Differentiating Leg Pain

Zaheed Tai, DO, FACC, FSCAI
Leg Pain Has a Differential Diagnosis

- Spinal canal stenosis
- Peripheral neuropathy
- Peripheral nerve pain
  - Herniated disc impinging on sciatic nerve
- Osteoarthritis of the hip or knee
- Venous claudication
- Symptomatic Baker’s cyst
- Chronic compartment syndrome
- Muscle spasms or cramps
- Restless leg syndrome

Also see Table 3 of Hirsch AT, et al. J Am Coll Cardiol. 2006;47:e1-e192.
## ACC Guidelines

### Table 3. Differential Diagnosis of Intermittent Claudication

<table>
<thead>
<tr>
<th>Condition</th>
<th>Location of Pain or Discomfort</th>
<th>Characteristic Discomfort</th>
<th>Onset Relative to Exercise</th>
<th>Effect of Rest</th>
<th>Effect of Body Position</th>
<th>Other Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent claudication</td>
<td>Buttock, thigh, or calf muscles and rarely the foot</td>
<td>Cramping, aching, fatigue, weakness, or frank pain</td>
<td>After same degree of exercise</td>
<td>Quickly relieved</td>
<td>None</td>
<td>Reproducible</td>
</tr>
<tr>
<td>Nerve root compression (e.g., herniated disc)</td>
<td>Radiates down leg, usually posteriorly</td>
<td>Sharp lancinating pain</td>
<td>Soon, if not immediately after onset</td>
<td>Not quickly relieved (also often present at rest)</td>
<td>Relief may be aided by adjusting back position</td>
<td>History of back problems</td>
</tr>
<tr>
<td>Spinal stenosis</td>
<td>Hip, thigh, buttocks (follows dermatome)</td>
<td>Motor weakness more prominent than pain</td>
<td>After walking or standing for variable lengths of time</td>
<td>Relieved by stopping only if position changed</td>
<td>Relief by lumbar spine flexion (sitting or stooping forward)</td>
<td>Frequent history of back problems, provoked by intra-abdominal pressure</td>
</tr>
<tr>
<td>Arthritis, inflammatory processes</td>
<td>Foot, arch</td>
<td>Aching pain</td>
<td>After variable degree of exercise</td>
<td>Not quickly relieved (and may be present at rest)</td>
<td>May be relieved by not bearing weight</td>
<td>Variable, may relate to activity level</td>
</tr>
<tr>
<td>Hip arthritis</td>
<td>Hip, thigh, buttocks</td>
<td>Aching discomfort, usually localized to hip and gluteal region</td>
<td>After variable degree of exercise</td>
<td>Not quickly relieved (and may be present at rest)</td>
<td>More comfortable sitting, weight taken off legs</td>
<td>Variable, may relate to activity level, weather changes</td>
</tr>
<tr>
<td>Symptomatic Baker’s cyst</td>
<td>Behind knee, down calf</td>
<td>Swelling, soreness, tenderness</td>
<td>With exercise</td>
<td>Present at rest</td>
<td>None</td>
<td>Not intermittent</td>
</tr>
<tr>
<td>Venous claudication</td>
<td>Entire leg, but usually worse in thigh and groin</td>
<td>Tight, bursting pain</td>
<td>After walking</td>
<td>Subsides slowly</td>
<td>Relief sped by elevation</td>
<td>History of iliofemoral deep vein thrombosis, signs of venous congestion, edema</td>
</tr>
<tr>
<td>Chronic compartment syndrome</td>
<td>Calf muscles</td>
<td>Tight, bursting pain</td>
<td>After much exercise (e.g., jogging)</td>
<td>Subsides very slowly</td>
<td>Relief sped by elevation</td>
<td>Typically occurs in heavy muscled athletes</td>
</tr>
</tbody>
</table>

Adapted from J Vasc Surg, 31, Dormandy JA, Rutherford RB, for the TransAtlantic Inter-Society Consensus (TASC) Working Group, Management of peripheral arterial disease (PAD), S1–S296, Copyright 2000, with permission from Elsevier (16).
## Differential Diagnosis of Intermittent Claudication

