



CARDIOVASCULAR

HEALTH CLINIC

Focused on Excellence | Focused on YOU

Introduction to Vascular Surgery

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Common Diagnoses, presentation, clinical work up and treatment plan

Peripheral Arterial Disease
Peripheral Venous Insufficiency



Objectives and Goals

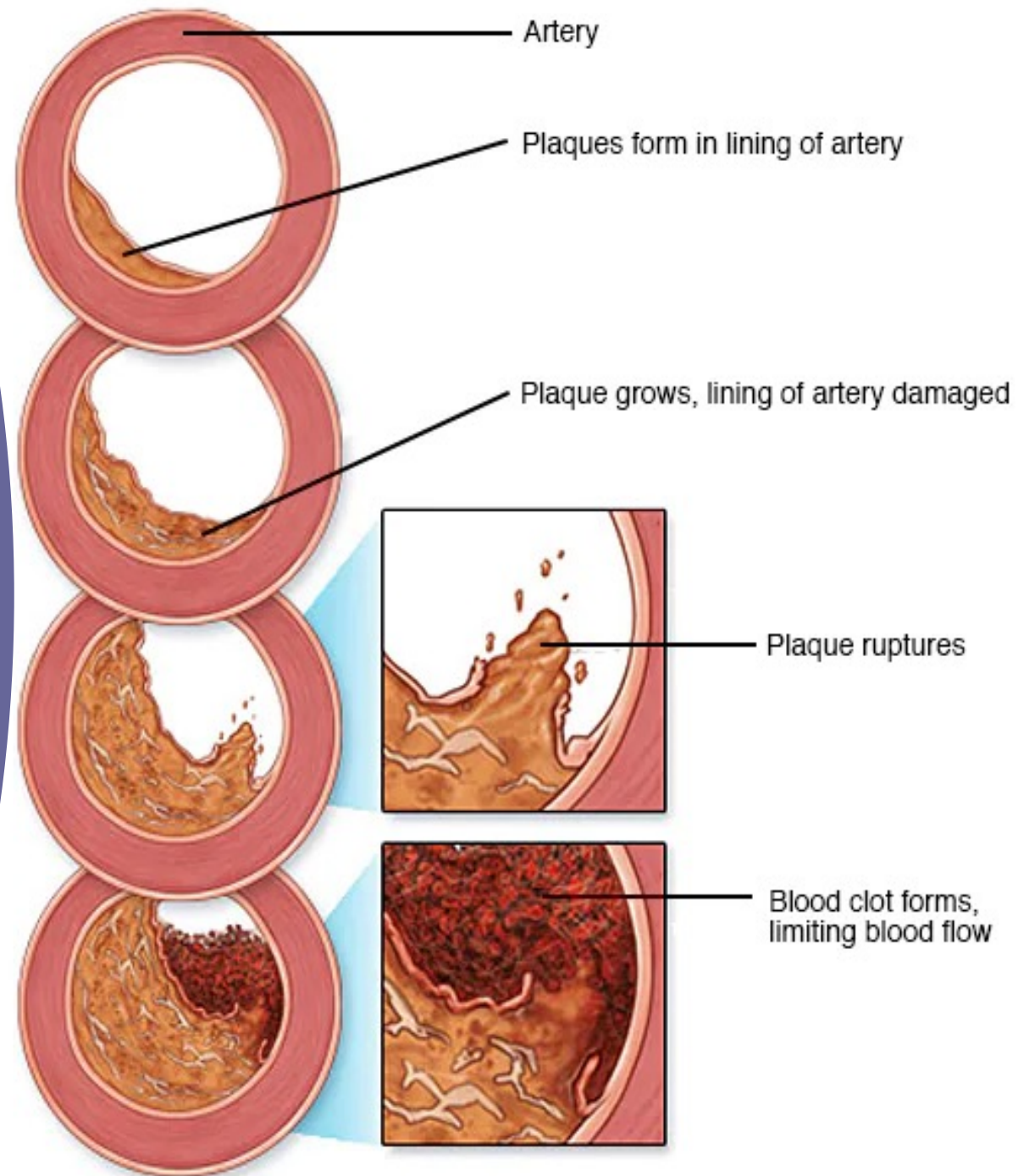
- At the end of this presentation you will be able to and feel more comfortable with:
 - Recognize immediately when a patient has PAD or PVI
 - Know the basic work up for these common vascular presentations
 - differentiate between the various causes of leg wounds and leg pain
 - partner with me to decrease amputation rates and increase awareness of PAD
 - optimize your patient from a medical standpoint and modify risk factors



**PERIPHERAL ARTERIAL
DISEASE
PAD**

Atherosclerosis

- Accumulations of large amounts of cholesterol ester in the arterial wall
- Formation of complex advanced plaque
- Plaque formation and evolution is dynamic and spans decades
- Inflammatory changes within the cap causes vulnerability to erosion or ulceration





Atherosclerotic related cardiovascular disease

*Leading Cause of Death in every region of the world

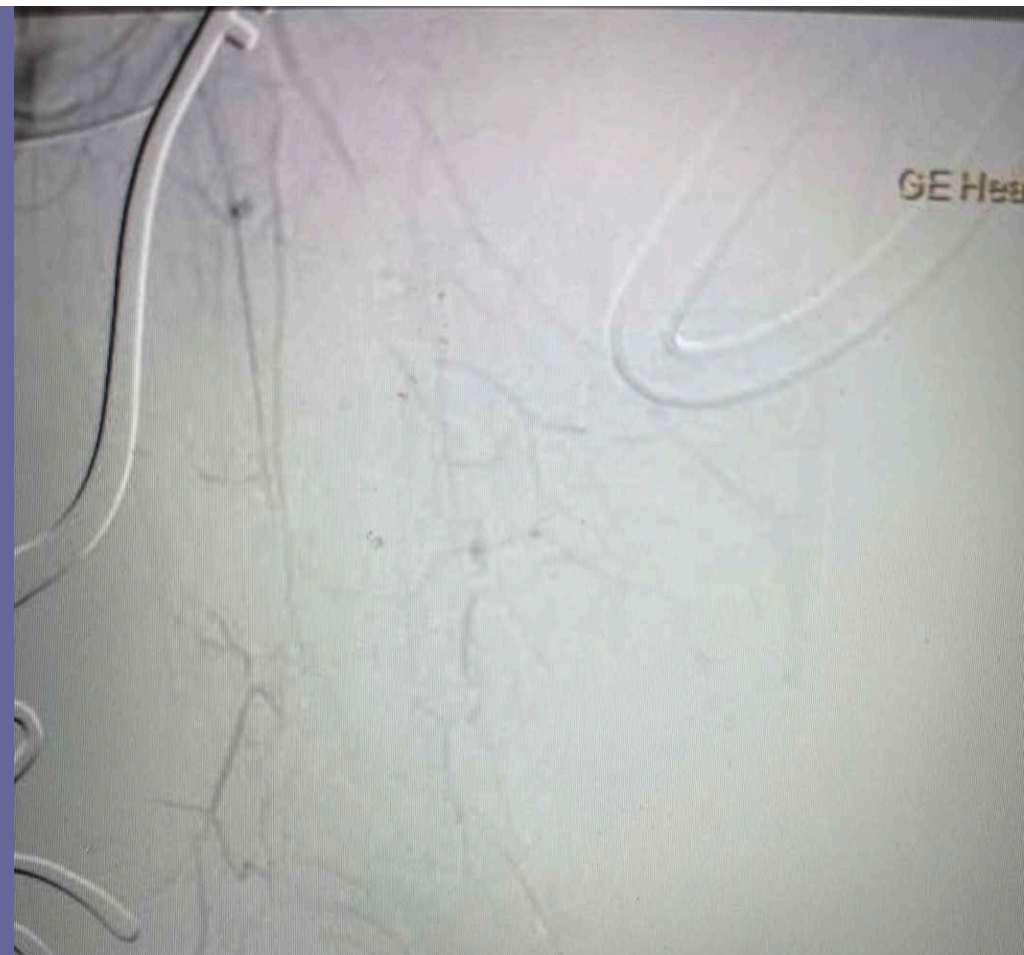
-Over the last few decades CHD age specific death rates fell by greater than 40% in high income countries

-80M + Americans have CVD with annual cost over \$400 Billion!

-8 Million Americans have PAD

-PAD disproportionately affects African Americans

- High Prevalence of cardiovascular risk factors in children and young adults:
- Sedentary Lifestyle, abdominal obesity, poor diets contribute to dyslipidemia and HTN



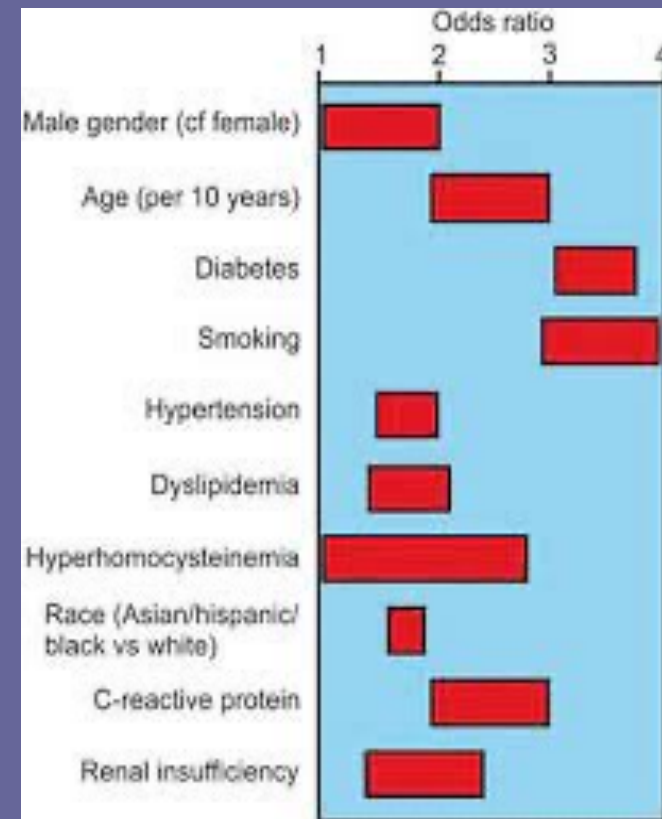
29 y/o F Heavy Smoker



29 y/o F

Risk Factors for Atherosclerosis

- Advanced age
- Race (Non-Hispanic Blacks)
- Male gender
- Hyperfibrinogenemia
- Diabetes Mellatus
- Hyperhomocysteinemia
- Smoking
- Hypertension
- Smoking
- Hypercoagulability
- HTN
- Elevated C –reactive Protein
- Dyslipidemia
- Chronic Renal Insufficiency



