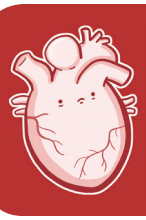

NOTES
PERIPHERAL ARTERY DISEASE

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Narrowing of the arteries in peripheral, non-coronary arterial circulation
- Vessels of the lower extremities are most commonly affected
- ↓ blood flow → arterial insufficiency → tissue ischemia
  - ↓ gas and nutrient exchange → tissue loss, ulcer formation → poor healing
  - Embolus formation → acute limb ischemia → tissue loss
  - Ischemic cells release adenosine → adenosine signals nerves → sensation of pain
  - *Claudication:* pain caused by poor circulation; occurs when oxygen demand is greater than oxygen supply
- Location of pain is dependent upon artery implicated
  - Lower aorta or iliac artery = pain in hips and buttocks
  - Iliac or common femoral artery = pain in thigh
  - Superficial femoral artery = pain in upper ⅔ of calf
  - Popliteal artery = pain in lower ⅓ of calf
  - Tibial or peroneal artery = pain in foot

TYPES

Occlusive (most common)

- Usually caused by blockage due to atherosclerosis
- Buildup of plaque → narrowed artery → ↓ blood flow

Functional

- Caused by a defect in the normal mechanisms that dilate and constrict arteries (e.g. inherited defects, injuries, certain drugs)
- Intermittent arterial constriction → ↓ diameter → ↓ blood flow

RISK FACTORS

- Smoking
- High blood pressure
- Diabetes
- Hyperlipidemia
- Metabolic syndrome
- Age > 60
- Obesity
- ↑ risk in black people of African descent

COMPLICATIONS

- ↑ risk of developing coronary artery cerebrovascular disease
- Tissue necrosis
- Amputation
- Pain

SIGNS & SYMPTOMS

- Often asymptomatic until significant occlusion develops
- Intermittent claudication
  - Muscle pain due to ↑ oxygen demand and ↓ supply
- Rest pain
  - Pain or burning sensation in forefoot and toes when legs elevated, pain relieved when legs are lowered (gravity assisting blood flow)
- ↓ lower peripheral pulses (e.g. pedal, tibial)
- Leg/foot ulcers that do not heal normally
  - Have classic punched out appearance
  - Often form on toe joints, malleoli, shin, base of heel, pressure points

    - Painful
    - Slow healing → ↑ risk of infection
- Cutaneous color changes
    - *Elevation pallor:* foot turns pale when raised due to circulation having to work against gravity as well as narrowed artery
    - *Dependent rubor:* foot turns red when lowered as gravity works increases perfusion
- Skin: cool, dry, shiny, hairless
- Nails: brittle, hypertrophic, ridged
- Signs of acute limb ischemia
    - See mnemonic

MNEMONIC: 5Ps

Signs of acute limb ischemia

Pain
Pallor
Pulselessness
Paresthesia
Paralysis (a surgical emergency)

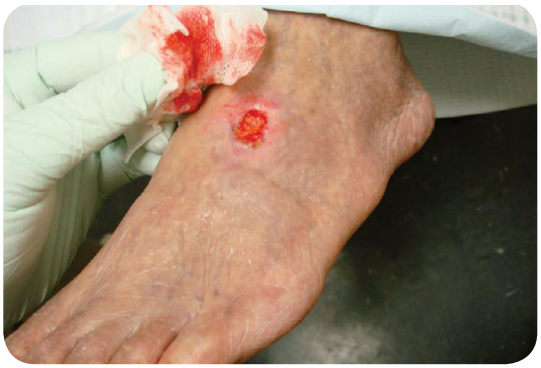

Figure 15.1 An arterial ulcer on the dorsum of the foot; a consequence of peripheral vascular disease. Note the punched out appearance.

DIAGNOSIS

DIAGNOSTIC IMAGING

Doppler ultrasound

- ↓ blood flow

OTHER DIAGNOSTICS

Auscultation

- Bruit (whooshing sound) heard on auscultation of suspected artery
    - Usually pulse of leg's iliac artery
    - Whooshing sound due arterial narrowing

Ankle-brachial index (ABI)

- *ABI* < 0.9: peripheral artery disease
- *ABI of* 0.4–0.9: claudication
- *ABI of* 0.2–0.4: rest pain
- *ABI of* 0–0.4: tissue loss, ulcers, gangrene

TREATMENT

MEDICATIONS

- Antiplatelet therapy

SURGERY

- Angioplasty, stent insertion
- Endarterectomy
- Bypass surgery to restore blood flow by diverting it around blockage
- Amputation

OTHER INTERVENTIONS

- Modify risk factors; e.g. smoking cessation, healthy eating habits, exercising regularly, managing diabetes
- Wound care