<table>
<thead>
<tr>
<th></th>
<th>Intermittent Claudication</th>
<th>Venous Claudication</th>
<th>Neurogenic Claudication</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Quality of pain</strong></td>
<td>Cramping</td>
<td>&quot;Bursting&quot;</td>
<td>Electric shock-like</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>Gradual, consistent</td>
<td>Gradual, can be immediate</td>
<td>Can be immediate, inconsistent</td>
</tr>
<tr>
<td><strong>Relieved by</strong></td>
<td>Standing still</td>
<td>Elevation of leg</td>
<td>Sitting down, bending forward</td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td>Muscle groups (buttock, thigh, calf)</td>
<td>Whole leg</td>
<td>Poorly localized, can affect whole leg</td>
</tr>
<tr>
<td><strong>Legs affected</strong></td>
<td>Usually one</td>
<td>Usually one</td>
<td>Often both</td>
</tr>
</tbody>
</table>
ARTERIAL DISEASE:

Also known as Peripheral Vascular Disease (PVD) or Peripheral Arterial Disease (PAD), lower extremity occlusive disease

= progressive narrowing or occlusion of lower extremity arteries resulting in decreased blood flow to limbs, thereby decreasing the amount of oxygen and nutrients delivered to tissues
Causes of PAD

1. Atherosclerosis
   - Most common etiology
   - Closely associated with coronary artery disease (CAD), cerebrovascular disease (CVD), AAA, renal artery stenosis, mesenteric ischemia, and their risk factors

2. Others
   - Aneurysms (hereditary or acquired)
   - Trauma / Radiation
   - Infection
   - Functional spasms (eg, Raynaud syndrome/dz)
   - Vasculitis (eg, Buerger’s disease, aka thromboangitis obliterans)
   - Anatomic abnormalities (eg, popliteal entrapment syndrome in young pts)
Major Risk Factors for PAD*

• Diabetes mellitus (OR 2.0 – 2.7)
• Current smoking (OR 1.4 for each 10 cig/day)
• Hypercholesterolemia (OR 1.2 for each 1 mmol/L elevation in cholesterol)
• Lipoprotein A, CRP
• HTN (OR 1.5 for mild, 2.2 for moderate)
• Age > 75 yrs (OR 1.2)
• Decreased risk w/ higher levels of HDL

* based on U.S. NHANES and Framingham Heart Study data
Clinical Presentation of PAD

• Progressive or acute
• Varying levels of severity, including life- or limb-threatening (stroke, AAA, limb ischemia)
  – Asymptomatic: 20-50% (dx by ABI screening)
  – Atypical leg pain: 40-50%
  – Classic claudication symptoms: 10-35%
  – Critical limb ischemia (or limb-threatenining ischemia): 1-2%
• Neurologic or abdominal symptoms
Mild Intermittent Claudication

• Classic / typical sx:
  – Intermittent cramping pain or discomfort, often in the calf, that occurs consistently and reproducibly w/ exertion, causing pt to stop walking, and is relieved by rest
  – May occur in any location on leg: buttocks and hip (Leriche’s syndrome) – usually more achy in nature, can be assoc w/ weakness / muscle atrophy and erectile dysfunction; thigh; feet (more commonly seen in Buerger’s disease if isolated)

• Atypical sx:
  – Similar to classic, but not severe enough to cause pt to stop walking or may not be relieved w/ rest
  – Heavy legs, fatigue
Critical Limb Ischemia

- Ischemic rest pain
  - Represents more severe decrease in limb perfusion; can lead to ischemic neuropathic pain (described as burning or throbbing pain)
  - Pain at rest, typically at night and involving foot and toes. Often paradoxically relieved w/ hanging feet over edge of bed or walking around
- Ischemic ulcers and/or gangrene
- Acute limb ischemia
  - Rapid or sudden decrease in limb perfusion that threatens tissue / limb viability. A vascular emergency!
  - 5 (or 6) P’s: PULSELESSNESS, PAIN, PALLOR, (PERISHING COLD or POIKYLOTHERMIA), PARESTHESIAS, PARALYSIS
- High risk of limb amputation (25%) and CV mortality (25%) at 1 year
Other associated signs/sx of PVD

