• Prevelance
• Economic Impact
• Anatomy
• Venous vs other etiology
• Guidelines
• May-Thurner
Significance

• Venous disease 2.5 million Americans (320 m total!)
• Age 40: 22%, Age 50: 35%, Age 60: 41%
• Venous leg ulcers (VLU)
  – Population: 0.6-2.0%
  – Venous disease: 20%
  – Polish study 40,000 patients (1.5%)
  – Edinburgh study 1500 patients 18-64 yo (1%)
  – German study (Bonn Vein Longitudinal Study) 3000 patients 18-79 yo 0.7% open ulcers
  – AVF National Venous Screening Program 2234 patients, advanced disease (CEAP 4-6) in 10%

VLU economic impact

• Treatment costs of 2.5 billion
  • US $2500 per patient, per month
    – Facility costs
    – Professional reimbursement
    – Labor costs (nurses) for wound care
    – Medications
    – Wound dressing/compression stockings
• VLU cause loss of 2 mil working days
• QOL in CEAP 6 pts worse than chronic lung disease, back pain or arthritis
• Treatment days lost, days lost to pain
Economic Impact

• 84 patients from a wound care clinic with CEAP 6
  – In 6 months each patient costs $15,732
  – 60% healed mean time 122 days (6-379), mean cost of $10,563 ($430-$50,967)
  – Outpatient facility fees $10,332, visiting nurse services $11,365
  – 20% did not heal in 6 months, Mean cost $33,907

• EXTENDED WOUND TIME MEANS MORE COST

• Inpatient admission (2/3 treatment for infection resistant to oral antibiotics) mean cost $33,629

• VLU treated with ablation ($11,960) vs best medical therapy ($12,304)
  – reduced recurrence rates (34% to 5%)

• VLU treated for outflow obstruction ($24,241 vs $11,960)
Economic Impact

• Ablation is targeted by insurance companies now, cost savings is in reducing time to heal, avoiding infection/hospitalization
• Wound care feeling pushback?
• Compression devices?
Anatomy

DEEP SYSTEM
SUPERFICIAL SYSTEM

femoral

great saphenous

perforators

small saphenous
Anatomy

Superficial circumflex iliac vein

Femoral vein

Anterolateral vein of thigh

Long saphenous vein

Popliteal vein

Anterior vein of leg

Posterior arch vein

Superficial inferior epigastric vein

Medial vein of thigh

Mid-thigh perforators (Dodd)

Gastrocnemius perforator (Boyd)

Lower leg perforators (Cockett)

Ankle perforators
Venous network

Vein Anatomy

- Femoral Vein (Deep Vein)
- Saphenous Vein (Superficial Vein)
- Perforator Vein (Communicating Vein)
- Varicose Veins
- Reticular Vein (Feeder Vein)
- Spider Veins
- Great saphenous vein
- Great saphenous vein
- Small saphenous vein
- Dorsal venous arch
Perforators = Ulcer

- Perforators?
  - Connect deep to superficial system
  - Many perforators big and small
  - Perforator reflux develops as a primary vein wall problem
  - Then contributes to venous hypertension
Anatomy

• Variation
• 31% of population have duplicated system
  – Duplication
  – Accessory

Basic Venous Disease

Risk Factors

• Three major epidemiologic studies – Tampere, Edinburgh, Framingham

• Pregnancy
  – 28% develop new varicose veins
  – Hydrostatic, hormonal
    • Higher estrogen levels assoc with increased venous distensibility
    • Saphenous vein contains estrogen and progesterone receptors
  – Prevalence of varicose veins with pregnancy
    • 0 – 32%
    • 1 – 38%
    • 2 – 43%
    • 3 – 48%
    • 4 – 59%
Risk factors

- **Family History**
  - 20% unaffected parents
  - 25-62% one affected parent
  - 90% two affected parents
- **Occupation** – all three studies demonstrated significance
  - Sitting vs standing prevalence of varicose veins 27% vs 36%
- **Gender**
- **Age**
- **Obesity?**
  - Women only, BMI > 30
  - Adipose tissue disrupts the cutaneous network