Stages of Chronic Limb Ischemia

Grade	Category	Clinical description	Objective criteria
0	0	Asymptomatic-no hemodynamically significant occlusive disease	Normal treadmill or reactive hyperemia test
I	1	Mild claudication	Completes treadmill exercise; AP after exercise > 50 mmHg but at least 20 mmHg lower than resting value
	2	Moderate claudication	Between categories 1 and 3
	3	Severe claudication	Cannot complete standard treadmill exercise, and AP after exercise < 50 mm Hg
II	4	Ischemic rest pain	Resting AP < 40 mmHg, flat or barely pulsatile ankle or metatarsal PVR; TP < 30 mm Hg
III	5	Minor tissue loss non-healing ulcer, focal gangrene with diffuse pedal ischemia	Resting AP < 60 mm Hg, ankle or metatarsal PVR flat or barely pulsatile; TP < 40 mm Hg
	6	Major tissue loss-extending above TM level, functional foot no longer salvageable	Same as category 5

AP: ankle pressure; PVR: pulse volume recording; TM: transmetatarsal; TP: toe pressure.



Differential Dx of Intermittent Claudication



Table 103-2 Differential Diagnosis of Intermittent Claudication

Condition	Location	Prevalence	Characteristic	Effect of Exercise	Effect of Rest	Effect of Position	Other Characteristics
Calf IC	Calf muscles	3%-5% of adult population	Cramping, aching discomfort	Reproducible onset	Quickly relieved	None	May have atypical limb symptoms on exercise
Thigh and buttock IC	Buttock, hip, thigh	Rare	Cramping, aching discomfort	Reproducible onset	Quickly relieved	None	Impotence May have normal pedal pulses with isolated aortoiliac disease Also may present as numbness
Foot IC	Foot arch	Rare	Severe pain on exercise	Reproducible onset	Quickly relieved	None	
Chronic compartment syndrome	Calf muscles	Rare	Tight, bursting pain	After significant exercise (e.g., jogging)	Subsides very slowly	Relief with elevation	Typically affects heavily muscled athletes
Venous claudication	Entire leg, worse in calf	Rare	Tight, bursting pain	After walking	Subsides slowly	Relief speeded by elevation	History of iliofemoral deep venous thrombosis, signs of venous congestion, edema
Nerve root compression	Radiates down leg	Common	Sharp lancinating pain	Induced by sitting, standing, or walking	Often present at rest	Improved by change in position	History of back problems Worse with sitting Relief when supine or sitting
Symptomatic Baker's cyst	Behind knee, down calf	Rare	Swelling, tenderness	With exercise	Present at rest	None	Not intermittent
Hip arthritis	Lateral hip, thigh	Common	Aching discomfort	After variable degrees of exercise	Not quickly relieved	Improved when not weight bearing	Symptoms variable History of degenerative arthritis
Spinal stenosis	Often bilateral buttocks, posterior leg	Common	Pain and weakness	May mimic IC	Variable relief, but can take a long time to recover	Relief by lumbar spine flexion	Worse with standing and spine extension
Foot/ankle arthritis	Ankle, foot arch	Common	Aching pain	After variable degrees of exercise	Not quickly relieved	May be relieved by not bearing weight	Variable; may relate to activity level and present at rest

IC, intermittent claudication.

Adapted from Norgren L, Hiatt WR, Dormandy JA, et al. TASC II Working Group. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). *J Vasc Surg.* 2007;45 Suppl S:22A.