- Skin changes on LE
  - Thin, brittle, and shiny
  - Cool temperature
  - Dusky erythema
  - Hair loss
  - Ulcers or non-healing wounds over pressure points
  - Pallor when legs elevated

- Diminished distal pulses
- Toenail changes (thickened and opaque)
- Impotence
- Weakness / decreased mobility / muscle atrophy
Arterial Ulcers

• Characteristics
  – Often located on toes or pressure points
  – Pale or cyanotic appearance w/ irregular margins
  – Painful, sometimes severe, often at night
  – Surrounding skin shiny and taut

• More to come on this…
Evaluation of PAD

• History
• Physical examination
• Diagnostic testing
  – Ankle-brachial index (ABI)
  – Segmental Doppler pressures and volume plethysmography
  – Duplex ultrasound imaging
  – Contrast Angiography
  – CT/MR Angiography
Physical Examination

• Inspection
  – Color
  – Surgical scars
  – Atrophic skin
  – Muscle wasting
  – Ulcers
  – Livedo reticularis
Physical Examination (con’t)

• Palpation
  – Skin temperature
  – Capillary refill
  – Pulses
    • Lower extremity: Dorsalis pedis, Posterior tibial, Popliteal, Femoral
    • Abdominal aorta, radial, and carotid
    • Radio-femoral delay
    • Radio-radial delay
Palpate pulses

Images courtesy of Mr. Nikolas Kosanovic M.D. General Surgery, Rural Clinical School, University of Melbourne
Physical Examination (con’t)

• Auscultation
  – Listen for bruits, mainly femoral, carotid, abdominal aorta and renal arteries
  – Assess with doppler if no palpable pulses
Non-invasive Diagnostic testing: Ankle brachial index (ABI)

- SBP at brachial artery
  - Take the highest reading of both arteries
- SBP at dorsalis pedis using doppler
- \( ABI = \frac{SBP_{DP}}{SBP_B} \)
- Interpretation:
  - \( >1.3 \) non-compressible art
  - \( 1 - 1.29 \) normal
  - \( 0.9 - 0.99 \) borderline
  - \( 0.41 - 0.9 \) mild to moderate PAD
  - \( <0.40 \) severe PAD
- May need to repeat w/exercise

Image courtesy of Mr. Nikolas Kosanovic M.D, General Surgery, Rural Clinical School, University of Melbourne
ABI interpretation

Figure courtesy of Mr. Nikolas Kosanovic M.D, General Surgery, Rural Clinical School, University of Melbourne
Non-invasive Diagnostic testing:
Segmental Doppler pressures and Volume plethysmography

• Segmental Doppler pressures:
  – Used to determine level and severity of PVD
  – Doppler cuff placed at proximal and distal thigh, calf, and ankle
  – Assess for 20mmHg or more reduction in BP between arterial segments of same leg or compared to same level on contralateral leg

• Plethysmography:
  – Measures arterial volume changes in the limb segment below the cuff, using a standardized volume of air in cuff, and then measuring pulsatile pressures
Variations in the contours of the pulse volume recording with segmental volume plethysmography reflect the severity of peripheral vascular disease. Mild disease is characterized by the absence of a dicrotic notch. With progressive obstruction, the upstroke and downstroke become equal, and with severe disease, the amplitude of the waveform is blunted.
Non-invasive Diagnostic testing: Duplex ultrasound

- Can accurately localize and quantify lesions
- Can differentiate between stenosis and occlusions, measure flow velocities in bypass grafts
- Requires experienced technician

Image courtesy of Mr. Nikolas Kosanovic M.D, General Surgery, Rural Clinical School, University of Melbourne
Invasive Diagnostic testing:
Contrast Angiography

