

Peripheral Venous Insufficiency

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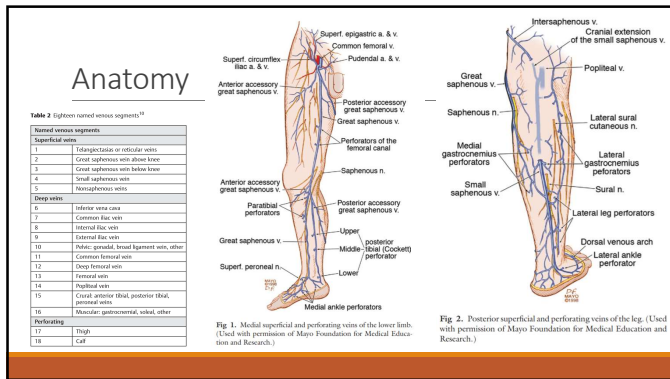
VASCULAR AND INTERVENTIONAL RADIOLOGY, ADVENT HEALTH SYSTEMS
CENTRAL TEXAS

Conflicts of Interest

None to report

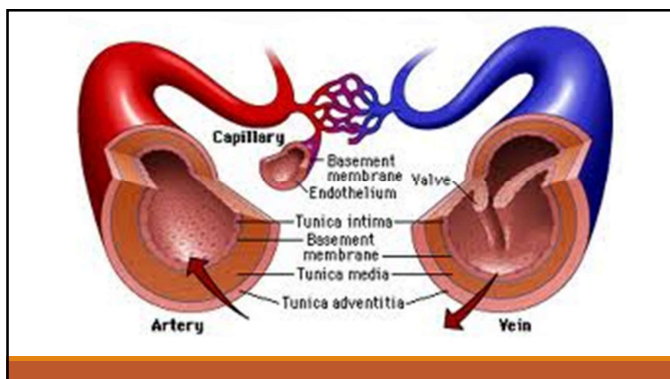
Peripheral Venous Insufficiency: Epidemiology

- Estimated prevalence: > 20% varicose veins, ~5% venous edema, skin changes, or ulceration in the Western adult population
- Female:male 3:1
- Prevalence of active venous ulcers estimated at ~0.5%, healed venous ulcers up to ~1.4 %
- ~2.5 million people with advanced CVD
- >50% of venous ulcers require more than 1 year of therapy
- Disability rated at 2 million workdays/year, responsible for early retirement in 12.5% of workers with venous ulcers
- Estimated \$3 billion annual cost of coverage
- Significant impact on QOL, often rated more bothersome to patients than chronic lung disease, back pain, or arthritis



Superficial Veins

- Not paired with corresponding named arteries
- Small saphenous vein – lateral to posterior, posterior to lateral malleolus, midline in upper calf, between two heads of the gastrocnemius, usually drains into the popliteal
- Great saphenous vein – anterior to medial malleolus, anteromedial to the knee, feeds the femoral vein at the saphenofemoral junction, multiple tributaries
- Accessory anterior saphenous vein
- Posterior accessory saphenous vein
- Deep veins are a viable treatment target in some cases of central venous reflux-mediated venous insufficiency but outside scope of current review





Pathophysiology

- The peripheral venous system normally acts as a store of blood and as a conduit to return blood to the heart
- Blood in the lower extremities must work against gravity and other forces to return blood to the heart
- Superficial veins are superficial to the muscular fascia, deep veins are below; perforating veins connect the two
- Bicuspid one way valves throughout the veins, usually 7-9 in GSV and SSV, increasing in number from proximal to distal leg
- Blood flow usually superficial to deep above the foot
- Primary varicose vein pathophysiology felt to be the result of either primary valvular incompetence or possibly congenital vein wall weakness

Pathophysiology

- Varicose veins have been shown to demonstrate disrupted and distorted muscle fiber layer with proliferation of the collagen matrix, leaving only elastic tissue and collagen as the sole components in the wall in the most severely affected areas
- Loss of contractility and sagging of muscular grid with resultant venous hypertension and vein dilatation
- This may be due to an intrinsic genetic defect of collagen synthesis (too much type I, not enough type III and fibronectin)
- Poor valve leaflet apposition due to expanded valvular annulus results in venous reflux
- Venous stasis ulcer formation may be due to complex inflammatory reaction with tissue hypoxia, humoral stimulation, and shift in fluid shear forces at the endothelial level due to venous hypertension
- Upregulation of endothelial attachment proteins, sequestration of activated leukocytes, immunocyte migration into affected connective tissues, increased free radical formation, hemosiderin deposition in the case of C4b disease, increased parenchymal cell death
- Secondary pathophysiology usually post-phlebitic from valve leaflet damage due to thrombus

