

NOTES **VENOUS DYSFUNCTION**

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

 Venous system defects affecting blood flow from lower extremities

CAUSES

 Blood clot partially/completely blocking way/venous valves failing to pump blood against gravity

Virchow's triad

- Hypercoagulability, increased clot formation
 - Factor V Leiden thrombophilia
 - Protein C and protein C deficiencies
- Venous stasis from prolonged immobilization (e.g. bed rest)
- Damage to endothelial lining

RISK FACTORS

- Prolonged immobility, hereditary clotting dysfunctions, high estrogen levels, obesity
- One venous dysfunction can lead to another

MNEMONIC: PHD

Virchow's Triad

Prolonged immobilization (stasis)

Hypercoagulability

Damage to endothelium

SIGNS & SYMPTOMS

- Localized pain, usually lower extremities
- Edema
- Pruritus

- Localized hyperpigmentation/skin discoloration
- Hard, cord-like veins/prominent dilated tortuous veins

DIAGNOSIS

DIAGNOSTIC IMAGING

Doppler ultrasound

 Assess vein diameter, thrombi, valve status, blood flow (anterograde vs. retrograde)

Venography

- X-ray, contrast medium injected into vein
- Assess status of vein network, detect thrombi

LAB RESULTS

■ D-Dimer: High sensitivity (~100%) and negative predictive value (~100%) for detection of venous thromboembolism

TREATMENT

MEDICATIONS

- Acute manifestation: unfractionated heparin/low-molecular-weight heparins
- Long-term management: oral anticoagulants (e.g. warfarin)
- Prior DVT
 - Long term anticoagulation therapy, antiplatelet treatment, parenteral anticoagulants

SURGERY

Vein transplant/repair/removal

OTHER INTERVENTIONS

 Preventative: calf exercises, compression stockings/devices, raise affected areas to decrease swelling

CHRONIC VENOUS INSUFFICIENCY (CVI)

osms.it/chronic-venous-insufficiency

PATHOLOGY & CAUSES

 Veins cannot push blood back to heart, resulting in blood pooling in leg

CAUSES

- Develops from varicosities, DVT, phlebitis
 - Varicose veins affect superficial veins. but blood sometimes rerouted to collateral veins deep in leg, preventing blood stagnation
- When deep veins carry more blood than normal
 - Deep veins stretch over time, blood pools
 - Blood flow stagnation in lower extremities causes inflammatory reaction in vessels, tissue, causing fibrosis, venous stasis ulcers

RISK FACTORS

 Biological females, inactive standing/sitting for long periods, aging, family history, ligamentous laxity, obesity, smoking, lowextremity trauma, prior venous thrombosis, arteriovenous shunt, pregnancy

SIGNS & SYMPTOMS

- Calf/ankle pain (most common symptom)
- Worse with prolonged standing/sitting, improves with leg elevation, movement
- Brown hyperpigmentation of skin

(hemosiderin deposits)

- Pruritus, stasis dermatitis
- Painless, wet ulcers, particularly on medial malleolus
- Edema
- Atrophie blanche: hypopigmented atrophic areas with telangiectasia (clusters of red/ purple capillaries), red dots

DIAGNOSIS

DIAGNOSTIC IMAGING

Doppler ultrasound imaging

- Most common diagnostic
- Modified vein diameter (increased = acute thrombus, decreased = chronic thrombus)
- Absent color flow: vein completely occluded
- Increased flow in surrounding superficial veins

Venography

 Most effective, but invasive and costprohibitive

TREATMENT

SURGERY

Vein transplant/repair/removal

OTHER INTERVENTIONS

• Preventative: calf exercises, compression stockings/devices, raise affected areas to decrease swelling



Figure 26.1 The clinical appearance of mild CVI. Hemosiderin deposition is clearly visible.

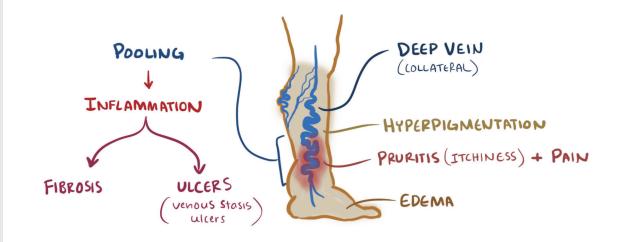


Figure 26.2 Illustration of varicose veins that have developed into a case of CVI.