- Gold standard evaluation of lower extremity ischemia
- Appropriate patient:
  - No contraindication (allergy, AKI, risk of cholesterol emboli)
  - Expected to undergo revasculartization
- Study is from aorta down, bilaterally
- CT or MR angiography can also be used
Medical Treatment of PAD

- Smoking cessation
- Aspirin 81-325mg daily (alternative if ASA-intolerant, clopidogrel 75mg daily)
- Treat underlying HTN, hypercholesterolemia, and DM
- ACE inhibitor for CV risk reduction
- For pts w/ intermittent claudication, supervised exercise program; cilostazol 100mg BID (PDE-3 inhibitory that reduces platelet aggregation; improves sx-free walking distance by 50%, avoid in pts w/ CHF); intermittent compression
Treatment of PAD

• Indications for intervention:
  – Limb salvage – ischemic rest pain, tissue loss, frank gangrene
  – Peripheral atheroembolisation
  – Incapacitating claudication, not responsive to medical tx

• For interim. claudication, revascularization w/:
  • Endovascular revascularization / percutaneous balloon angioplasty (PTA)
  • Stents (usually reserved for aortoiliac lesions)

• For critical limb ischemia
  • Refer to Endovascular specialist immediately
  • Bypass grafts
  • Amputation – Reserved for patients with frank gangrene or persistent painful ischemia non-amenable to vascular reconstruction; Survival rate: 50% at 3 yrs, 30% at 5 yrs
VENOUS DISEASE:

*Topics: Chronic Venous Insufficiency, Varicose Veins, Venous Ulcers*

= chronic dilation or reflux of veins in the lower extremity

Independent of peripheral arterial disease, but can be just as debilitating.
Venous Anatomy

• Superficial, deep, and perforating / communicating veins
• One-way valves prevent pooling of blood in legs
• Muscle contractions help facilitate blood return to heart
• Flows superficial ➔ deep veins

Lower Extremity Venous Anatomy

Superficial Veins

Deep Veins

Images courtesy of Up-To-Date Overview and management of lower extremity chronic venous disease and http://www.health.com/health/library/ndp01_zm2340.00.html
Chronic Venous Disease / Insufficiency

- **Chronic Venous Disease** = arise from venous valve incompetence resulting in retrograde blood venous blood flow, lasting for an abnormal duration and with associated signs / symptoms

- **Chronic Venous Insufficiency** = represents more advanced state of above, including edema, skin changes, and ulcerations

http://advancedvenoussolutions.com/why/insufficiency.html
Venous Insufficiency

- Risk Factors/History
  - Previous DVT
  - h/o Varicosities
  - Reduced mobility
  - Obesity
  - Vascular Ulcers
  - Phlebitis
  - Traumatic injury
  - Cardiomyopathy
Clinical Signs/Sx of Venous Disease

Symptoms
- Limb discomfort (tiredness, heaviness)
- LE pain (generalized achiness or localized) or swelling
  - Worse w/ standing
  - Improves w/ elevation or walking
- Paresthesias (tingling, burning)
- Tightness in legs
- Skin irritation/itching or discoloration / redness
- Bleeding
- Muscle cramps
- Generalized fatigue

Signs
- Evidence of dilated veins, including telangiectasias, reticular veins, and varicose veins
- Leg edema
  - Can be unilateral in early stages
  - Often localized only to legs/feet
- Skin changes
- Ulcers
- “Blistering”
- Superficial thrombophlebitis
Edema

Mild

Reticular veins are dilated bluish subdermal veins, one to three millimeters in diameter. The deeper blue reticular veins contrast the bright red, fine telangiectasias. Mild ankle oedema in this patient is evident at medial ankle below the malleolus.

Moderate to severe

Significant pitting oedema of lower leg with skin changes consistent with chronic venous stasis, including stasis dermatitis and lipodermatosclerosis.
Differential Dx for LE edema (it’s long!)