Clinical Evaluation: Symptoms

- Leg pain
 - Worse with standing/dependence, better with elevation/walking
 - Direct association with veins, non-radicular, not exacerbated with joint movement
- Aching, especially later in day of after prolonged position of dependency
- Leg Swelling
 - Usually correlates with degree of insufficiency
- Itching or burning, usually in the vicinity of a vein
- Restless leg syndrome
- Hair loss in the lower legs
- Venous Claudication
 - Thigh/leg pain and tightness following vigorous exercise, resolving with rest
 - Also associated with deep venous reflux/occlusion

Clinical Evaluation: Signs Corona phlebectatica

Table II. Presence of corona according to the CEAP clinical classes

CEAP clinical class	Corona absent	Corona present	Sensitivity (%)
C0	44	0	0
C1	34	14	29.6
C2	53	30	36.1
C3	37	17	31.5
C4	16	24	60
C5-C6	7	11	61



Clinical Evaluation: Signs Atrophie blanche



Clinical Findings: Risk Factors and Comorbidities

- Ligamentous laxity (herniation, pes planus)
- Hyperestrogenism
- Parity (greater association with multiparity)
- Obesity
- Phlebitis
- Hereditary
 - strong family history; up to 90% penetrance if both parents affected
 - inheritable disorders such as Parkes-Weber, Klippel-Trenaunay, HIT, ataxia telangiectasia
- History of DVT (e.g., May-Thurner)
- Age
- Gender (more common in females)
- Arteriovenous shunting
- Western lifestyle?

Clinical Findings: Widmer Classification

Classification	Symptom
I	Corona phlebectatic paraplantis (ankle flare), subclinical mild edema
II	Hyperpigmentation, lipodermatosclerosis, atrophie blanche, edema, eczema
III	Healed or active ulcer

Clinical Findings: CEAP

Table 1. CEAP classification for chronic venous disease¹⁰

CEAP Classification	
Clinical	Pathologic
C0	Asymptomatic, no signs of disease
C1	Edema, no signs of disease
C2	Edema, no signs of disease
C3	Edema, no signs of disease
C4	Edema, no signs of disease
C5	Edema, no signs of disease
C6	Edema, no signs of disease
C7	Edema, no signs of disease
C8	Edema, no signs of disease
C9	Edema, no signs of disease
C10	Edema, no signs of disease
C11	Edema, no signs of disease
C12	Edema, no signs of disease
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C91	Edema, no signs of disease
C92	Edema, no signs of disease
C93	Edema, no signs of disease
C94	Edema, no signs of disease
C95	Edema, no signs of disease
C96	Edema, no signs of disease
C97	Edema, no signs of disease
C98	Edema, no signs of disease
C99	Edema, no signs of disease
C100	Edema, no signs of disease

CEAP

Clinical

Etiologic

Anatomic

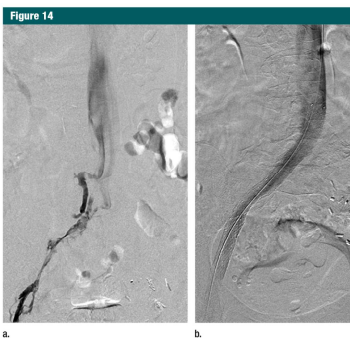
Pathophysiologic

Post Thrombotic Syndrome

- Chronic complication of DVT
- > 1/3 of DVT patients will develop PTS, 5-10% will develop severe PTS
- Elastic compression stockings and other compression therapy are mainstays of treatment
- DVT has high recurrence, ~30% within 8 years if only 3-6 months anticoagulation
- Especially for early DVT, acute intervention can prevent long term symptoms
- Risk factors include inherited or acquired hypercoagulable disorder, prolonged stasis, injury, surgery, malignancy, OCPs, HRT, elevated BMI, anatomic factors (e.g., May Thurner's), pitting edema, h/o DVT or PE



Figure 1. Postthrombotic syndrome.



