Peripheral Arterial Disease (PAD)

Arteriosclerosis Obliterans (ASO)
Aneurysmal Disease
Acute Arterial Occlusion
Thromboangiitis Obliterans (Buergers Disease)
Arteriosclerosis Obliterans (ASO)

Overview:
- Manifestation of atherosclerosis
- 95% of chronic occlusive arterial disease
- Generalized disease of aorta and branches
- Slow progression
ASO

Demographics:

- Age > 50 years old
- Male > female x 2
- 1/3 have clinical CAD
- > 50% have severe CAD by cath
- Up to 1/3 = diabetic
- ¼ have significant carotid stenosis
ASO

Risk Factors:
- Diabetes, smoking, HTN, Lipids, Family hx PAD
- Diabetes & Smoking = highest rel. risk

Pathophysiology:
- Atheromatous plaque
  - arterial narrowing
  - complete arterial occlusion (due to plaque or thrombus)
- Tissue ischemia, collaterals
ASO

Clinical

- Claudication = muscle ischemia
  - aorto-iliac disease = hips, thighs, buttocks
  - femoral - popliteal disease = lower leg
  - popliteal - tibial disease = foot

- Rest Pain

- Ulceration
  - foot, toes = most common
  - painful
  - may progress to gangrene

- Acute Occlusion = thrombus, embolism
ASO - Clinical

* **Pulses** = location of decreased pulse clue to site of stenosis

* **Ankle-Brachial index** = ratio of DP or PT / highest brachial systolic BP

  1.0-1.4 = normal
  0.91-0.99 = borderline
  0.4 - 0.9 = abnormal
  \( \leq 0.4 \) = severe, indicates ↑ risk of development of critical limb ischemia

*NB = may not correlate with symptoms

**Bruit**s = not indicative of degree of stenosis
ASO - Clinical

v Skin color changes
  § pallor with elevation
  § reactive hyperemia (rubor) and delayed venous filling (>15 sec) with dependency

v Trophic changes:
  § hair loss; dry, scaly skin
  § cool foot
  § ± chronic hyperemia

v Ulcers: spontaneous, post-traumatic
  § pale, painful, irregular border
ASO – diagnostic tests

ABI’s = very sensitive (~90%) and specific (>95%) in detecting presence of PAD using 0.9 as a cut-off

Exercise ABI’s = may help to differentiate claudication from pseudoclaudication and to assess functional status in patient’s with PAD

Ultrasound = simple, inexpensive, location and severity, f/u

CTA = pre-intervention
MRA = pre-intervention

Contrast Angiography = at time of intervention
ASO

v Treatment

- 75% = stable course
- 25% = progressive
  * ¼ = amputation
- Critical Limb Ischemia (rest pain, tissue loss) = prompt eval for revasc
- Morbidity / Mortality = CAD, stroke
### ASO

#### Treatment:

- **Asymptomatic** = **risk factor mods** (including statin tx), ± anti-platelet rx (class IIa), +/- ACEI, long term f/u

- **Mild to moderate disease**
  - medical treatment = risk factor mod., ASA or clopidogrel (class I), Cilostazol (may improve sx, avoid with CHF/LV dysfunction)
  - cornerstone = walking (supervised vs structured home program 30-45 min ≥ 3x/week)

- **Severe or rapidly progressive disease**
  - endovascular intervention, surgery
  - goal = symptom relief, functional improvement, limb salvage
Abdominal Aortic Aneurysm (AAA)

Overview:

- Defined = minimum A-P diameter ≥ 3cm.
- Etio. = atherosclerosis (90-95%), hereditary, inflam, infect, aortopathy (Marfans, Bicuspid AV)
- Prevalence
  - 2% of elderly
  - 10% at autopsy in males > 60 y.o.
  - males > females 5-8 x
- Most common arterial aneurysm
- 98% = infra-renal
AAA

* Risk Factors = male, age > 60, smoking, family hx, hx aneurysm in another artery

* Progression = enlargement 1-4 mm/yr. (<4cm AAA) to 7-8 mm/yr (large)

* Complications = rupture, thrombo-embolism, compression, erosion

*10% of patients with lower extrem ASO have an AAA = so screening reasonable for AAA (class IIa) in pts with sx PAD

  - NB: The pathophysiology of AAA is distinct from ASO
AAA

v Clinical:

§ Symptoms = with active enlargement or rupture
  § Abdominal or back pain
  § Less common
    § G-I bleeding (d/t erosion)
    § rupture into IVC
    § lower extrem. emboli

§ 90% ruptures = retroperitoneal

§ Ominous triad = abdominal/back pain, pulsatile abdominal mass, low blood pressure
AAA-Clinical

Physical Exam:

- incidental finding
- pulsatile mass = epigastric
- 25% = bruit
- tenderness = pending rupture
- rupture = “shock”
AAA