- Increased capillary hydrostatic pressure
  - CHF, including cor pulmonale
  - Renal disease
  - Medication effect causing Na retention (Ca-channel blockers)
  - Pregnancy
  - Localized venous obstruction (e.g., thrombus, valve incompetence)
  - Cirrhosis or hepatic venous obstruction
  - Acute Pulm Oedema
- Hyopalbuminemia (i.e., decreased oncotic pressure)
  - Nephrotic syndrome
  - Protein-losing enteropathy
  - Liver disease
  - Malnutrition

- Increased capillary permeability
  - Trauma
  - Burns
  - Inflammation
  - Sepsis
  - Allergic reactions
  - ARDS (Adult Respiratory Distress Syndrome)
  - Diabetes mellitus
  - Malignant ascites
  - Iatrogenic, IL-2 therapy

- Lymphatic obstruction (or increased interstitial oncotic pressure)
  - Lymph node dissection
  - LN enlargement due to malignancy
  - Hypothyroidism
  - Malignant ascites
Skin Changes seen with Chronic Venous Insufficiency

• Leathery, thick skin at ankle or leg due to irritation / scratching, but underlyng skin is thin and fragile and can easily ulcerate
• Stasis dermatitis = Hyper-pigmentation from hemosiderin deposition creating a reddish-brown appearance with diffuse or spotty pigmentation
• Lipodermatosclerosis
• Pale or white lesions (atrophie blanche)
• Autoeczematous reaction
Stasis Dermatitis

• Aka “congestion or stasis eczema”

• Hyper-pigmentation from hemosiderin deposition creating a reddish-brown appearance with scaliness in diffuse or spotty pattern

• An inflammatory process that presents insidiously w/ itching, skin discoloration, scale, thin skin of one or both legs

• Commonly effects medial malleolus region first
Lipodermatosclerosis

• Localized chronic inflammation (panniculitis) leading to fibrosis of the skin and subcutaneous tissues of the lower leg
• Skin appears hyper-pigmented, red, scaly, and hard or indurated
• Can involve most of the leg circumferentially
• “Inverted champagne bottle shape”
Atrophie blanche

- Localized, usually circular atrophic, pale to whitish skin areas surrounded by dilated capillaries (telangiectasias) and hyperpigmentation
- Most often seen on the medial distal leg near the malleolus, or can occur within lipodermatosclerotic skin, where they correspond to points of avascular fibrosis
- Do not represent healed venous ulcers. They are vulnerable to future ulceration because of poor perfusion
Autoeczematous reaction

- Eczematous, dry, scaly rash can cause difficult-to-control pruritis. Can lead to many excoriations from scratching, that can be a source of infection.
- Rashes mimicking the dermatitis on the legs can appear as eczematous patches on other body sites, or can present as a generalized body rash, an auto-eczematous or "id" reaction.
**Varicose Veins**

- Dilated, elongated, tortuous subcutaneous veins $\geq 3$ mm in diameter
- Incompetent venous valves
Varicose Veins

• Prevalence
  – Generally F > M, 10-30% of general population, increases w/ age

• Risk Factors:
  – Age, family hx, female, obesity, sedentary lifestyle, prolonged standing, smoking, trauma to LE, prior venous thrombus (deep or superficial), pregnancy / high estrogen states, lax ligaments (flat feet, hernias), AV fistulas
Varicose Veins

Primary
- Varicose veins caused by venous insufficiency due to venous wall weakness
- Example: due to age or pregnancy

Secondary
- Varicose veins caused by venous insufficiency due to venous damage from other etiology
- Example: deep vein thrombosis or leg injury
Clinical Signs/Sx of Varicose Veins

- Similar to venous insufficiency
- Dilated superficial vessels
- Itching
- Pain
- Bleeding – can be quite significant
- Skin changes
- Tired, achy, heavy legs
Clinical Evaluation for Venous Insufficiency and Varicose Veins

• Inspection
  – Inspect patient standing
  – Venous stars - bluish vessels that may distend above the skin surface and are usually 1-2 mm in diameter
  – Superficial thrombophlebitis - red, painful lump
  – Hemosiderin deposition
  – Venous eczema
  – Ulcers
  – Scars from previous vein surgery or veins harvested for CABG