Critical Limb Ischemia

- 46 y/o F
- Buerger's Disease
- Atherosclerosis
- Heavy tobacco

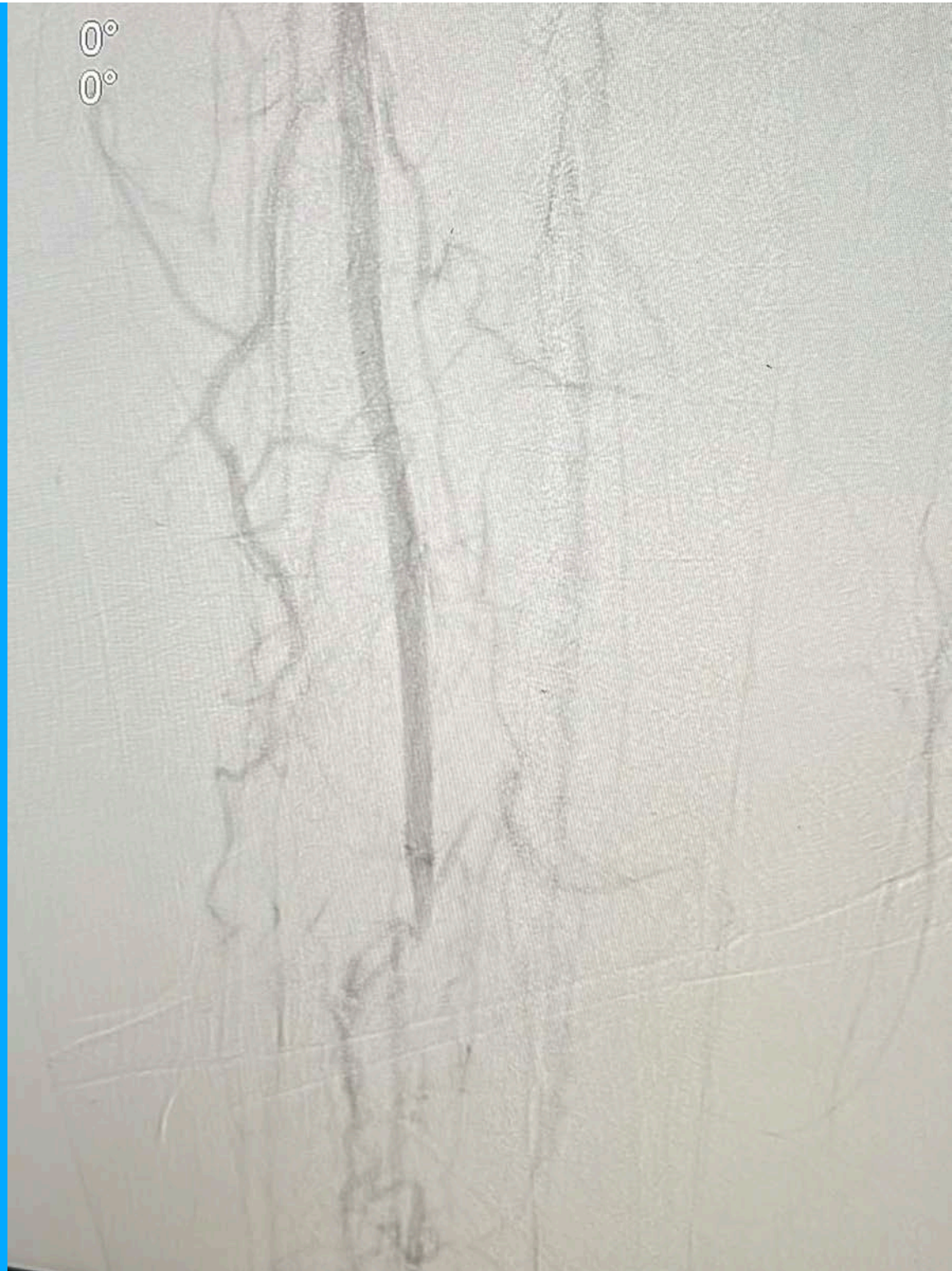


Diagnostic Angiograms





Diagnostic Angiograms

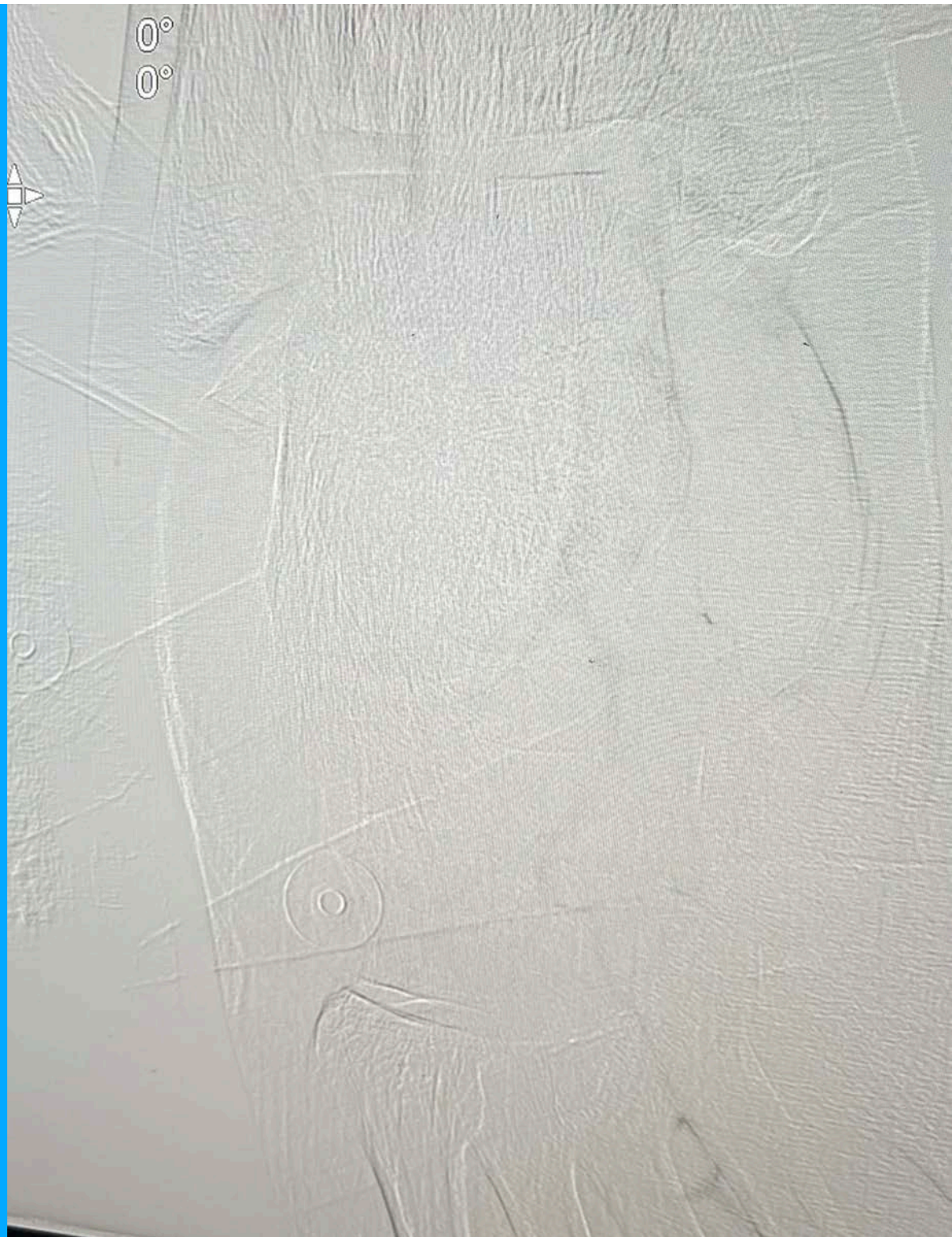




Diagnostic Angiograms



Diagnostic Angiograms





**Surgery-
below
knee
popliteal
artery to
dorsalis
pedis
artery
bypass**



Doppler evaluation post bypass





CLI Overview

- Critical limb ischemia or popularly known as CLI is an arterial blockage or usually multiple levels of blockages in the arteries of the lower extremities, which reduces arterial blood flow in that particular body part.
- CLI is an advanced stage of peripheral artery disease (PAD) and is defined as ischemic rest pain, arterial insufficiency ulcers, and gangrene. (Rutherford 4+)
- CLI can results in severe pain in the feet or toes, where the blood flow is minimal even while resting.
- Most cases are the progressive result of atherosclerosis



Risk Factors

- Hypertension
- Hypercholesterolemia
- Tobacco
- Diabetes

- Less commonly: Buerger's, thromboangitis obliterans, arteritis, emboli/thrombosis

Risk Factors--Diabetes

- Atherosclerosis develops at a younger age and progresses rapidly
- Frequently associated with severe disease
- Atherosclerosis affects more distal vessels less amenable to traditional revascularization
- Atherosclerosis in the distal arteries in combination with diabetic neuropathy contribute to the higher rate of limb loss





Clinical Correlation :

ABI of 0.4 or less

Ankle systolic pressure of 50
mmhg or less

Toe systolic pressure 30
mmhg or less

Typical physical findings:

Absent or diminished pedal
pulses

Shiny smooth skin

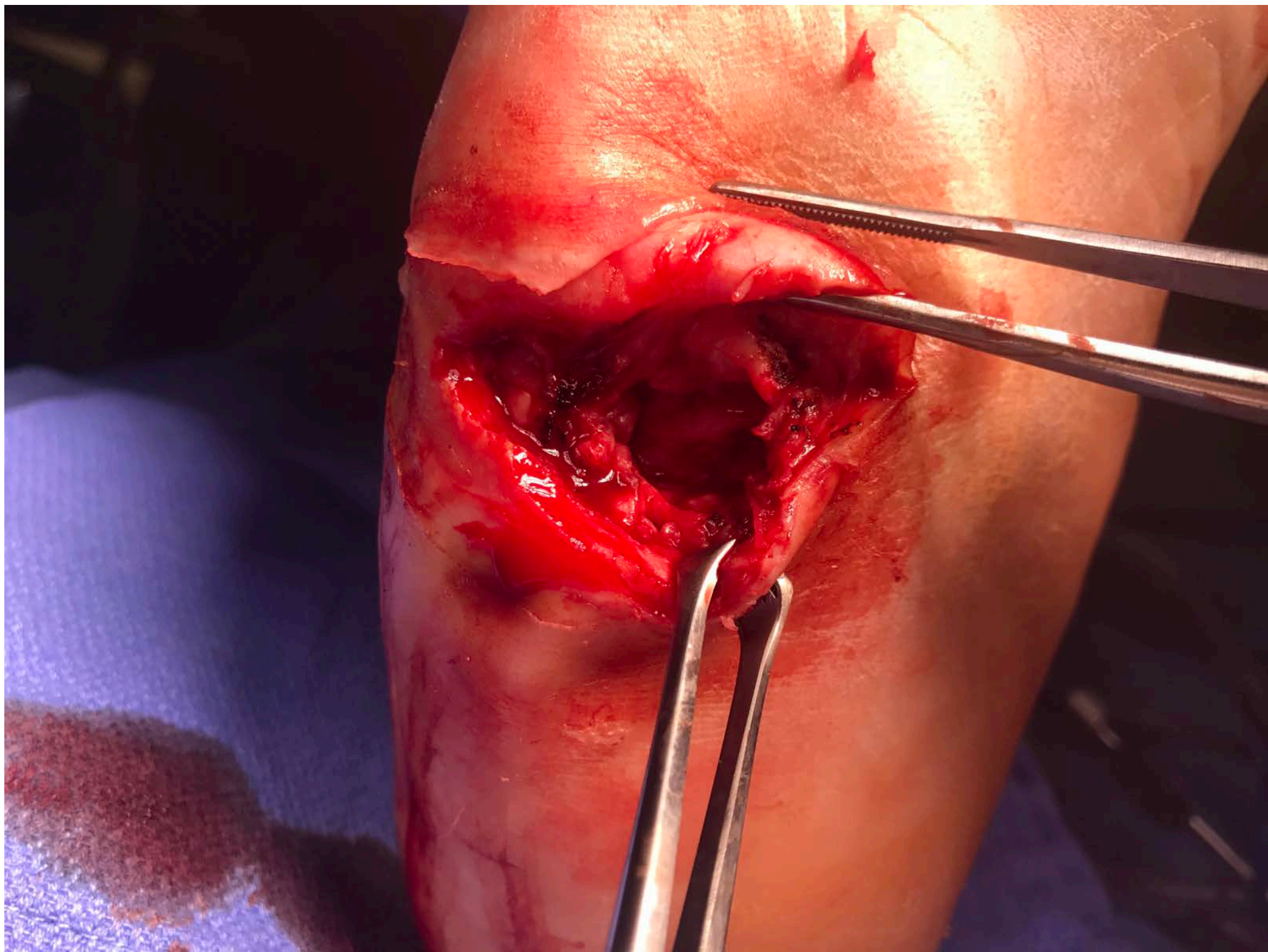
Muscle wasting of the calves

- Warning signs:
- Progressive gangrene
- Rapidly enlarging wounds
- Continuous ischemic rest pain

Non healing wounds

- Usually found in areas of foot trauma
- Failure to respond to a 4-12 week trial of conservative therapy
- Gangrene usually is found or starts on the toes
- Pts with chronic CLI have a 3 year limb loss rate of 40%








Conservative Treatment

- Risk Factor Modification:
 - smoking cessation
 - BP Control
 - glycemic control
 - reduction of lipid levels
- Medicine
 - Antiplatelets: asa substantially decreases the risk of MI, Stroke and death in pts with PVD, also reduces the rate of arterial reocclusion after angioplasty and bypass grafting



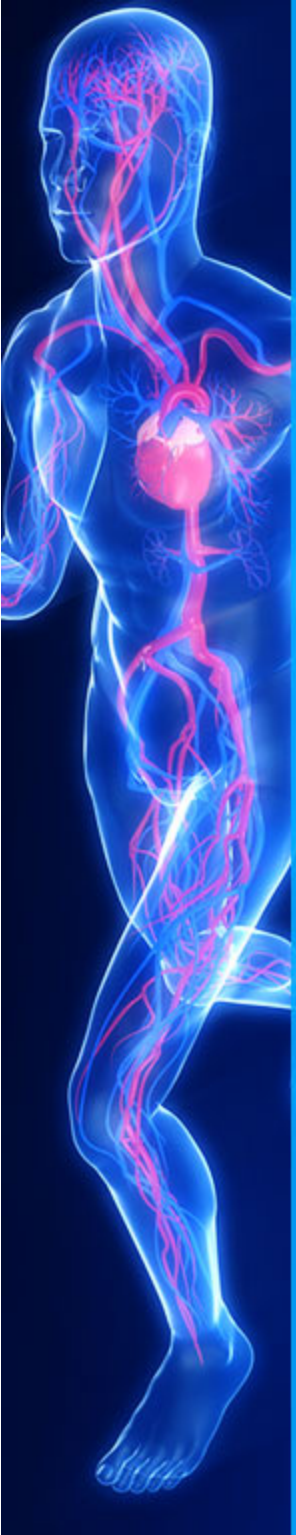
Rivaroxaban and ASA in PVD-COMPASS Trial