Clinical Findings: C0

- No visible or palpable signs of venous disease
- Treat with conservative measures

Clinical Findings: C1

Telangiectasias/Reticular veins

- Mildly dilated intradermal venules, usually > 1 to 3 mm (telangiectasias generally classified as < 1 mm)
- Dilated, blue tinged, tortuous
- May be isolated findings in 50-66% of affected individuals
- Women more commonly affected than men
- May be amenable to localized treatment with sclerotherapy



Treatment Options for C1 Disease

- Usually cosmetic
- Not often associated with venous reflux
- Treatment of choice: sclerotherapy
- May respond secondarily to GSV or SSV ablation
- Treat bigger veins before smaller veins
- Laser therapy available (1064 nm Nd-YAG), but no difference in outcome and increased pain

Clinical Findings: C2

Varicose Veins

- Subcutaneous dilated veins ≥ 3 mm
- Estimated prevalence: ~29 million
- Blood pooling in stagnant venular segments with resultant chronic inflammation and phlebitis
- Can be associated with aching, burning, itching, pain, and cramping
- Often cosmetically undesirable by patients
- Various treatments available including stab phlebectomy, sclerotherapy, treatment of greater saphenous vein



Treatment Options for C2 Disease

- Ablative outcomes are at least as good as surgery (up to 84% effectiveness)
- May require multiple treatments to be effective
- No difference in clinical success and patient outcome between ablative technique (EVLT) and sclerotherapy, though closure rates higher with endovenous ablation or surgery at ~88% vs 72% for foam alone
- For cases of truncal reflux, studies have shown that delayed sequential phlebectomy or further sclerotherapy after endovenous ablation of the truncal vein limits need for further treatments
- Concomitant therapy may be better for patients with > 6 mm varicosities or > 6 mm distal thigh GSV

Advanced Chronic Venous Insufficiency: C3-C6

Clinical Findings: C3

Edema

- Pitting edema
- Usually at the ankle, occasionally extends into the leg before the feet and toes
- Mimic: lymphedema, which is more likely to involve the feet and toes
 - Usually NON-pitting
- Treatment with compression therapy



Clinical: C3 Edema

- Superficial venous pressure normally 12 mmHg when supine, up to 104 mmHg standing up, and up to 30 mmHg with walking
- Becomes elevated 60-90 mmHg with venous insufficiency
- With venous insufficiency, pressure can increase significantly due to incompetent valves, distending the vessel, increased venous capillary pressure, increased venous filtration of blood, and more interstitial fluid
- Acute etiologies include DVT, trauma, infection, allergy, sepsis
- Chronic causes include regional venous problems, systemic issues like CHF, nephrotic syndrome, or other rarer causes
- Medication related (gabapentin, amlodipine, pregabalin, prednisone, NSAIDs, MAOIs, antipsychotics, hydralazine, minoxidil, CCBs, beta blockers, clonidine, acyclovir, pramipexole, etc.

Clinical Findings: C4a

Pigmentation/Eczema

- Pigmentation: Dark brown discoloration of skin
- Sometimes may be seen with stasis dermatitis
 - Usually in the ankle but may extend to the more proximal leg or foot
 - Usually at the ankle, occasionally extends into the leg before the feet and toes
- Eczema: erythematous dermatitis below the knee
 - May blister, weep, erupt
 - May occur independent of varicose veins



Clinical Findings: C4b

Lipodermatosclerosis

- Chronic inflammation and fibrosis of the skin/subcutaneous tissues of lower leg
- Fibrosing panniculitis of the subcutaneous tissue which "scars down" the skin to subcutaneous tissue
- Glomerular-like capillaries which are absent in the region of fibrotic scars (atrophie blanche, livedoid vasculopathy)
- Can be associated with contracture of the Achilles tendon
- Eczema: erythematous dermatitis below the knee
 - May blister
 - May occur independent of varicose veins



Clinical Findings: C5

Healed Venous Ulcer

- Atrophic skin
- Pigmentation changes
- History of ulceration
- Mimic: atrophie blanche: localized depressed region of abnormal white colored skin surrounded by hyperpigmentation and dilated capillaries
 - Often seen in the absence of a history of ulceration



Clinical Findings: C6

Active Venous Ulcer

- Full thickness skin defects
- Usually in the ankle region, but can be more proximal, even circumferential in the distal to mid calf
- If above mid-calf or on the foot, look for other etiology
- Usually located over the medial malleolus
- Usually shallow, generally irregular margins, usually covered by yellow fibrinous slough
- Slow healing
- IB evidence for procedural intervention



Features of venous and arterial ulcers

	Venous	Arterial
History	History of varicose veins, deep vein thrombosis, venous insufficiency or venous incompetence	History suggestive of peripheral arterial disease, intermittent claudication, and/or rest pain
Classic site	Over the medial gaiter region of the leg	Usually over the toes, foot, and ankle
Edges	Sloping	Punched out
Wound bed	Often covered with slough	Often covered with varying degrees of slough and necrotic tissue
Exudate level	Usually high	Usually low
Pain	Pain not severe unless associated with excessive oedema or infection	Pain, even without infection
Oedema	Usually associated with limb oedema	Oedema not common
Associated features	Venous eczema, lipodermatosclerosis, atrophie blanche, haemosiderosis	Trophic changes; gangrene may be present
Treatment	Compression is mainstay	Appropriate surgery for arterial insufficiency; drugs of limited value