• Palpation
  – Pulses
  – Warm
  – Hard or tender varicosities

• Neurologic

• Try to localize incompetent vein
Trendelenburg Test

- Ask the patient to lie down
- Elevate leg to empty engorged veins
- Press on the saphenofemoral junction
  - Locate the femoral artery
  - The saphenofemoral junction is medial and 2cm below it
- Stand patient up and watch for varicose veins
  - If varicose veins are controlled the saphenofemoral junction is incompetent
  - If not, there is an incompetent perforator below

Tourniquet test

- Same principle as Trendelenburg test
- Lie patient supine
- Elevate leg to empty engorged veins
- Apply tourniquet to thigh, lower thigh, then below knee and stand patient up
- If varicose veins are controlled, incompetence is above the tourniquet
- If not it’s below tourniquet

http://orthoinfo.aaos.org/topic.cfm?topic=A00534
Complications of Chronic Venous Insufficiency

- Cellulitis
- Venous ulcers
- Hemorrhage
- Thrombophlebitis
Diagnostic Testing

• Often dx is clinical
• Diagnostic testing for:
  – Confirm dx
  – Localize site
  – Determine severity and etiology (ie, reflux, obstruction, both)
  – Dx concurrent PAD
  – Identify pts who may benefit from invasive intervention

• Duplex Ultrasound
  – Preferred method (accurate, non-invasive, reproducible, inexpensive)
  – Assesses deep and superficial veins for presence and direction of blood flow (therefore assesses for reflux and obstruction)

• Descending venography
  – Rarely used (uses contrast, expensive, invasive, uncomfortable, risk of phlebitis)
Duplex Ultrasound

Patent vein compresses

Thrombosed vein will not compress

Images courtesy of Mr. Nikolas Kosanovic M.D, General Surgery, Rural Clinical School, University of Melbourne
Medical Management

- Avoid long periods of standing or sitting
- Exercise
- Weight loss
- Compression Stockings
- Leg elevation
- Medications/herbals
  - Horse chestnut seed extract (Escrin)
  - Aspirin
  - Pentoxifylline (for more rapid healing of venous ulcers)
  - Stanozolol (anabolic steroid that stimulates fibrinolysis and may help w/ lipodermatosclerosis)
  - No role for diuretics for edema purely due to CVI
- Skin care w/ cleansing, emollients, barrier creams (petroleum, zinc oxide), topical steroids
Compression Stockings

<table>
<thead>
<tr>
<th>Class</th>
<th>Uses</th>
<th>Pressure at Ankle (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Mild venous insufficiency or varicose veins</td>
<td>14-17</td>
</tr>
<tr>
<td>II</td>
<td>Treatment and Prevention of Venous Ulcer Recurrence</td>
<td>18-24</td>
</tr>
<tr>
<td>III</td>
<td>Treatment of severe venous hypertension and ulcer prevention in large diameter calves</td>
<td>25-35</td>
</tr>
</tbody>
</table>
Compression Stocking Application
Indications for Invasive Treatment

• Not responsive to medical therapy
• Large or multiple ulcers
• Perforator or saphenofemoral reflux

Contraindications:
  – DVT
  – Severe edema
  – Infection
Invasive Therapy for Varicose Veins

• Non-Surgical
  – Sclerotherapy (chemical ablation)
  – Thermal Ablation (superficial surface lasers or endovenous light/radiofrequency)

• Surgical
  – Ligation and Stripping (mechanical ablation)
  – Microincision and phlebectomy (mechanical ablation)
  – Vein bypass
Venous Ulcers

- Characteristics
  - Exquisitely tender
  - Shallow
  - Exudative w/ granulomatous red base
  - Irregular borders, but well demarcated
- Usually located on distal leg over medial aspect
- Multiple or single
  - Can extend around circumference of leg if not treated
- Surrounding skin hyperpigmentation
Medical Treatment of Chronic Venous Ulcers

- Wound management
- Compression stockings
- Pentoxyfylline
- EMLA cream
Surgical Management: Chronic venous ulcers

- Surgical debridement
- Skin grafting with bilayer artificial skin plus compression bandages
- Venous surgery
Recurrent Ulcers