- Large multicenter international randomized control trial
- COMPASS trial recently demonstrated that rivaroxaban 2.5mg BID + ASA daily significantly reduces major cardiac limb events in patients with PAD.
- Of all the trials assessing antithrombotic mgmt in PAD. ASA and rivaroxaban 2.5mg BID is the only regimine, when compared to asa alone, which demonstrates reductions in BOTH MACE and MALE in the setting of stable PAD, while also maintaining an acceptable safety profile

Ischemic Rest pain

- Correct underlying systemic contributors
- Pain control prn
- If pain persists after 4-8 weeks of conservative tx and optimization, surgical intervention should be discussed





Surgical Intervention: Revascularization Vs Amputation

- **Revascularization**

- More cost effective
- Associated with better perioperative morbidity and mortality

- **Amputation**

- Long term cost over lifetime 500k
- Mortality:
 - 13-40% in 1 year
 - 35-65% in 3 years
 - 39-80% in 5 years

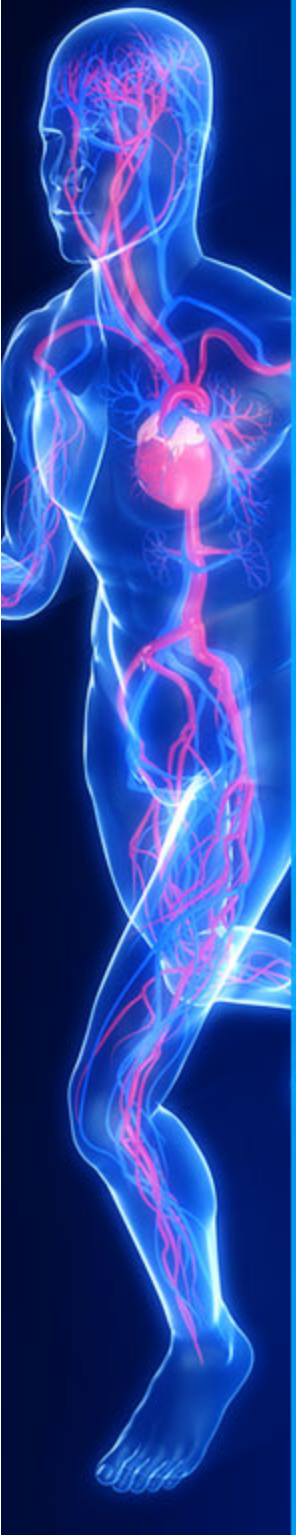
Amputation stat pearls

Every three min in America-a limb is lost due to DM

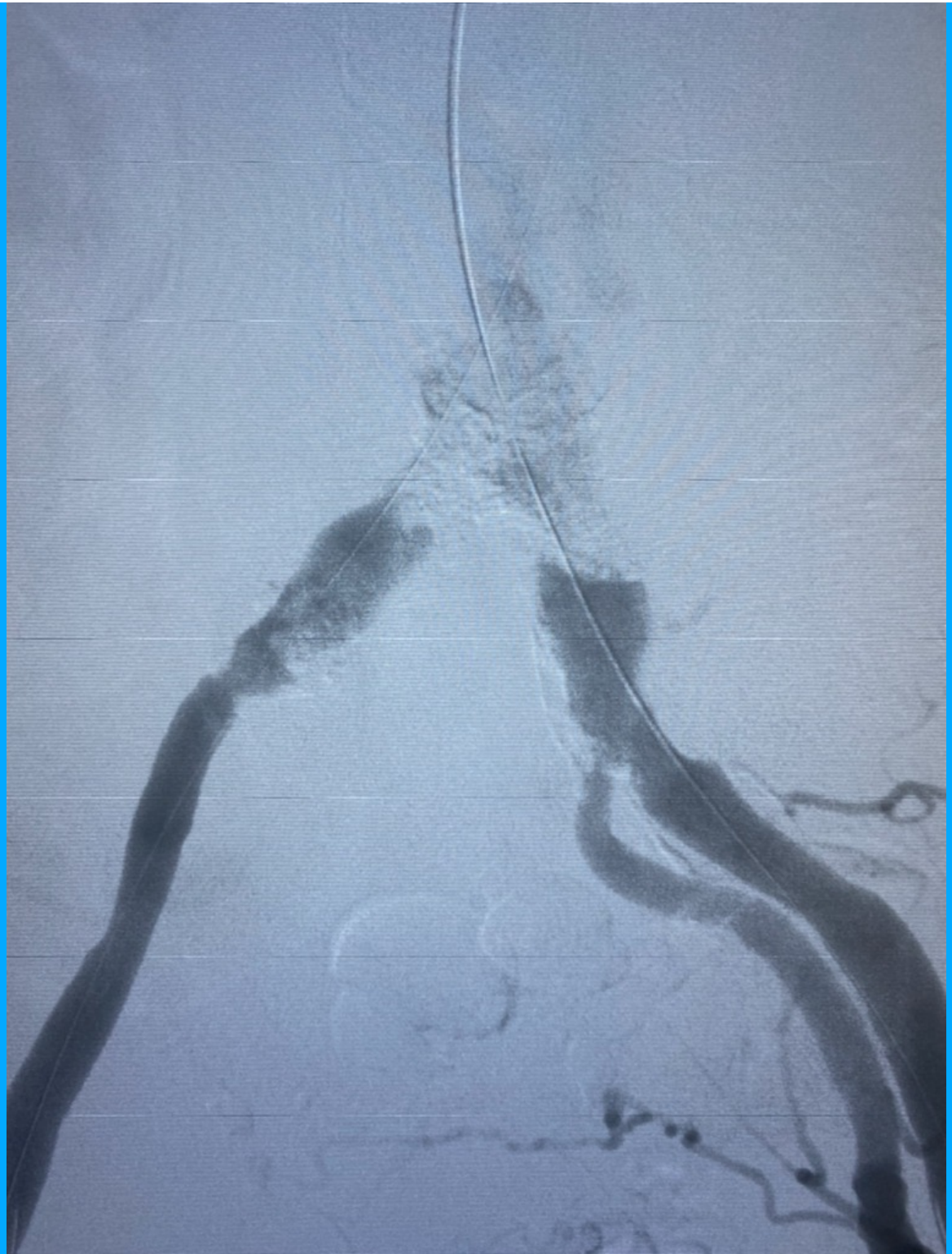
Over 154,000 amputations occur every year with the majority of these being preventable (up to 85%)

Major amputations increased from 2009-2015 by 50%

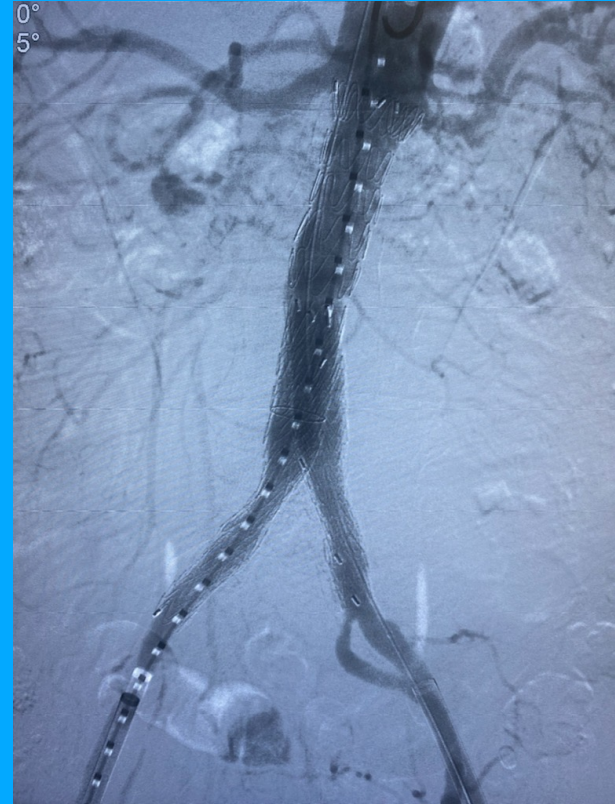
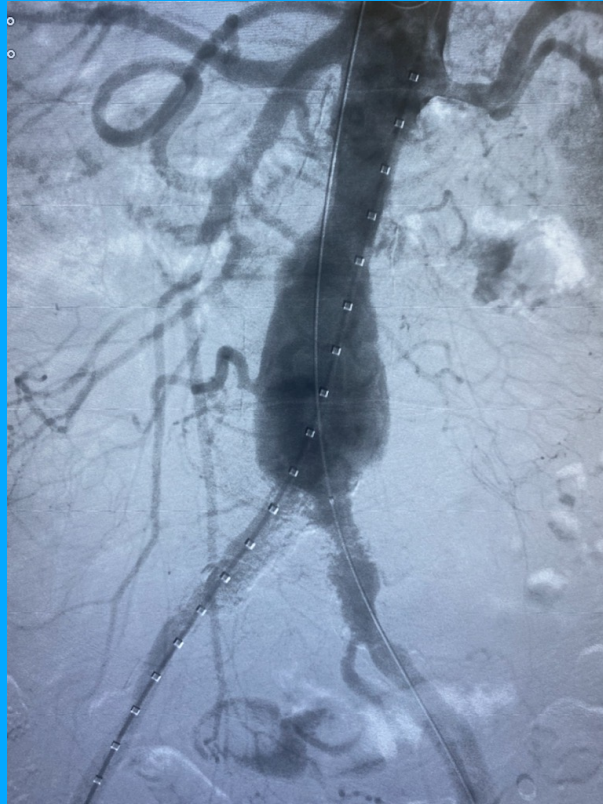
85% of the amputations worldwide were the result of a diabetic foot ulcer