Pathology

• Primary (congenital) vein wall weakness
  – Focal defect in wall integrity
  – Normal collagen matrix is disrupted
  – Matrix provides strength, elastasia, compliance, tone
  – Histology: vv have no muscle fibers, proliferative collagen matrix
  – So maybe pressures are normal, but diseased vein cannot handle these normal pressures?
Digging deeper

• Intrinsic genetic defect of collagen synthesis?
• Smooth muscle cells from varicose veins demonstrate increased expression of Type 1 collagen, decreased Type 3 collagen

= Connective Tissue Disease?!?!
Inflammatory Changes

- WBC trapping due to decreased blood flow
- CVI patients have 28.6% decrease in circulating WBCs
- WBC accumulate and release toxic oxygen metabolites and proteolytic enzymes leading to capillary damage, increased permeability, fibrin cuff formation, and microlymphatic damage
- VV: increased T-lymphocytes and macrophages
- LDS patients have increased WBC in tissue specimens
- LDS elevated proinflammatory cytokines IL-1a and IL-1b
Microcirculatory Changes

- Decreased capillary counts, increased capillary diameters, and endothelial damage
- Increased capillary permeability leads to accumulation of fluid, macromolecules (proteins), and extravasated RBCs in the interstitial space
- Pericapillary fibrin cuff formation leads to decreased oxygen diffusion
- Fibrin deposition inhibits new collagen formation which impairs healing and leads to fibrosis
- Fragmentation and destruction of microlymphatics
CVI: Disease Progression
CEAP 4-6

- Hyperpigmentation
- Venous Eczema
- Lipodermatosclerosis
- Atrophie Blanche
- Venous Ulcer
CEAP Classification

- **CLINICAL CLASSIFICATION**
  - C0: no visible or palpable signs of venous disease.
  - C1: telangiectasies or reticular veins.
  - C2: varicose veins.
  - C3: edema.
  - C4a: pigmentation and eczema.
  - C4b: lipodermatosclerosis, atrophie blanche.
  - C5: healed venous ulcer.
  - C6: active venous ulcer.
  - S: symptoms including ache, pain, tightness, skin irritation, heaviness, muscle cramps, as well as other complaints attributable to venous dysfunction.
  - A: asymptomatic.

- **ETIOLOGIC CLASSIFICATION**
  - Ec: congenital.
  - Ep: primary.
  - **Es:** secondary (postthrombotic).

- **ANATOMIC CLASSIFICATION**
  - s: superficial veins.
  - p: perforator veins.
  - d: deep veins.

- **PATHOPHYSIOLOGIC CLASSIFICATION**
  - Pr: reflux.
  - Po: obstruction.
  - Pr,o: reflux and obstruction.
  - Pn: no venous pathophysiology identifiable.
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Edema

- Due to capillary damage and increased permeability
- Macromolecule accumulation in interstitial space
- Begins in perimalleolar region and ascends up the leg
- Edema leads to pain by increasing intracompartmental pressure and subcutaneous volume and pressure
- Venous distension leads to tenderness along varicosities
- Lymphatic damage leads to lymphedema
Hyperpigmentation

- Red-brown color changes in gaiter distribution
- Extravasated erythrocytes, hemosiderin-laden macrophages, and melanin deposition
- Xerosis (dry skin) and pruritis often associated and may lead to venous eczema
Venous Eczema

- Very pruritic condition
- Usually begins at the ankle - medial malleolus
- Begins as sharply demarcated erythematous papules and vesicles
- Eventually becomes diffuse, poorly defined and may demonstrate serous exudate and crust
- Less often warm to touch (ddx: cellulitis)
- Contact dermatitis and secondary infection
Lipodermatosclerosis (LDS)

- Acute LDS
  - Painful, erythematous/purple well demarcated indurated plaques
  - Warm and tender to the touch
- Chronic LDS
  - Inverted champagne bottle appearance distal lower leg
  - Hyperpigmentation and fibrosis of dermal and subcutaneous tissue
  - Highly associated or exclusive to CVI
LDS: Skin Architecture Changes