- Continue wearing compression stockings to prevent recurrence

- Tx strategies:
  - Duplex ultrasound
  - Consider ablation or surgical intervention
Neuropathic Claudication

- Peripheral neuropathy
  - Sensory
  - Autonomic
  - Motor
Risk Factors

- DM (high blood sugars)
- Spinal Cord Injury
- Hypertension
- Smoking
- EtOh
- Trauma
- Obesity
- Elevated TG
Signs & Symptoms

- Relief of pain with ambulation
- Parasthesia of extremities
- Altered gait
- Orthopedic deformities
- Diminished reflexes
- Altered sensation
- Intolerance to touch
- Calluses
- Fissures/cracks of the heels
Risk Factors for Ulceration

**Sensory Neuropathy**

- Largest single risk factor for diabetic foot ulcers
  - Burning, tingling, "pins & needles", numbness or "dead" feeling
  - Repeated unrecognized stress, pressure, friction & shearing.
  - Lack sensation to feel foreign objects, heat changes, discomfort or pain.
Risk Factors for Ulceration

**Autonomic Neuropathy**

- Impairs skin integrity, sweat regulation & blood flow.
- Leads to:
  - thick, dry cracked skin, fissures
  - callus build-up at pressure points
Risk Factors for Ulceration

Motor Neuropathy

- Loss of muscle tone in the foot
- Foot deformities:
  - Hammer toes
  - Claw toes
- Metatarsal heads become prominent
- Changes in pressure distribution & gait pattern
Under diagnosis of neuropathy

- Fundamental problem in primary care.
- Impedes early identification, management & prevention of ulcers.
Risk Factors for Ulceration

Elevated Pressures & Foot Deformity

- Pes Planus - flat foot
- Pes Cavus - high arch
- Charcot Foot - (significant disruption of the bony architecture)
- Lesser toe deformities
CHARCOT FOOT

Diabetic Neuropathic Osteoarthropathy

• Occur in presence of peripheral sensory neuropathy, autonomic neuropathy & trauma.

• Presentation: painless, unilateral oedema, erythema, with or without foot deformity, bounding pedal pulses. Post tibial dysfunction in later stages.

• Note:
  – Acute charcot can mimic cellulitis & DVT
  – Radiological findings can be normal at first
  – Strict immobilization of foot for management
  – Patient education, protective footwear to prevent ulcerations
Risk Factors for Ulceration

**Calluses**

- Presence of callus in an insensitive foot is highly predictive of subsequent foot ulceration.
- Breakdown of underlying tissues
- Regular debridement
- Pressure relief: insoles / moulded orthotics
- Footwear

*Calluses increase pressure on underlying tissue by 30%*
Risk Factors for Ulceration

Limited Joint Mobility

- Hallux rigidus
- Hallux limitus
- Hammer toes
- Claw toes

Limited joint mobility can cause increased ground reaction forces under weight-bearing joints. This can lead to ulceration.
Risk Factors for Ulceration

**Previous Ulceration & Amputation**

- Skin texture
- Scar tissue reduced tensile strength.
- Pressure points
What to look for & assess!

**Dermatological:**
- Color
- Temperature
- Texture
- Errythema
- Edema
- Lesions
- Fissures
- Callus
- Ulcers
- Nail disorders

**Vascular:**
- Pedal pulses
- digital hair
- capillary revascularization
- Varicosities
- ABI, TPI, PPG
- Edema
- Transcutaneous oxygen concentrations
- Angiography
- MRI
A BRIEF COMPARISON OF LOWER EXTREMITY ULCERS

Arterial vs Venous vs Neuropathic Ulcers
Distribution Of Ulcers

<table>
<thead>
<tr>
<th>Location</th>
<th>Non-venous</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calf</td>
<td>8%</td>
<td>5%</td>
</tr>
<tr>
<td>Gaiter</td>
<td>43%</td>
<td>87%</td>
</tr>
<tr>
<td>Foot</td>
<td>49%</td>
<td>8%</td>
</tr>
</tbody>
</table>