DEEP VEIN THROMBOSIS (DVT)

osms.it/deep-vein-thrombosis

PATHOLOGY & CAUSES

- Blood clotting in deep leg veins (iliofemoral, popliteal, femoral veins)
- Arterial clots usually due to artery wall damage; venous clots don't require vein damage
- Valves inside veins can lower blood oxygen levels → venous stasis-associated hypoxemia can activate reactive oxygen species, other hypoxia-inducible factors → tissue factor released into blood
 - □ Tissue factor activation → prothrombin turns into thrombin → fibrin fibers form net → traps red blood cells, white blood cells, platelets → venous thrombus

CAUSES

- Virchow's triad
- Antiphospholipid syndrome
- Prolonged immobilization (bed rest. orthopedic casts, long-distance air travel)
- Genetic
 - Antithrombin, protein C, S deficiencies

RISK FACTORS

 Pregnancy, oral contraceptives, old age, major surgery (e.g.orthopedic surgery), malignancy, obesity, trauma, heart failure

COMPLICATIONS

- Pulmonary embolism (PE) most common
 - Can cause pulmonary infarction, death
- Post-thrombotic syndrome
 - Develops in 50% of individuals with
- Extreme cases: phlegmasia cerulea dolens (blue, painful, swollen leg, possible venous gangrene)

SIGNS & SYMPTOMS

- 50% asymptomatic due to venous collateral channels
- Localized inflammation around clot
- High venous pressure engorges visible superficial veins
- If PE occurs: sudden dyspnea, chest pain Fatal if enough lung tissue affected

DIAGNOSIS

DIAGNOSTIC IMAGING

Doppler ultrasound imaging

- Most common diagnostic
- Modified vein diameter
 - Increased: acute thrombus
 - Decreased: chronic thrombus
- Absent colour flow: vein completely occluded
- Increased flow in surrounding superficial veins

Venography

Most effective, but invasive/cost-prohibitive

LAB RESULTS

- D-dimers → rule out DVT
 - Increased level: plasmin dissolves thrombus

OTHER DIAGNOSTICS

Wells' score

- Higher score indicates increased chance of DVT (Scale of -2 to 9 points)
 - High score = high chance: > 2 points
 - Moderate score = moderate chance: 1-2 points
 - Low score = low chance: < 1 point</p>

WELLS' SCORE

CRITERIA	POINTS
ACTIVE MALIGNANCY	+2
SWELLING IN ONE CALF ≥ 3 CM	+1
SWELLING OF UNILATERAL SUPERFICIAL VEINS	+1
PITTING EDEMA IN ONE LEG	+1
HISTORY OF PREVIOUS DVT	+1
UNILATERAL SWELLING OF LEG	+1
LOCALIZED TENDERNESS	+1
CAST IMMOBILIZATION OF LEGS AND PARESIS / PARALYSIS	+1
RECENT IMMOBILITY > 3 DAYS OR SURGERY REQUIRING GENERAL ANESTHETIC IN PAST 3 MONTHS	+1
LIKELY ALTERNATIVE DIAGNOSIS	-2

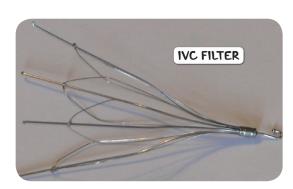


Figure 26.3 An IVC filter, used to prevent embolization of the deep vein thrombus into the pulmonary vasculature.

TREATMENT

MEDICATIONS

- Acute manifestation: unfractionated heparin/low-molecular-weight heparins
- Long-term management: oral anticoagulants (e.g. warfarin)
- Prior DVT: long term anticoagulation therapy, antiplatelet treatment, parenteral anticoagulants

OTHER INTERVENTIONS

• Preventative: calf exercises, compression stockings/devices, raise affected areas to decrease swelling



Figure 26.4 Clinical appearance of a deep vein thrombosis of the right leg. The lower leg is erythematous and swollen.