- Subcutaneous fat necrosis, lymphohistiocytic infiltrate and septal fibrosis
- Tissue hypoxia and WBC activation stimulates transforming growth factor (TGF) production which accelerates tissue fibrosis
- Leads to extracellular matrix turnover and eventually venous ulceration
Livedo Reticularis

- AKA Livedoid Vasculitis
- Cutaneous infarction
- Atrophic stellate cutaneous scar dotted with dilated capillaries centrally and peripherally
Livedo Reticularis

- 99% have chronic venous insufficiency
- Pericapillary fibrin cuffs impede oxygen diffusion
- WBC trapping theory: release destructive proteolytic enzymes and superoxide metabolites
- Microthrombi in capillaries
Venous Ulcer

- 80% of lower extremity ulcers are venous in nature
- 40% of patients have an open ulcer for over one year
Venous Ulcers

- Microlymphangiopathy, dilatation and elongation of capillaries, occlusion of capillaries by microthrombi or WBCs, reduction in capillaries, leakage of plasma proteins and RBCs
- Capillary changes lead to reduction of blood flow, sludging, microthrombi and occlusion of capillaries
- Protein-rich edema and fibrin formation leads to decreased oxygen diffusion
- Altered oxygen supply and demand
Ulcer formation

- Due to extensive inflammatory response
- Tissue hypoxia, shift in fluid shear forces of the endothelium
- Capillary leukocytes, mast cell migration, connective tissue lymphocytes, oxygen free radical production, = parenchymal cell death

Medial Malleolar = Venous Ulcer
DDX: Lower Extremity Ulcers
Rash ???
DIAGNOSIS

- Must order an ultrasound to \textit{“Evaluate for GSV reflux”}
- Evaluate lower ext veins
- Vein disease
- Lower ext edema
- Leg pain

r/o DVT = Deep system
Ultrasound

• Tech doing the ultrasound is critical
• False negative due to
  – Positioning
  – Hydration
  – No one knows!!!
• If ultrasound results are not what is expected clinically – repeat
• Tilt 15/stand, warm room, hydrated patient
• Veins are dynamic!!
Ultrasound

- Identify the GSV by the saphenous compartment
Ultrasound

• Identify the Saphenofemoral junction, follow saphenous vein south
Ultrasound

Saphenofemoral junction
AKA SFJ
“junctional reflux”
Ultrasound
Ultrasound

Venous Reflux Examination

PATIENT NAME: Lisa Aupeperle-hutchins
DATE: 08/27/2014
PHYSICIAN: MARY COSTANTINO, MD

No GSV reflux laterally

No DVT

No RV reflux

Vein reflux at 5.6 sec.

Venous reflux 10 cm, reflux >6 sec.

No DVT
Ultrasound

• Take away points:
  – Order if you can be confident where ever you send patient is going to rule out superficial disease
  – Few people are really trained to do venous ultrasound

• I never trust anyone else’s ultrasound
• Ultrasound is used for surgical planning
Treatment

• Ablation
  – Standard of care
  – Works
  – Fast
  – Less expensive
  – No anesthesia
  – Office setting
  – Essentially no recovery
• “Stripping”
  – No reason to do this
• Compression
  – Works
  – Temporary relief
  – Proven best treatment for ulcers
• “Nothing to do about it”
  – What most patients are told
Treatment

• Remember that this is an elective procedure
  – Uncover the disease
  – Get the patient in compression stockings
  – Patients can decide if treatment is something they want to pursue
Compression

- 20-30 mm Hg
- Patients need a lot of guidance
- Thigh high, open toe
- Insurance covers to varying degree
- Range from 10-100$
- Last about a year with semi-regular use
- Everyone should have a pair
My opinion:
-it doesn’t matter which one is used, as long as patient is treated with ablation, and not stripping
-don’t listen to anyone who touts ‘superior technology’
-this is not an area where the latest and greatest device is better
-company selling points is how much marketing they will do for you
Success

• Ablation
  – 355 limbs
    • Technical success 100%
    • GSV occluded
      – 229/229 1 week (100%)
      – 202/203 1 month (99.5%)
      – 157/158 3 month (99.3%)
      – 99/99 6 months (100%)
      – 41/41 1 year (100%)