Venous Ulcers



Arterial Ulcers



Therapies for Treatment of CVI: Compression

- Consensus primary treatment: COMPRESSION
- Prevention of venous thromboembolism (DVT or PE)
- Reduction of discomfort and edema
- Reduce the progression of post thrombosis syndrome
- Prevention of skin breakdown, venous ulcers, and skin breakdown
- Compression increases fibrinolysis in the blood, increases reabsorption of fluid into the capillaries, supports incompetent valves, counteracts retrograde flow, increases venous return
- Useful for edema, CVI, DVT/PE prophylaxis, ulcer management, varicose veins, pregnancy, post sclerotic therapy, superficial thrombophlebitis, leg pain, hypertrophic scar, dermatitis, lymph edema

Therapies for Treatment of CVI: Compression

Compression Pressure in mmHg	Indication
10-15	Fatigue, aches
15-20	Pregnancy, post travel, moderate edema, mild varicosities
20-30	Moderate to severe varicosities, edema with pregnancy DVT, ulcer prevention, CVI, thrombophlebitis
30-40	Lymphedema, severe CVI, ulcers (top of it above thigh)

Therapies for Treatment of CVI: Compression

- Types of compression therapy
- Unna boots
- Compression bandages
- Intermittent compression devices
- Casts
- Ace wraps
- Compression garments
- Underappreciated factor: COMPLIANCE vs NONCOMPLIANCE (97% vs 55% complete healing, 29% vs 100% recurrence)

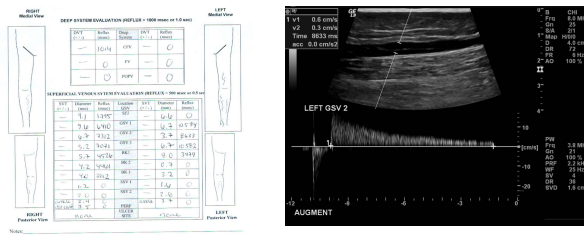
Therapies for Treatment of CVI: Pharmacologic

- ASA – beneficial for wound healing, trials all done with concomitant compression
- Stanozolol – anabolic steroid PO, stimulates fibrinolysis, may be used to treat lipodermatosclerosis
- Pentoxifylline – may be useful as an adjunctive with compression therapy
- Anticoagulation – we tend to use Xarelto more frequently in our clinic, but many options are available

Diagnostic Imaging

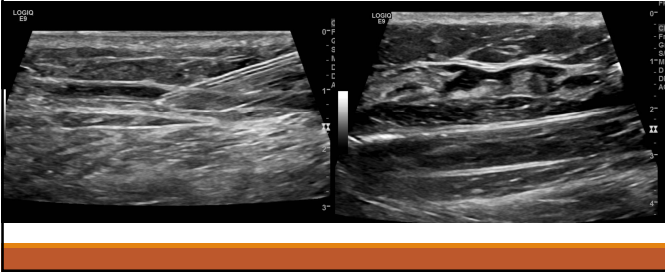
- Duplex ultrasound provides the most functional and anatomic data
 - Superficial peripheral venous reflux > 500 msec, dilated veins > 3 mm
 - Deep venous reflux > 1000 msec
- Role of CT and MRI are not well defined but not very significant in the absence of excluding other etiologies, e.g. May Turner's
- Venography usually not necessary
- ABIs correlate with wound healing, ABI < 0.8 may indicate concurrent arterial and venous disease

Diagnostic Imaging



Management of C4-C6 Disease: Interventional Perspective

Ablation Technologies

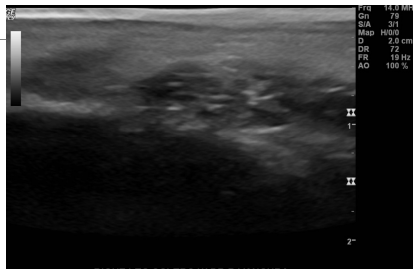


Sclerotherapy

• Various technologies available including STS, polidocanol, hypertonic saline, ethanolamine

• Useful for perforating veins and can also be used in the GSV

• Requires compression after treatment



Surgical Treatments

• GSV stripping

• CHIVA

• SEPS

• Stab phlebectomy

• Venoplasty

• Generally similar outcomes to endovenous ablation, but more invasive