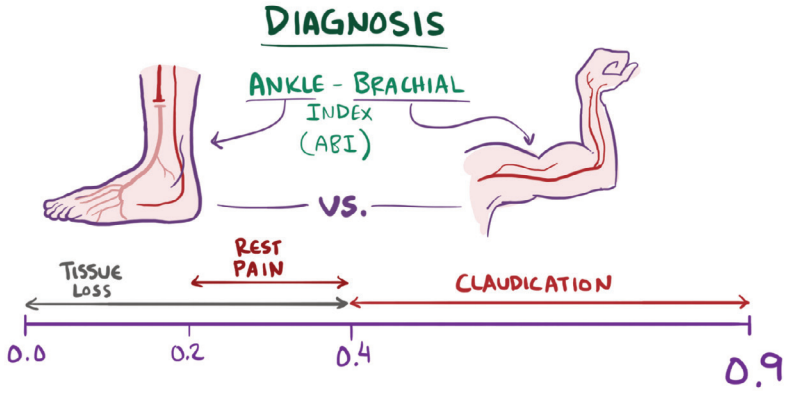

Figure 15.2 Illustration depicting the Ankle-branchial index.

ARTERIOLOSCLEROSIS

osms.it/arteriolosclerosis

PATHOLOGY & CAUSES

- *Arteriosclerosis:* a general term for diseases where the artery wall becomes thicker, harder, and less elastic
  - *Arteriolosclerosis:* a disease of the small arteries and arterioles characterized by stiffening and thickening of the vessel wall due to high blood pressure or diabetes, manifested primarily in the kidneys

TYPES

Hyaline arteriolosclerosis

- Accumulation of proteins and pink hyaline material → ↑ thickness and stiffening of vessel wall → ↓ compliance → ↓ blood flow → tissue hypoxia
  - Sustained high-pressure in vessels → serum proteins pushed into blood vessel walls → protein build-up in tunica media
  - Chronic high blood glucose → endothelial cells become glycosylated → endothelial dysfunction → basement membrane becomes "leaky" → serum proteins move into endothelial cells and build up into tunica media

Hyperplastic arteriolosclerosis

- Smooth muscle cell hyperplasia → very small lumen → ↓ blood flow → tissue hypoxia
  - Malignant hypertension → smooth muscle cells lining arteriole exposed to plasma proteins → concentric layers of smooth muscle cell proliferation ("onion-skinning")

RISK FACTORS

- Diabetes mellitus
- Chronic hypertension
- Malignant hypertension

COMPLICATIONS

- Arteriolonephrosclerosis
- Formation of intraluminal thrombi
- Chronic renal failure

SIGNS & SYMPTOMS

- Clinical manifestations of chronic kidney disease
  - Anemia (fatigue, activity intolerance, pallor)
  - Fluid and electrolyte imbalance (edema, muscle weakness, palpitations)
  - Uremia (anorexia, mental status changes)
  - Renal osteodystrophy

DIAGNOSIS

LAB RESULTS

- Signs of arteriolonephrosclerosis
- ↑ blood urea nitrogen
- ↑ creatinine
- ↓ hemoglobin
- ↓ hematocrit
- Proteinuria
- Oliguria

TREATMENT

OTHER INTERVENTIONS

- Management of diabetes and hypertension; support renal function

ATHEROSCLEROSIS

osms.it/atherosclerosis

PATHOLOGY & CAUSES

- *Arteriosclerosis:* a general term for diseases where the artery wall becomes thicker, harder, and less elastic
  - *Atherosclerosis:* atheromatous plaques on the tunica intima of large and medium vessels
- Damage to endothelium → low-density lipoproteins enter endothelial wall → LDL oxidation → uptake of LDL by macrophages → foam cell formation → cytokine and growth factor release from foam cells → formation of thrombogenic fatty streak → platelets release platelet-derived growth factor → migration of smooth muscle cells from vascular media to intima → fibrous cap → atherosclerotic plaque → chronic inflammation
- Calcium deposits into plaque → stiffening of arteries

RISK FACTORS

- Family history of coronary heart disease
- Smoking
- Hypertension
- Dyslipidemia; especially low HDL
- Metabolic syndrome
- Males ≥ 45; females 55 ≥ or premature menopause without hormone replacement therapy

COMPLICATONS

- Cardiovascular and coronary heart disease
  - Myocardial infarction, heart failure, death
- Cerebrovascular disease
  - Transient ischemic attack, stroke
- Peripheral artery disease
  - Leg ulcers, amputation
- Aortic aneurysm