• Brusing 21%, pain 15%, paresthesia 4%

Jung Ah Park, Sang Woo Park, Il Soo Chang, Jae Joon Hwang, Song Am Lee, Jun Seok Kim, Hyun Keun Chee, Ik Jin Yun, The 1470-nm Bare-Fiber Diode Laser Ablation of the Great Saphenous Vein and Small Saphenous Vein at 1-Year Follow-up Using 8-12 W and a Mean Linear Endovenous Energy Density of 72 J/cm. JVIR , in press.
Procedure

- In office
- Not stressful
- No surgery center or hospital charges
- Family welcome to stay
- Versed 7 mg oral sedation
- No sedation
Ablation

Source: SIR
Ablation
Consensus Guidelines

Consensus Guidelines

• 2.2: A basic practical knowledge of venous physiology and venous leg ulcer pathophysiology for all practitioners caring for venous leg ulcers
• Guideline 3.2: Recommend identification of nonvenous causes of ulcers
• Guideline 3.9: Recommend comprehensive venous duplex ultrasound in all patients with suspected venous ulcer.
• Guideline 3.11: CTV, MRV, venography, or IVUS in patients with suspected VLU if advanced venous diagnosis is required for iliac vein obstruction.
Guideline 3.12: Recommend that all patients with VLU be classified by CEAP, VCSS, and venous QOL assessment.
• Guideline 5.1: In all patients with VLU recommend compression over no compression to promote healing.
  – 9 RTC
• Guideline 5.2: In all patients with VLU recommend compression to decrease ulcer recurrence.
Guideline 6.1: In all patients with ACTIVE ulcer recommend ablation. (CEAP 6)
Guideline 6.2: In all patients with VLU recommend ablation to decrease ulcer recurrence.
Guideline 6.3: In all patients with HEALED VLU recommend ablation. (CEAP 5)
Guideline 6.4: In all patients with skin changes at risk for VLU recommend ablation. (CEAP 4)
Procedure

• Quick ultrasound to confirm original findings
• Prep and drape leg
• Access into GSV using ultrasound
• Thread fiber to junction
• Pull fiber back 2.0 cm from the junction
• Tumusent
• Turn fiber on, and pull catheter out, ablating along the way
• 25 minutes
Tumuscent

• 950 cc lactated ringers, 50 cc 1% lidocaine
• Three purposes
  – Anesthesia
  – Collapses vein around fiber
  – Heat sink around vein to prevent damage to surrounding tissue
• NOT JUST PAIN CONTROL
• Lesson learned: cases in OR without tumescent, because patient is under anesthesia
Procedure

• Additional procedures
  – Second vein
    • Accessory
    • Lesser saphenous
  – Phlebectomy
    • Watchful waiting vs same day
  – Ultrasound guided sclerotherapy

– Often insurance dictates what is done
– No right answer, depends on set up, patient population, physician preference
Venous Ulcer Treatment

  - ESCHAR Study - RCT of venous surgery + compression vs. compression alone in C6 patients
  - 12 month ulcer recurrence rate 12% vs. 28%

- JVS 2012; 55:446-50
  - Combined percutaneous therapy vs. compression alone in C5 patients
  - 12 month ulcer recurrence rate 4.8% vs. 67%
Ablation before and after

Painful weekly debridement

Weeks later...
Ablation before and after

Ulcer for 3 years

Weeks later…
May-Thurner
(Iliac Vein Obstruction)
Common iliac vein compressed  Common iliac vein stented
IVUS Helps Discover May-Thurner Syndrome

IVUS Imaging of Normal Iliac Vein

IVUS Imaging of Compressed Iliac Vein
IVUS catheter
May Thurner

- Unilateral LEFT disease
- Extensive left leg thrombus
- Left iliac thrombus, unprovoked
Cases
Summary

- Cost a lot
- Hurts
- Recurr
- Multidisciplinary treatment with wound care and compression
- Send for ablation
Livedo Reticularis
Thank you