

2018

ACOI

Internal Medicine Board Review

Peripheral Vascular Disease

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Peripheral Vascular Disease (PVD)

Arteriosclerosis Obliterans (ASO)

Aneurysmal Disease

Acute Arterial Occlusion

Thromboangiitis Obliterans (Buerger's
Disease)

Arteriosclerosis Obliterans (ASO)

v Overview:

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

ASO

v 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
- § 1/4 have significant carotid stenosis

ASO

v Risk Factors:

- § Diabetes, smoking, HTN, Lipids, ↑ homocysteine
- § Diabetes & Smoking = highest rel. risk

v Pathophysiology:

- § Atheromatous plaque



arterial narrowing



complete arterial occlusion
(due to plaque or thrombus)

- § Tissue ischemia, collaterals

ASO

v 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
 - ≤ 0.4 = severe, indicates \uparrow risk of development of critical limb ischemia
 - *NB = may not correlate with symptoms

Bruits = 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
 - § \pm 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

* 1/4 = amputation

§ Critical Limb Ischemia (rest pain, tissue loss) = prompt eval for revasc

§ Morbidity / Mortality = CAD, stroke

ASO

v 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 (not with CHF/LV dysfunction)
 - § cornerstone = walking (supervised vs home program 30-45 min \geq 3x/week)

- § Severe or rapidly progressive disease
 - § endovascular intervention, surgery
 - § goal = symptom relief, functional improvement, limb salvage

Abdominal Aortic Aneurysm (AAA)

v Overview:

- § Defined = minimum A-P diameter \geq 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 = HTN, smoking, ?familial
- * 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

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

v Physical Exam:

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

AAA

v Diagnostic tests:

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

AAA

v Pre-op Evaluation

- § Pulmonary

- § Cardiac

 - § Nuclear Stress/Persantine

 - § Cardiac Catheterization

 - § positive stress test

 - § symptomatic

v Operative mortality = related to age and presence of CAD

v 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 \geq 5.5cm

§ Size > 0.5cm/6 mos.

§ 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

v 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)

§ Buerger's disease/Arteritis

§ Local trauma

§ Hypercoagulable states

Acute Arterial Occlusion

v 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

v 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

v Treatment =

§ Heparin = prevent thrombus propagation

* Unless category III / irreversible = amputation

§ Rapid Evaluation and Revascularization =
endovascular, surgery

Thromboangiitis Obliterans – Buerger's Disease

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

Buerger's Disease

v Etiology = uncertain, but...

- § Smoking is related to progression or remission
- § Cessation of smoking = improved prognosis
- § Continuation of smoking = disease progression
- § Ischemic symptoms = distal extremities.

Buerger's Disease

v 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

v Treatment

§ STOP SMOKING

§ Surgical revascularization = generally dismal long term results

§ Sympathectomy (thoracic, lumbar) = symptom relief

§ Amputation = gangrene, severe infection, debilitating pain

§ “Growth Factor” angiogenesis = ?