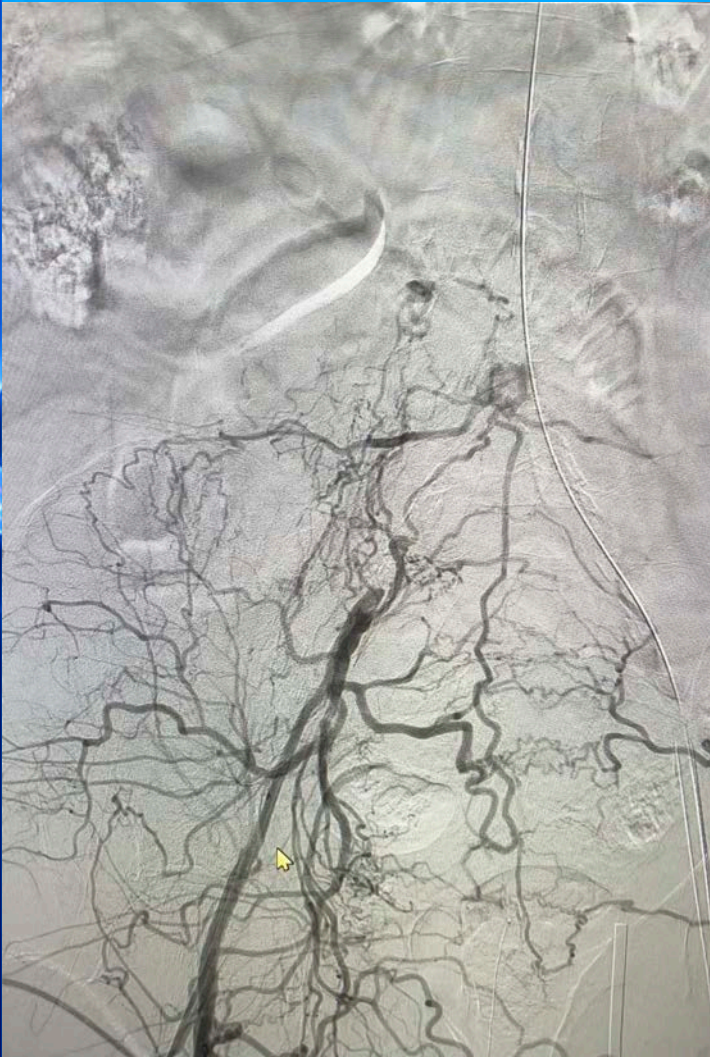
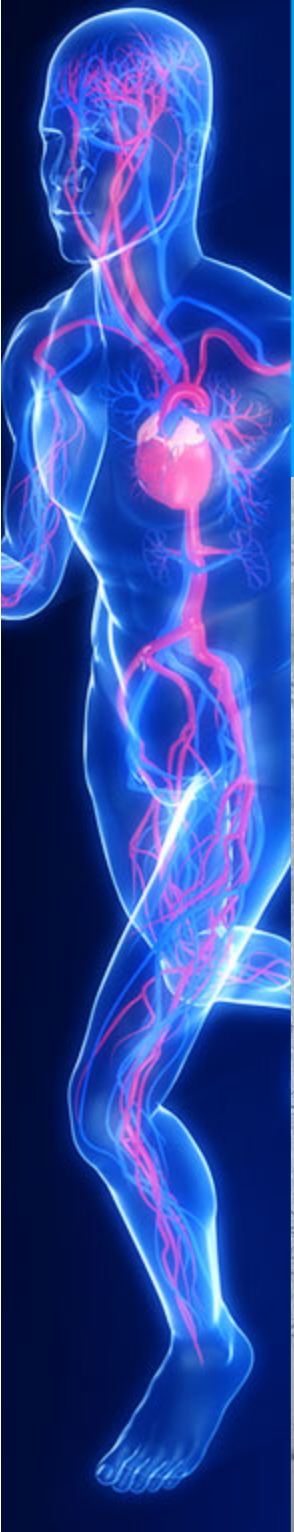
**Case Example:
67 y/o M poor
open surgical
candidate CLI
with rest pain
and infrarenal
AAA**



CLI WITH REST PAIN AND AAA



**Case
example: 63
y/o F CLI with
rest pain RLE**



CLI with rest pain 63 y/o F



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Pedal Access- Posterior Tibial Artery

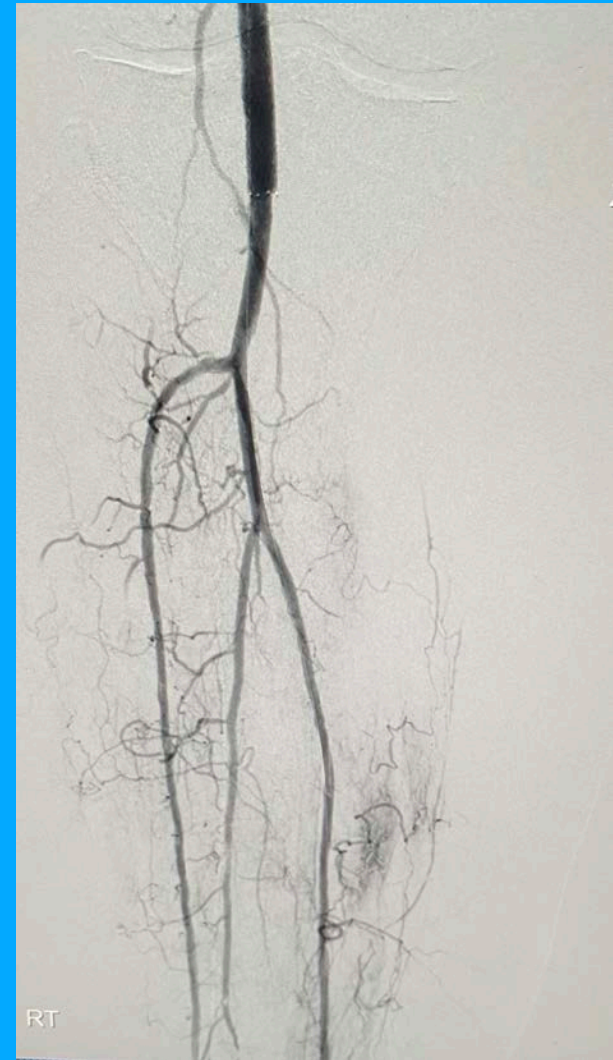


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Pedal Access- Anterior tibial Artery