100% 100%
Common Sites Of Venous, Arterial And Neuropathic Ulceration

Image courtesy of Mr. Nikolas Kosanovic M.D, General Surgery, Rural Clinical School, University of Melbourne
<table>
<thead>
<tr>
<th></th>
<th>Arterial Ulcer</th>
<th>Venous Ulcer</th>
<th>Neuropathic Ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Location</strong></td>
<td>Toes or pressure points</td>
<td>Medial malleolus, Lateral/post. Calf</td>
<td>Sole of foot or bony prominences</td>
</tr>
<tr>
<td><strong>Appearance</strong></td>
<td>Irreg. margin, pale, cyanotic</td>
<td>Well-demarcated but irreg. margin, red base, exudative</td>
<td>“Punched out” appearance, red, often deep, infected</td>
</tr>
<tr>
<td><strong>Foot Temp</strong></td>
<td>Cool and dry</td>
<td>Warm</td>
<td>Warm and dry</td>
</tr>
<tr>
<td><strong>Pain</strong></td>
<td>Present, sometimes severe</td>
<td>Mild</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Pulses</strong></td>
<td>Absent</td>
<td>Present</td>
<td>+/-</td>
</tr>
<tr>
<td><strong>Veins</strong></td>
<td>Collapsed</td>
<td>Dilated, varicose, telangiectasias, reticular</td>
<td>Dilated</td>
</tr>
<tr>
<td><strong>Sensation</strong></td>
<td>Variable</td>
<td>Normal</td>
<td>Absent (no vibr sense)</td>
</tr>
<tr>
<td><strong>Ulcer w/in callous</strong></td>
<td>No</td>
<td>No</td>
<td>Often</td>
</tr>
<tr>
<td><strong>Foot deformities</strong></td>
<td>No</td>
<td>No</td>
<td>Often</td>
</tr>
<tr>
<td><strong>Skin changes</strong></td>
<td>Shiny, taut</td>
<td>Reddish-brown pigmentation, atrophie blanche</td>
<td>Shiny, taut, or doughy</td>
</tr>
</tbody>
</table>
Arterial Ulcers Affecting Heel And Shin

Images courtesy of Mr. Nikolas Kosanovic M.D, General Surgery, Rural Clinical School, University of Melbourne
Venous Ulcers Lower Leg Above Malleoli, but May Affect The Dorsum Of The Foot

Images courtesy of Mr. Nikolas Kosanovic M.D, General Surgery, Rural Clinical School, University of Melbourne
Neuropathic Ulcers On Sole Of Foot And Dorsum Of Toe Joints

Images courtesy of Mr. Nikolas Kosanovic M.D. General Surgery, Rural Clinical School, University of Melbourne
<table>
<thead>
<tr>
<th>Origin</th>
<th>Cause</th>
<th>Location</th>
<th>Pain</th>
<th>Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Main arteries</td>
<td>Atherosclerotic lower extremity PAD, Buerger's disease, acute arterial occlusion</td>
<td>Toes, foot</td>
<td>Severe</td>
<td>Irregular, pink base</td>
</tr>
<tr>
<td>Venous</td>
<td>Venous disease</td>
<td>Malleolar</td>
<td>Mild</td>
<td>Irregular, pink base</td>
</tr>
<tr>
<td>Skin infarct</td>
<td>Systemic disease, embolism, hypertension</td>
<td>Lower third of leg</td>
<td>Severe</td>
<td>Small after infarction, often multiple</td>
</tr>
<tr>
<td>Neurotrophic</td>
<td>Neuropathy</td>
<td>Foot sole</td>
<td>None</td>
<td>Often deep, infected</td>
</tr>
</tbody>
</table>

Adapted from J Vasc Surg, 31, Dormandy JA, Rutherford RB, for the TransAtlantic Inter-Society Consensus (TASC) Working Group, Management of peripheral arterial disease (PAD), S1–S296, Copyright 2000, with permission from Elsevier (16).

PAD = peripheral arterial disease.
Thank you!

Questions??