- Diagnostic tests:
  - Abdominal x-ray = calcific outline
  - Ultrasound = inexpensive, serial testing
  - CT Angiography or MRA = pre-op testing

- Who should be screened with US:
  - Male (and possibly Female) 65-75 and ever-smoked
  - Male (and possibly Female) >/= 60-65 yo with fam hx (1st degree relative) of AAA
  - Patient with aneurysm elsewhere
AAA

* Pre-op Evaluation
  ᵃ Pulmonary
  ᵃ Cardiac
    ᵃ Pharmacologic Nuclear Stress = ?
    ᵃ Cardiac Catheterization = ?
      ᵃ positive stress test
      ᵃ symptomatic

* Operative mortality = related to age and presence of CAD

* Leading cause of Peri-Op Death = CAD
AAA

* Monitoring

< 4 cm = US q 2-3 yrs
4 – 5.4 cm = US q 6-12 months

* Surgery or endovascular repair

§ Timing:

§ Diameter > 5.5 cm
§ Size/expansion > 0.5cm/6 mos or > 1 cm/yr
§ Symptomatic = emergent
§ Rupture = high mortality
Thoracic Aortic Aneurysm

Abnormalities of Aortic Media

Bicuspid Aortic Valve

Marfan’s

Turner’s Synd

Loey’s-Dietz Synd

Familial/non-syndromic

screen pt. and 1st degree relatives
Popliteal Artery Aneurysm

70% of all lower extrem aneurysms
Can be bilateral
↑ Risk of AAA
Complications:
  * distal emboli
  * thrombosis in-situ
  * rupture = uncommon
Surgical repair = symptoms or > 2 cm dia
Acute Arterial Occlusion

Etiology = embolic, thrombotic, traumatic

Embolic =

Cardiac origin = 80-90%
  Afib = 75%
  LV thrombus
  Endocarditis
  Left atrial myxoma

Non-Cardiac
  Aortic plaque ulceration or disruption
  Embolus from aneurysm
  Paradoxical venous thrombo-embolism
Acute Arterial Occlusion

v Etiology (continued):

§ Thrombotic =

§ ASO

§ Lower extrem. Aneurysm (with thrombosis in-situ)

§ Buergers disease/Arteritis

§ Local trauma

§ Hypercoagulable states
Acute Arterial Occlusion

Clinical = 6 - “P”s

- Pain = rapid onset
- Polar (poikilothermia) = cold limb
- Pallor = with venous collapse
  - progression to bluish mottling = tissue ischemia and necrosis
- Pulseless =
  - Thrombus propagation with time
- Paresthesia = ischemic neuropathy
  - May progress to complete loss of sensation and motor function
- Paralysis = ischemic nerve injury and muscle rigidity
  - chance of limb salvage ↓
Acute Arterial Occlusion

v Pathophysiology:

§ Release of $K^+$, myoglobin, lactic acid = hyperkalemia, acidosis, renal failure

§ Flow restoration = wash-out of $K^+$ and lactic acid, arrhythmia, hemo. Instability, post-revasc compartment syndrome.
Acute Arterial Occlusion

Differential Diagnosis =

- Phlegmasia cerulea dolens = acute extensive DVT
  - Lower extrem. cyanosis, acute swelling, edema, leg vein distension, ↓ or absent pulses
- Ergotism
- Acute aortic dissection
- Low Cardiac - Output State in patient with pre-existing ASO/stenosis = ↓ perfusion pressure
Acute Arterial Occlusion

**Treatment =**

- Heparin = prevent thrombus propagation
  - * Unless category III / irreversible = amputation
- Rapid Evaluation and Revascularization = endovascular, surgery

- After acute tx: Evaluate for an embolic source if suspected.
Thromboangiitis Obliterans – Buerger’s Disease

- Pathophysiology = inflam. occlusive disease of small and medium size peripheral arteries and veins in **young male smokers**
- Age of onset = < 50 y.o./freq. < 30 y.o.
- Absence of Risk Factors = except smoking
- > 90% male
- Prevalence mid and far-east
Buerger’s Disease

- Etiology = uncertain, but...
  - Smoking is related to progression or remission
  - Cessation of smoking = improved prognosis
  - Continuation of smoking = disease progression
  - Ischemic symptoms = distal extremities.
Buergers Disease

Clinical =

- Claudication
  - instep of foot
  - hand = “writers cramp”

- Numbness/paresthesias, Raynauds

- Ulceration, gangrene

- Thrombophlebitis = superficial or deep
  - classically migrating and transient (“phlebitis migrans”)

Buerger’s Disease

Treatment

- STOP SMOKING
- Surgical revascularization = generally dismal long term results
- Sympathectomy (thoracic, lumbar) = symptom relief
- Amputation = gangrene, severe infection, debilitating pain
- “Growth Factor” angiogenesis = ?