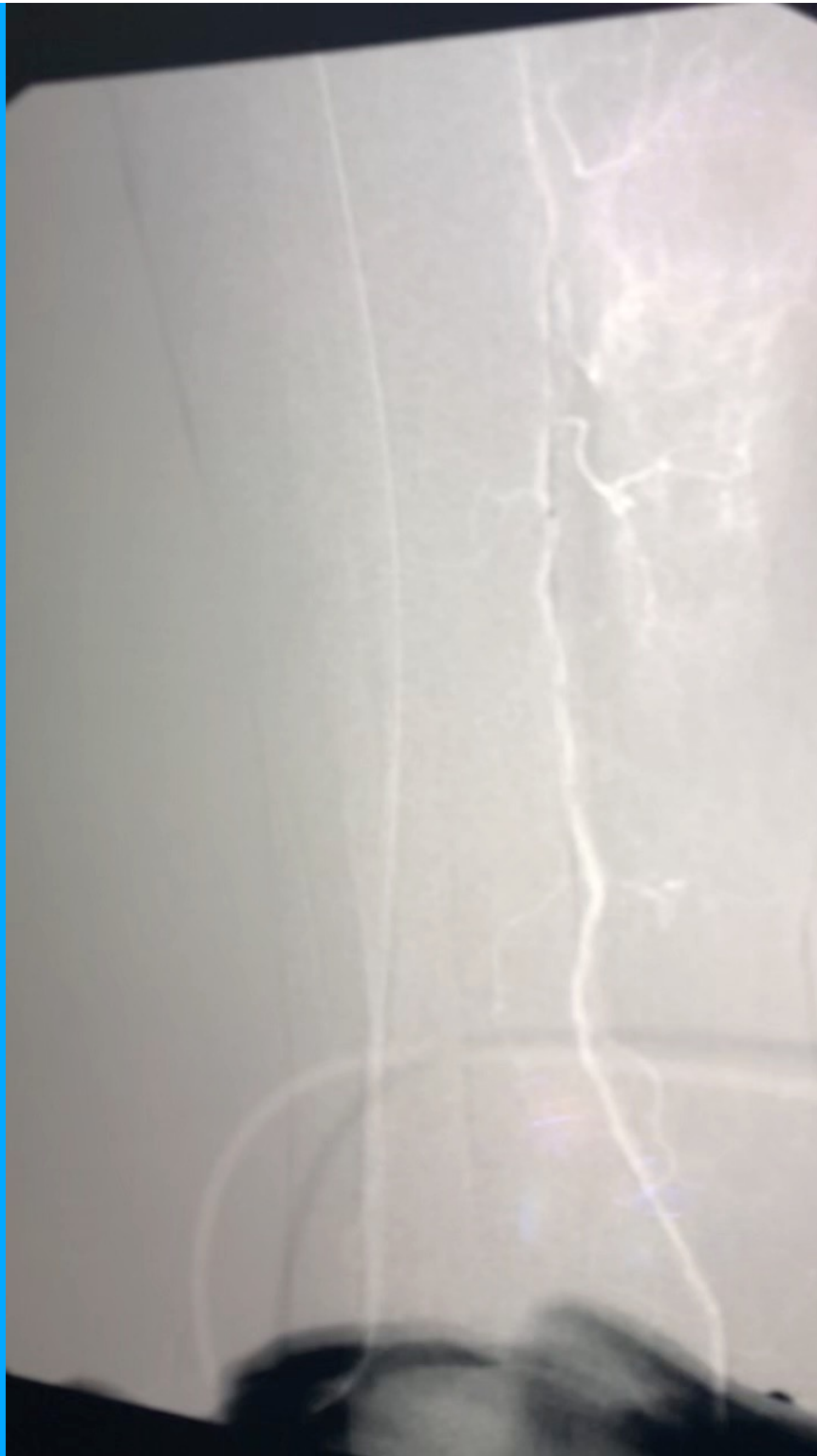


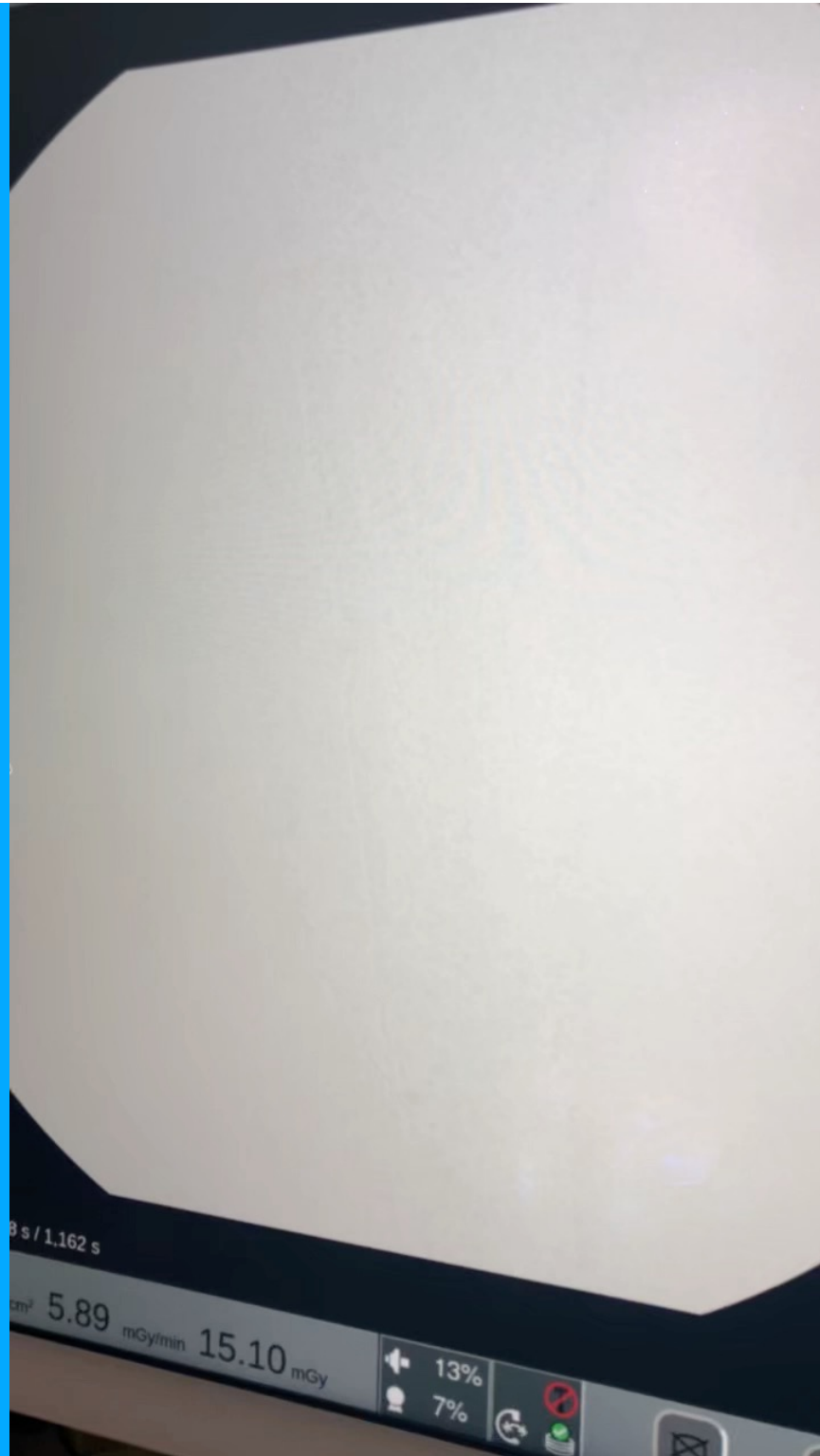
87 y/o F CLI rest pain with discoloration



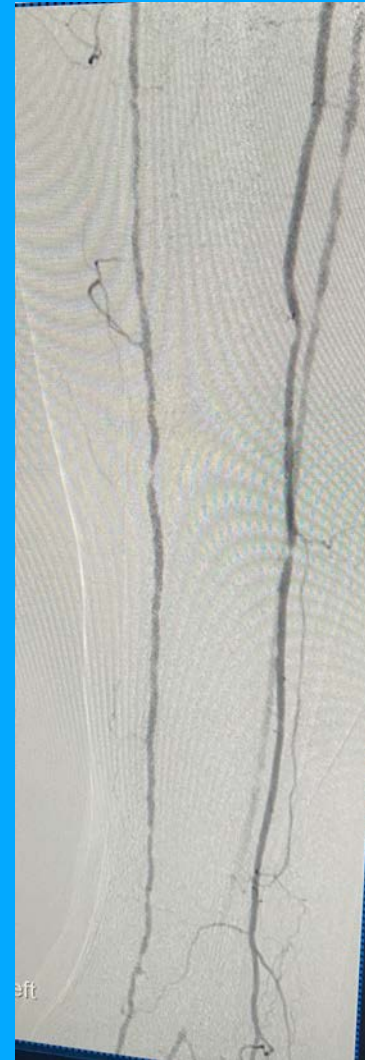
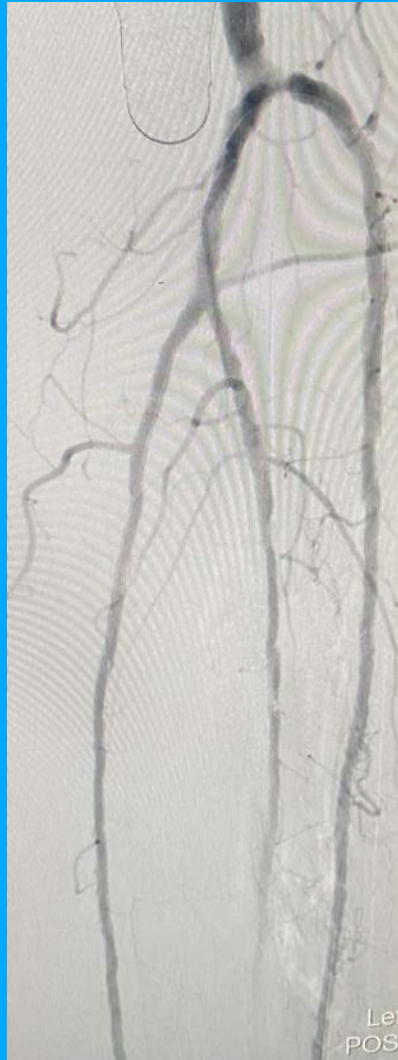
58 y/o M DM foot wound



