

## Peripheral Arterial Disease

### PAD

- Chronic occlusive disease that limits blood flow of arteries into the lower extremity
- Caused by arteriosclerosis
- Initially asymptomatic, but progresses to:
  - Intermittent claudication
  - Severe ischemia
    - Leads to venous stasis, ulcers, gangrene, loss of limbs, etc (things you see in diabetic patients)
- More than 50% affected are asymptomatic
- Many of those with symptoms exhibit lower extremity symptoms ONLY with exercise (intermittent claudication)
- Severe ischemia will exhibit
  - Pain at rest
  - Ulcerations
  - Gangrene

### PAD Risk Factors

- Similar to those seen with atherosclerosis and CAD
  - Smoking
    - Greater risk for endothelial damage
  - Hyperlipidemia
    - Leads to HTN
  - Hypertension
  - Diabetes Mellitus
  - Positive family history
- People with lower extremity atherosclerosis have a significant atherosclerotic disease elsewhere
  - Coronary artery disease
  - Cerebrovascular disease

### Presentation

- Location of symptoms is variable
  - Buttock and thigh (*saddle like distribution*)
  - Calf
  - Foot
- Two characteristic types of pain
  - Intermittent claudication
  - Ischemic ...

### Differential Considerations

- Must differentiate from
  - L spine DJD with radiculopathy
    - Aching/burning in buttock, thigh and calf
    - Mechanical maneuvers should be provocative
  - Venous stasis
    - Aching in calves/ankles
    - Dangling legs increases pain
    - Thermal change in LE
  - Peripheral neuropathy
    - Dysesthesias or burning LE pain
    - No change with dangling legs

### Management

- Prevention by eliminating risk factors
- Regular exercise
  - *Best thing a person can do*

### ★ Vascular claudication vs neurogenic claudication

To differentiate have pt walk until calves cramp up, then have them keep walking but bend forward and put hands on knees.  
 If gets better = **neurogenic claudication**  
 If gets worse = **vascular claudication**  
 The two have very different treatments

### Pathology

- Slowly evolving process of damage to vessel walls
  - Seen at origins of branches from major vessels
  - Thought to be under greater stress, torsion, and turbulence at these points
- Stresses disrupt endothelium of vessel exposing deeper tissues
- Platelets adhere to exposed surface forming small thrombi
- Platelets release factors that promote migration of smooth muscle cells and macrophages to area
- These proliferate and generate matrix of lipids and connective tissue in region
- This matrix then thickens, stiffens, and develops cholesterol and calcific deposits
- These enlarge by damaging adjacent endothelium
- Surface of this region becomes rough and ragged, shedding thrombi and plaque into circulation
- Risk factors amplify vessel injury or disrupt healing process
  - HTN injures endothelium
  - Hyperlipidemia increases plaque formation
  - Smoking induces vasoconstriction and platelet aggregation

### Diagnostic Strategy

- ABI (*ankle arm index*)
  - Should be >1
  - If symptoms occur with exercise likely between .5 and 1
  - If symptoms are at rest, less than .5
- Duplex sonography
  - Color doppler measures vessel lumen
- MRA
  - Non invasive used prior to surgery

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- *Have them walk until it hurts, stop for a min and then keep going for more steps until it hurts, then pause again, then keep going, etc*
- *This will re-perfuse the tissues*
- Eliminate tobacco use
- Normalize
  - Glucose
  - Lipids
  - Blood pressure

Anterior Choroidal and Lenticulostriate are branches of the middle cerebral artery which supplies the internal capsule which is the artery most commonly injured in hemiparetic stroke

#### What causes strokes?

- Vascular etiology is inferred by the abrupt onset of symptoms
- This is supported by
  - Patient age
  - Risk factors
    - Hypertension
    - Atherosclerosis
    - Smoking
    - Anything that alters blood viscosity risk for ischemic event
      - (Blood thinner is a risk for hemorrhagic event)
  - Occurrence of symptoms referable to the territory of a particular cerebral vessel

★ CT is study of choice for strokes in geriatric population

#### Onset

- Strokes begin abruptly
- Deficits may be max at the outset or may progress over seconds to hours (embolic vs. thrombosis vs. recurrent embolic)
- Active progression as result of an underlying vascular disorder or has done so in recent minutes is termed stroke in evolution or progressing stroke
- Focal cerebral deficits that develop slowly (over weeks to months) are suggestive of tumor, inflammatory or degenerative disease, not stroke

Venous bleed tamponades at diastole because venous blood (unlike arterial blood) can be shunted all over the place

#### Deficit Duration

- When symptoms/signs resolve completely in less than 24 hours, this is termed TIA
- Usually resolve in less than 30 minutes
- Recurrent TIAs with identical features are result of thrombosis or recurrent emboli from within cerebral circulation
- TIAs that differ in character from event to event suggest a cardiac source

#### Focal Involvement

- Hemorrhage produces a less predictable pattern because of complications like
  - Increased ICP
  - Cerebral edema
  - Brain compression and shift
  - Dispersion of blood through subarachnoid space and ventricles
- Anterior circulation disruption will produce symptoms indication hemispheric dysfunction like
  - Aphasia, apraxia, agnosia
  - Hemiparesis
  - Hemisensory disturbances
  - Visual field defects
- Posterior circulation disruption causes symptoms of brainstem dysfunction like
  - Coma, drop attacks
  - Nausea and vomiting
  - Vertigo
  - Ataxia
  - Cranial nerve palsies
  - Crossed sensorimotor deficits

Vertebral Basilar (posterior circulation) supplies "below the tent" and the occipital lobe  
Carotids (anterior circulation) supply cerebral hemispheres

