube for transport (can cause tension pneumothorax with pleural chest tubes or tamponade with mediastinal chest tubes)
  - Use portable suction or transport on gravity drainage with tubing from suction chamber open to air
Chest Tubes

- Complications can include infection and bleeding.
- Rare but serious complication is the development of unilateral pulmonary edema in response to rapid re-expansion or rapid evacuation of pleural fluid.
  - Capillary permeability can increase as result of rapid treatment, resulting in pulmonary edema.
- Strategies for prevention:
  - Avoiding suction with the initial drainage of a large pleural effusion or the expansion of a large pneumothorax.
  - Clamping after the 2 liters of initial drainage.

Chest Tubes: Removal

- No air leak on water seal for 24 hours (no continuous bubbling in water seal).
  - Lack of fluctuation in the water seal can indicate that the lung has reexpanded.
- Drainage < 50-100 ml for 24 hours.
  - Post cardiac surgery chest tubes may be removed when drainage is < 100 ml in 8 hours.
- Complete expansion of lung confirmed by chest x-ray.
- Improvement in respiratory status.
- Use of gauze pad and asking the patient to take a deep breath and hold (performing the Valsalva maneuver).
  - This creates a positive pressure in the pleural space and prevents the development of a pneumothorax during removal.
- The use of an occlusive dressing (petrolatum gauze) with pleural chest tube removal prevents the entry of air into the pleural space.
- Traditional method of removal involves removing at the end of inspiration.
- Chest x-ray typically several hours after chest tube removal.
Success through Collaboration

Questions?

Open Discussion