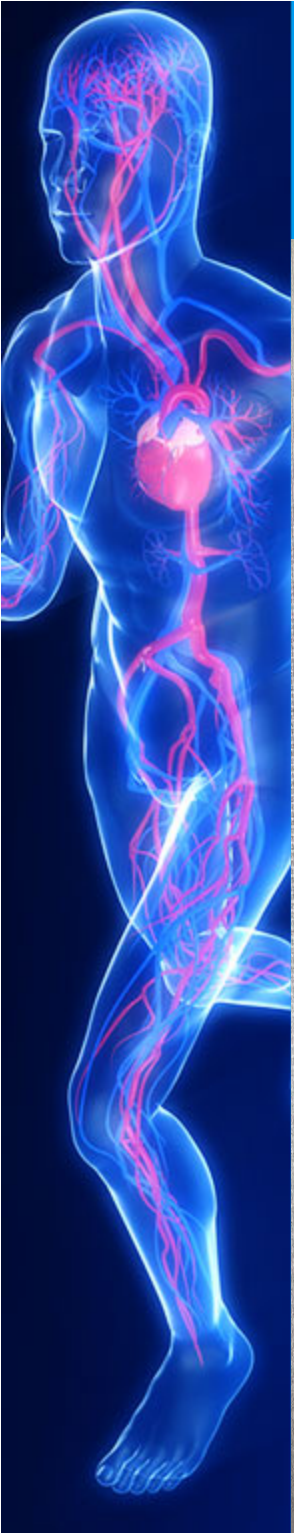




52 y/o F DM foot wound



CLI with gangrene

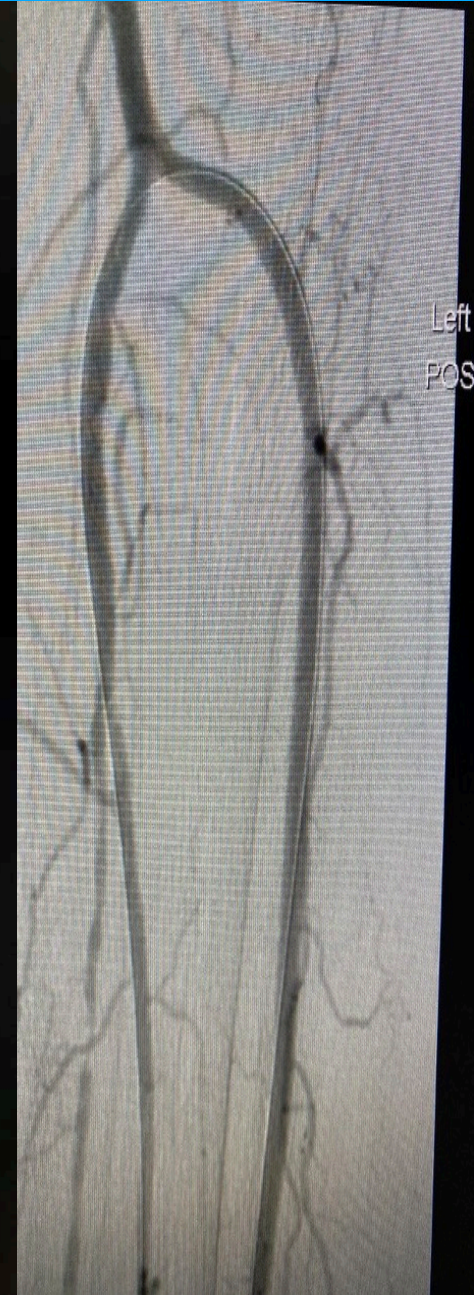


Left
PRE
Intervention



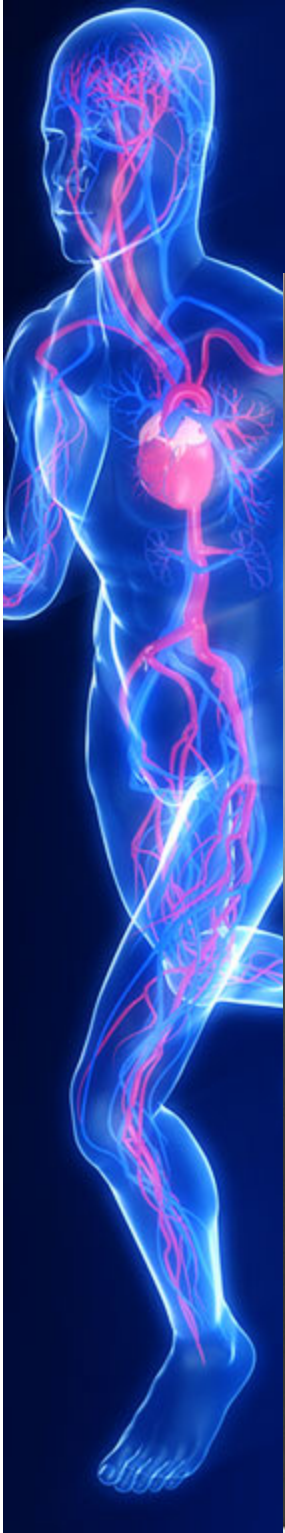
Atherectomy

Left



Left
POST

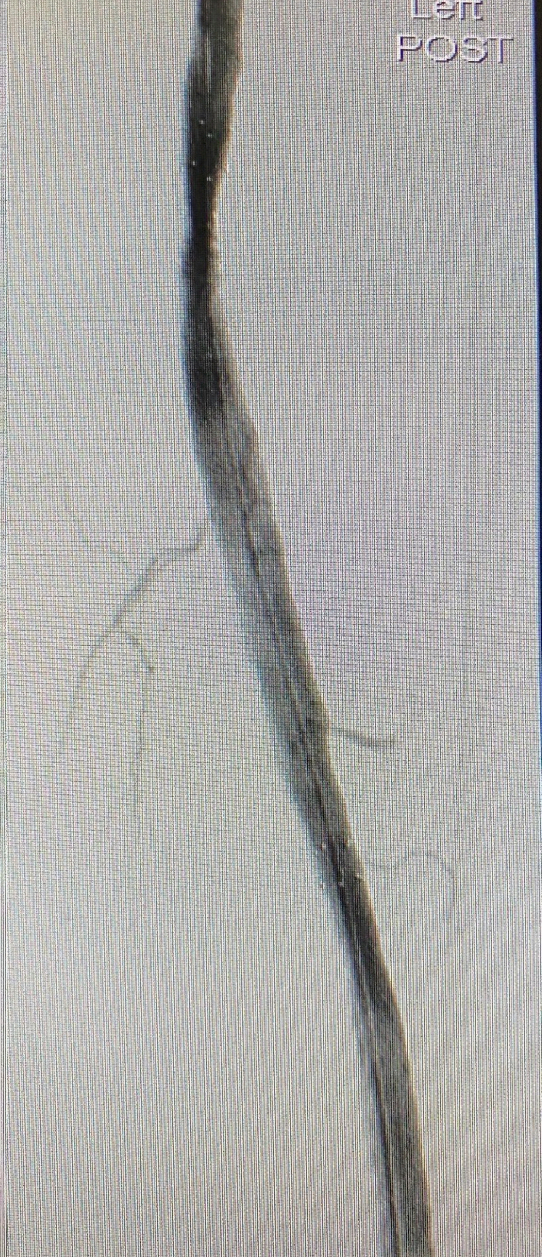
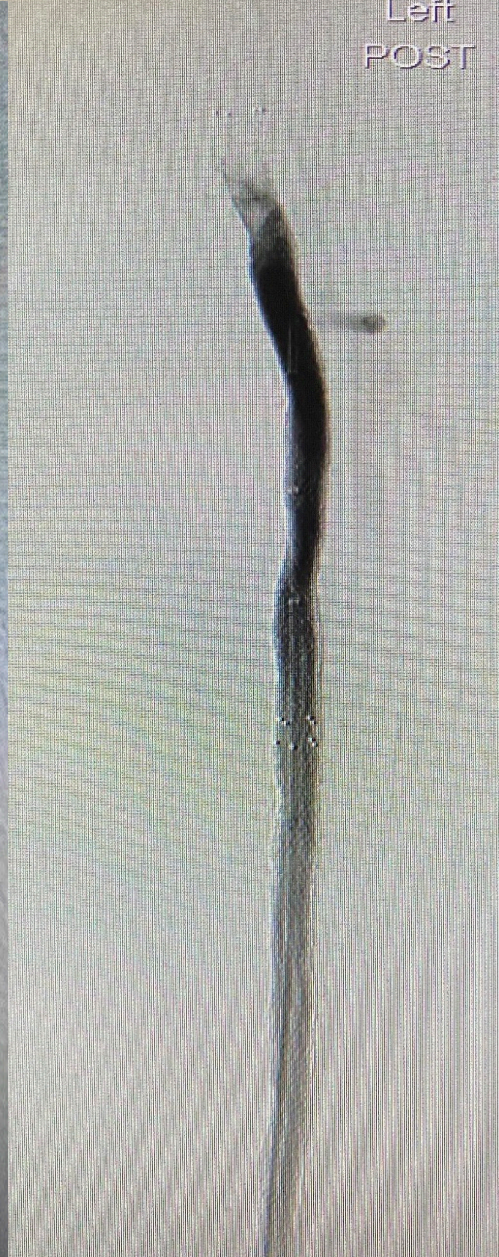
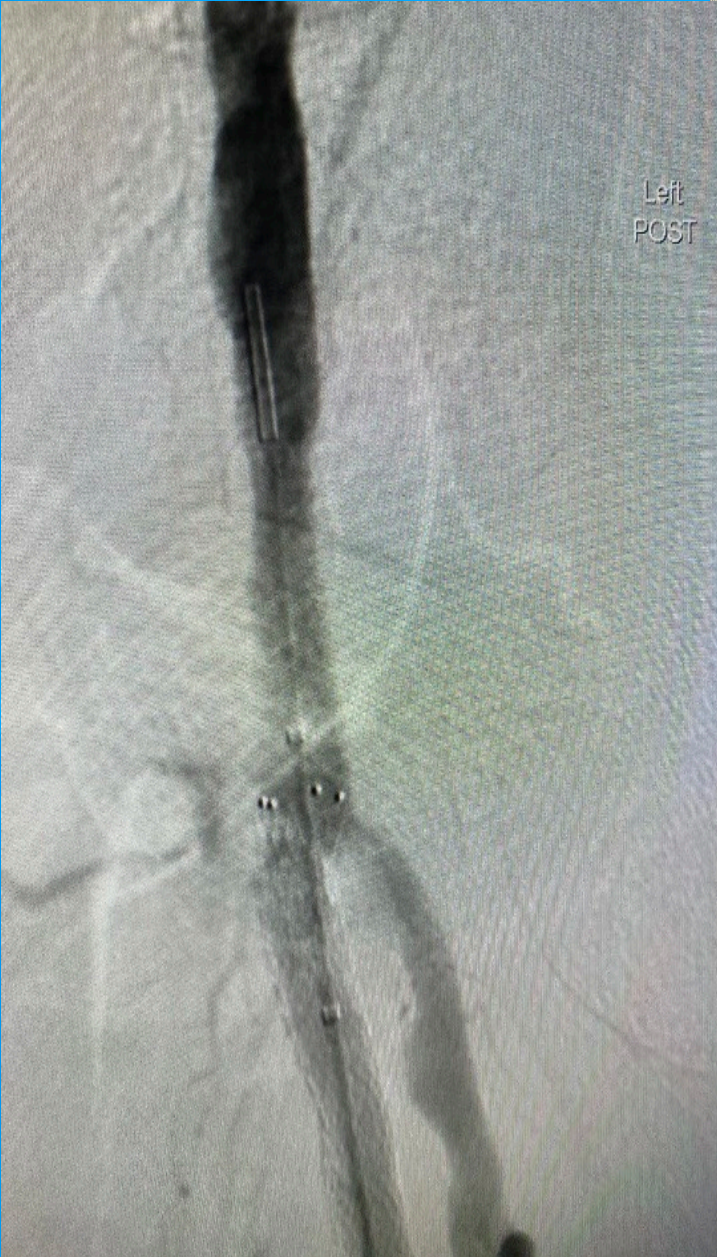
CLI with gangrene



CLI with gangrene, severe multi-level disease pre-intervention



CLI with gangrene, severe multi-level disease, post-intervention



Dr. Warrens Key Take Home PAD



Prevention, Early detection
and early treatment options
can save a leg!