SIGNS & SYMPTOMS

- Symptoms vary according to extent and location of blockage
- Carotid artery
  - Weakness, difficulty speaking, dizziness, difficulty walking, blurred vision, numbness of face/arms/legs, severe headaches
- Peripheral arteries
  - Claudication, presence of ulcers
- Coronary arteries
  - Angina
- Cerebral arteries
  - Auscultation of bruit, neurological complaints (e.g. visual changes, facial paresis)

DIAGNOSIS

- History and presence of clinical manifestations indicating occlusive disease

DIAGNOSTIC IMAGING

Angiography

- Vascular calcifications, stenosis, occlusion, collateral circulation

Ultrasound

- Luminal stenosis, atheromatous calcification (hyperechoic foci producing an acoustic shadow)

Magnetic resonance angiography

- Thickened arterial wall, heterogeneous signal within vessel wall (lipid rich necrotic core, plaque, fibrous cap)

LAB RESULTS

- hs-CRP (high-sensitivity C-reactive protein) test
  - ↑ CRP indicates "silent atherosclerosis" before cardiovascular event
- Fasting lipid profile

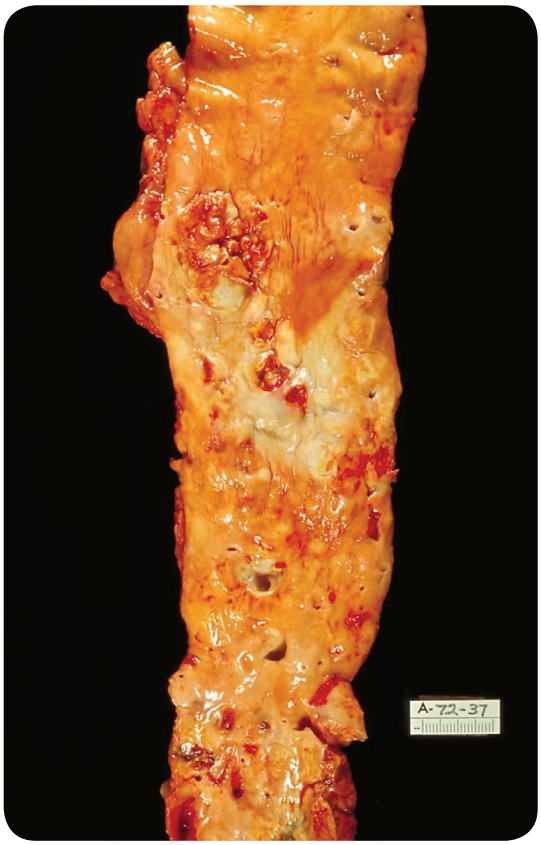

Figure 15.3 The abdominal aorta at post mortem showing moderate atherosclerosis.

TREATMENT

- *Goal:* reduce risk of complications with management risk factors; e.g. lipids, blood glucose, hypertension

MEDICATIONS

- Antiplatelets
- Antilipemic agents
- Antihypertensives

SURGERY

- *Complications:* stents, bypass grafts, angioplasty, carotid endarterectomy (CEA)

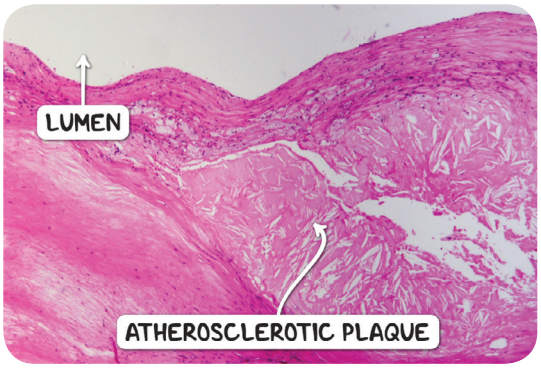

Figure 15.4 An atherosclerotic artery. Note how the plaque protrudes into the lumen. It is composed primarily of cholesterol with an outer rim of foamy macrophages.

PERIPHERAL ARTERIAL VS. VENOUS DISEASE

	ARTERIAL	VENOUS
PULSES	Diminished or absent	Normal
APPEARANCE	Ulcers: "punched out" and concentric; mostly on toes, heels, other bony prominences; minimal exudate; poor healing Skin: shiny, loss of hair, thick, brittle toenails Elevation pallor, dependent rubor	Ulcers: irregular borders; "stocking" distribution, most common near medial malleolus, significant exudate Skin: hemosiderin deposits cause brawny discoloration; stasis dermatitis and pruritis
TEMPERATURE	Cool	Warm
CAPILLARY REFILL	> 3 sec	< 3 sec
HARDNESS	Soft, thin skin	Hardened, leathery skin
EDEMA	Not present	present
SENSATION (PAIN)	Intermittent claudication Painful ulcers	Dull, achy pain relieved by elevation Painful ulcers

Mnemonic: PATCHES