Chronic Venous Insufficiency (CVI)

Background
1. Definition
   - Poor blood flow in leg veins
   - Pooling of lymph in lower extremities
   - Incompetence of lower extremity valves

Pathophysiology
1. Pathology of disease
   - Venous hypertension from obstruction or reflux through incompetent valves
   - Venous hypertension causes edema formation, fibrin deposition, erythrocyte and leukocyte sequestration, thrombocytosis, and inflammation
   - Often associated with valve reflux in saphenous veins and obstruction in iliac vein
   - Perivascular inflammation caused by cytokine responses leading to tissue damage and skin changes
   - Damage to lower extremity venous valves due to previous DVT
2. Incidence, prevalence
   - Affects 2-5% of Americans
   - Incidence increases with age, higher among women
   - Peak incidence in females 40-49 years old; males 70-79 years old
   - Worsens with pregnancy
   - Venous leg ulcers affect 2.5 million Americans each year
3. Risk factors
   - Age, increasing
   - Smoking
   - Elevated levels of circulating estrogen
   - Family/personal history of DVT
   - Obesity
   - Sedentary lifestyle, prolonged standing
4. Morbidity/Mortality
   - Venous stasis ulcers and associated morbidity
   - Increases risk for DVT as associated morbidity/mortality

Diagnostics
1. History and physical exam are sufficient to make diagnosis
2. History
   - Persistent dependent bilateral leg edema that improves overnight and/or with leg elevation
     - Intermittent ankle edema consistent with superficial venous disease
     - More prominent edema beyond ankles consistent with deep disease
   - Persistent hyperpigmentation (hemosiderin staining)
   - Non-healing ulcers
   - Varicose veins - indicate venous hypertension
   - Leg pain that worsens when legs dependent and relieved by elevation, support stockings, and walking (all lower venous pressures)
   - Itching
Nocturnal leg cramps
- Restless legs

3. Physical exam
- Bilateral lower extremity edema
  - Usually significant at 1+ to 4+
- Lipodermatosclerosis (induration, tissue fibrosis)
- Atrophic blanche (smooth, white, atrophic plaques of sclerosis)
- Hemosiderin deposition from red blood cells makes skin appear reddish or brown
- Lower extremity ulcers, especially near medial malleolus where venous pressure is maximal
- Lower extremity varicose veins may or may not be present
- Negative Homan’s test

4. Diagnostic testing
- Duplex venous ultrasonography may be used to r/o DVT and diagnose valvular insufficiency; can detect thrombosis, post-thrombotic changes, patterns of obstructive flow, and reflux
- Venography remains the gold standard; can diagnose valvular insufficiency; provides more detail than ultrasound
- Ankle-Brachial index (ABI) to exclude arterial occlusion

Differential Diagnosis
1. Deep venous thrombosis (DVT)
   - Usually unilateral, positive Homan’s test, positive duplex venous ultrasound, positive D-dimer

2. Cellulitis
   - Febrile, localized area of erythema on one leg, streaking, scant unilateral lower extremity edema

3. Diabetic foot or leg ulcer
   - Underlying diagnosis of diabetes, persistent non-healing ulcer, usually super infected

4. Peripheral arterial disease
   - Claudication, rest pain, decreased ABIs
   - Ischemic ulcers deeper with gangrenous edges
   - Absent or decreased distal pulses, cold and blue foot, can be bilateral

5. Contact dermatitis or psoriasis, polyarteritis nodosa, or allergic dermatitis
   - Absent or scant edema at area of skin discoloration, history of new products against skin of lower extremities
   - Skin biopsy to differentiate

6. Acanthosis nigricans or hemosiderosis
   - Hyperpigmentation more diffuse and may involve other body areas

7. Congestive Heart Failure
   - No associated skin hyperpigmentation, no decreased edema with leg elevation, other associated signs and symptoms of CHF: elevated JVP, crackles on lung exam

8. Liver failure, renal failure, hypothyroidism, or lymphedema
   - Edema elsewhere in addition to bilateral lower extremity edema: ex. periorbital edema
Lymphedema cannot be clinically differentiated from CVI²

9. Hypoalbuminemia ⁷
   - Associated with inflammatory syndrome
   - Slower wound healing

**Therapeutics**

1. Compression stockings
   - Graduated compression (higher pressure at ankle than below knee)
   - Class I stocking (20-30mmHg) to control edema
   - Class 2 (>30-40mmHg) or Class 3 (>40mmHg) to control dermatitis or ulcers²⁸

2. Alternate compression can be done by bandages
   - Multi-layered elastic bandages appear superior, especially in venous ulcer treatment
     - Produce more rapid venous ulcer healing than topical meds or absorbency dressings

3. Elevation of legs above heart for 30 minutes 3-4 x /day reduces edema

4. Exercise improves function of calf muscle pump

5. Drug therapy
   - **Horse chestnut seed extract**
     - 300 mg BID
     - Significant improvement in leg pain, edema, and pruritis⁹
   - **Pentoxifylline** modestly improves ulcer healing when combined with compression²
     - Meta-analysis showed greater efficacy for complete or partial ulcer healing versus placebo or no treatment (SOR A)¹⁰
     - Used as adjunct to compression therapy
     - GI side-effects include nausea, indigestion, and diarrhea
   - **Phlebotinics** may increase blood flow, varied results¹¹
   - **Topical steroids** such as intermediate potency triamcinolone 0.1% cream or **Elocon 0.1%** cream BID⁴
   - **Diuretics**
     - Short course for severe edema
     - **Furosemide** 40 mg once a day
   - **Systemic antibiotics** - if signs of ulcer infection (fever, lymphangitis, increasing erythema, swelling or pain) to cover staphylococcus, streptococcus, proteus, pseudomonas and other skin flora – use for limited time²
     - **Cephalexin** 500 mg BID
     - **Bactrim DS** BID

6. Procedures and Surgical treatment
   - **Ablation of varicosities and angiomas**
     - Sclerosant injections
     - Laser ablation
   - **Ablation of saphenous veins**
     - Foam sclerotherapy
     - Radiofrequency ablation
     - Laser ablation
     - Surgical stripping
Subfascial endoscopic perforator surgery (SEPS) to interrupt perforator veins
• Percutaneous stenting of chronic occlusions
• Deep valve reconstruction to correct reflux (available in specialized centers)²,⁵

Follow-Up
1. Adherence to elevating legs and wearing compression stockings requires physician reinforcement
2. Patients with ulcers need frequent follow-up for wound care
3. Considerations for referral
   • Non-healing ulcers
     ▪ Failure to decrease in size in three months
     ▪ Failure to heal in twelve months
   • Arterial insufficiency
   • Persistent stasis dermatitis
   • Contact dermatitis
   • Diagnostic uncertainty
   • Superficial venous surgery

Prognosis
1. Compression and elevation can control symptoms and complications²
2. Poor prognostic signs for progression to chronic disease include:
   • Both reflux and obstruction present
   • Involvement of multiple segments of venous system
   • Ipsilateral recurrent DVT¹²
3. Disease progression more rapid if occurs after thrombosis versus primary disease¹²
4. Ulcer recurrence rate reduced with superficial venous surgery
   • Compression therapy still required following surgery⁵

Prevention
1. Lifestyle modifications
   • Exercise involving lower extremities
   • Smoking cessation
   • Weight loss
2. Elastic compression stockings to prevent post-thrombotic syndrome⁵,⁶
3. Avoid topical products containing common sensitizers such as lanolin, neomycin, nickel

References


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