If can't feel pulses in feet
need a vascular
assessment and work up



Most chronic foot wounds
have a vascular aspect that
can be optimized



PERIPHERAL VENOUS INSUFFICIENCY (PVI)

CHRONIC VENOUS DISORDERS (CVD)/ Chronic Venous
Insufficiency (CVI)



Chronic Venous Disorders (CVD)

Telangiectases (spider veins)

Reticular Veins

Varicose veins

Leg edema (dysfunctional venous tone with valve incompetence)

Abnormal calf pump function

Hyperpigmented skin changes

Dermal sclerosis

Ulcer formation

* chronic venous disorders (CVD) with manifestations specific to abnormal venous function are termed chronic venous insufficiency (CVI)

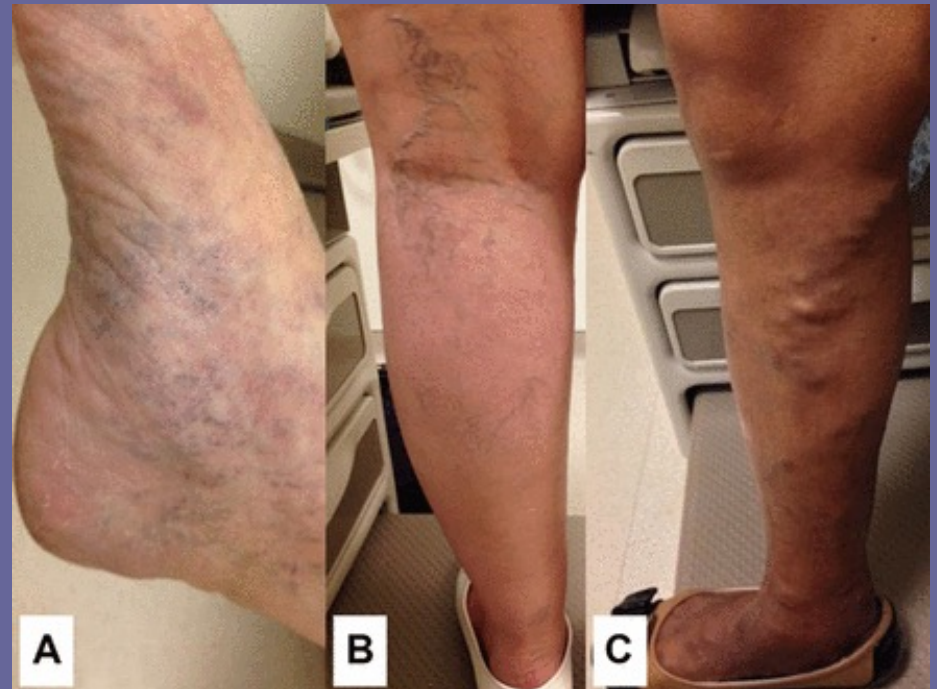
Chronic Venous Insufficiency

- Skin pigmentation
- Venous Eczema
- Lipodermatosclerosis
- Atrophie blanche
- Healed or active ulcers



CVD

- Very common
- Varicose veins affect over 25 million adults in the US
- First National Screening program in the US identified 32% of participants with varicose veins





Risk Factors for Varicose Veins

- Female predilection for varicose veins, males for CVI
- Older age
- Family history
- History of phlebitis
- Standing occupation
- *San Diego population study found Ethnic differences with Varicose veins being more prevalent in Hispanics (26%) and least prevalent in Asians (19%)
- *More advanced disease seen more commonly in non Hispanic whites and less commonly in African Americans

CVI Pathophysiology

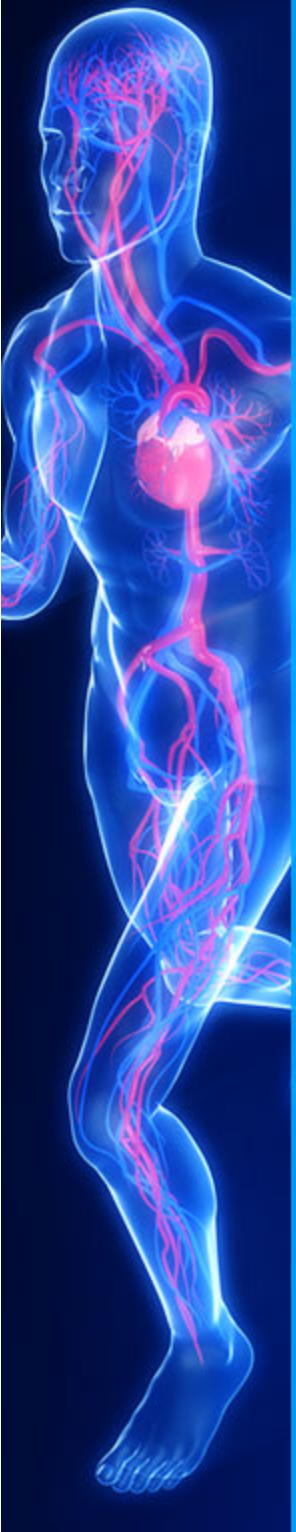
- Increased venous pressure +impaired return of blood =venous pathology

- ***Genetic predisposition

- Several mechanisms: valvular incompetence of deep or superficial veins, perforator valve incompetence, venous obstruction or a combo
- Muscle pump dysfunction
- Induction of venous HTN especially with standing

*Dysfunction of the deep system is most often a consequence of DVT

*Dysfunction of the superficial system from pre-existing weakness, direct injury, superficial phlebitis, excessive venous distention from hormonal effects or high pressure



History and Physical Exam- examine in both supine and erect

- Most common Sx and So telangeictasias, reticular and varicose veins
- Vvs become painful with progressive dilatation and tortuosity as a result of distention
- May bleed, itch or become inflamed

Clinical*

- C₀ - No clinical signs
- C₁ - Small varicose veins
- C₂ - Large varicose veins
- C₃ - Edema
- C₄ - Skin changes without ulceration
- C₅ - Skin changes with healed ulceration
- C₆ - Skin changes with active ulceration

Etiology*

- E_C - Congenital
- E_P - Primary
- E_S - Secondary
(usually due to prior DVT)

Anatomy*

- A_S - Superficial veins
- A_D - Deep veins
- A_P - Perforating veins

Pathophysiology*

- P_R - Reflux
- P_O - Obstruction

"Early application of compression should be performed to correct swelling and progressive scarring and to initiate the healing process by improving the venous microcirculation."

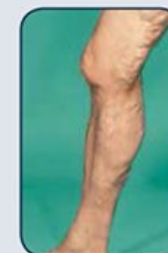
Kistner R. Specific Steps to Effective Management of Venous Ulceration. Supplement to Wounds June 2010.

*Fronek HS, Bergan JJ, et al. The Fundamentals of Phlebology: Venous Disease for Clinicians. 2004. pg 151.

Clinical Classifications with examples



C₁ - telangiectasias or reticular veins



C₂ - varicose veins



C₃ - edema & corona



C₄ - lipodermatosclerosis and eczema



C₅ - ulcer scar



C₆ - active ulcer

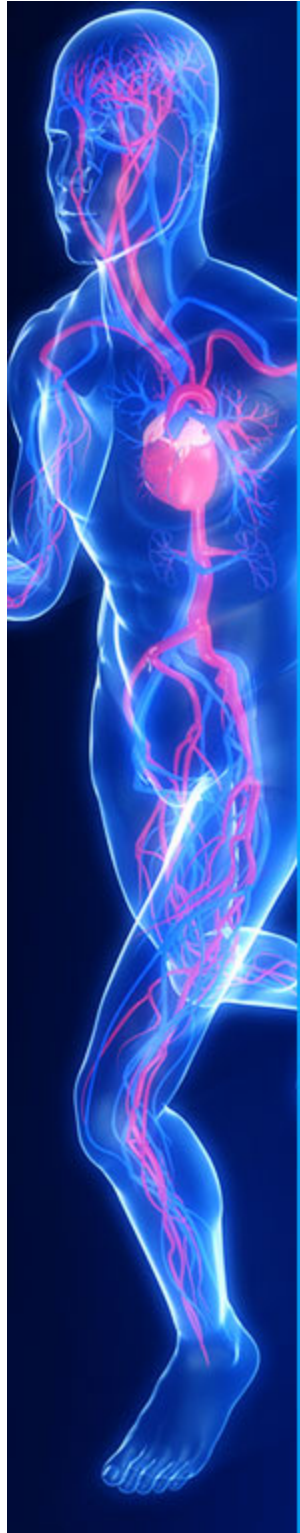


H&P CVD Common Symptoms

- Pain, Swelling, Ulceration
- Heaviness or aching aggravated by standing and relieved by elevation (note how this is different slightly from arterial)
- Chronic obstruction of the deep system may lead to venous claudication with intense cramping during ambulation (exertion induced venous HTN)
- Edema begins in the foot and ankle and progressed through the day
- Development of unilateral edema is suggestive of venous etiology

CVI Diagnostics—Primarily Made With Physical Exam

- Venous duplex -reversal of flow in the superficial venous system lasting longer than 0.5 sec indicated valvular incompetence (think of veins and valves like an elevator)
- CT with IV contrast useful if concern of iliac vein stenosis/May Thurner, Pelvic congestion or extensive DVT



CVI Treatment Plans

- Medical Mgmt
- Compression
- Wound and Skin care
- Exercise
- *Intervention including Sclerotherapy, Endovenous RFA and Laser Ablation, Endovascular therapy, phlebectomy

Table 53-5 Treatment of Venous Disorders Based on Pathophysiology

Venous Pathophysiology	Primary Treatment	Secondary Treatment*
Reflux		
Superficial	Compression	Ablation, HLS, sclerotherapy, foam, phlebectomy, pharmacologic
Deep Perforator	Compression Compression	Valve reconstruction Ablation, foam, ligation, SEPS
Obstruction (nonacute)		
Central	Compression, venous stenting	Venous stenting
Peripheral	Compression	Valve reconstruction
Muscle pump dysfunction	Compression	Structured exercise

*Ablation indicates endovenous radiofrequency and laser ablation. HLS indicates high ligation and stripping. Pharmacologic includes the micronized purified flavonoid fraction (Daflon), horse chestnut seed extract. SEPS indicates subfascial endoscopic perforator surgery. Central obstruction indicates vein segments involving the femoroiliacal segments, and peripheral vein segments involve the femoropopliteal segments.



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Thank YOU SO MUCH!

Questions?

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