THROMBOPHLEBITIS

osms.it/thrombophlebitis

PATHOLOGY & CAUSES

- Vein inflammation caused by clot in deep leg veins
- Increased coagulability (Virchow's triad)
- Potential locations
 - Upper limbs (usually at site of IV cannula)
 - Lower limbs (coupled with varicose veins)
 - Periprostatic venous plexus in biological
 - Pelvic venous plexus in biological females
 - Large veins of cranium, dural sinuses
 - □ Portal vein

TYPES

Migrating thrombophlebitis

 Occurs in several different locations, usually in pancreatic carcinomas due to proclotting factors secreted by tumoral cells

Superficial thrombophlebitis

- Thrombus develops in vein near skin's surface
 - Mondor's syndrome: thrombophlebitis of subcutaneous veins of breast/arm / penis; presents as lump

Suppurative (septic) thrombophlebitis

 Infection from IV cannula; possible purulence

CAUSES

- Most commonly: needle/catheter
- Prolonged immobilization: bed rest, orthopedic casts, long-distance air travel
- High estrogen: pregnancy, estrogen replacement therapy, oral contraceptives
- Hereditary clotting disorders: protein D/C deficiencies/factor V Leiden mutations
- Vasculitis, Behcet's disease

COMPLICATIONS

• DVT, superficial thrombophlebitis, pulmonary embolism

SIGNS & SYMPTOMS

- Pain, inflammation/swelling, hard, cord-like
- Sometimes asymptomatic, can be revealed by applying pressure
 - Hoffman's sign (forced dorsiflexion on foot creates soreness behind knee); not 100% accurate

DIAGNOSIS

DIAGNOSITC IMAGING

Venous duplex ultrasound

- Thrombosed veins thickened, poorly compressible
- Completely occluded vein = hypoechoic (low level echoes)
- No internal flow present distal to clot

Imaging studies

- Thrombus detection (e.g. CT venography (CTV) with contrast, magnetic resonance (MR) venography)
- Blood coagulation tests (e.g. elevated D-dimers)

LAB RESULTS

Blood coagulation tests

Elevated D-dimers

OTHER DIAGNOSTICS

- Inspection of affected area
 - Pulse (weak/absent)
 - Blood pressure (high)
 - Temperature (high)

TREATMENT

MEDICATIONS

 Acute manifestation: unfractionated heparin/low-molecular-weight heparins

COMMON FEMORAL VEIN

Long-term management: oral anticoagulants

OTHER INTERVENTIONS

• Preventative: calf exercises, compression stockings/devices, raise affected areas to decrease swelling

DIAGNOSIS

VENOGRAPHY ULTRA SOUND BLOOD CLOT IN



Figure 26.5 Illustration showing blood clots discovered via imaging studies.

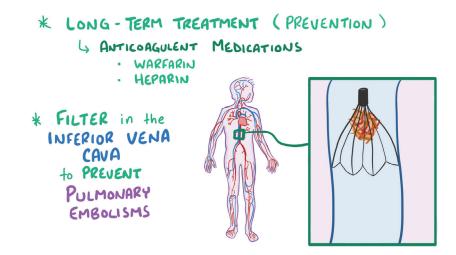


Figure 26.6 Illustration showing a surgically-implanted filter in the inferior vena cava preventing a pulmonary embolism.

VARICOSE VEINS

osms.it/varicose-veins

PATHOLOGY & CAUSES

- Enlarged, twisted superficial veins (most commonly in leg)
- Downward gravitational pull causes walls of veins to stretch over time, blood leaks backwards → extra blood volume twists veins → veins become varicose
- Blood sometimes rerouted to collateral veins deep in leg

TYPES

Varicocele

- Abnormal enlargement of pampiniform venous plexus in scrotum
- Mechanism same as varicose veins
- Most common in left testicle
 - Left testicular vein brings blood to left renal vein at 90° angle → difficult → blood backs up → vein becomes varicose → loops back and forth on itself
 - "Bag of worms" appearance

CAUSES

- Obesity, pregnancy, standing for long periods of time, menopause
 - Pelvic vein reflux (PVR): ovarian vein reflux, internal iliac vein reflux
- Hyperhomocysteinemia destroying structural proteins in vessels
- Chronic alcohol use

COMPLICATIONS

- Chronic venous insufficiency
- Venous ulcers
 - Can develop into carcinomas, sarcomas over time (rare)
- Superficial thrombophlebitis

SIGNS & SYMPTOMS

- Twisted superficial veins
- Edema, pain (usually in evening)
- Pruritus in affected area/stasis dermatitis because of undrained waste in leg
- Prolonged bleeding, slowed healing in injuries to adjacent areas
- Restless legs syndrome

DIAGNOSIS

DIAGNOSTIC IMAGING

Doppler ultrasound

- Used to discover subcutaneous varicosities. assess saphenofemoral junction
- If blood reflux spotted during Valsalva manoeuvre → valve incompetence
- Reflux > 1s → surgical intervention

OTHER DIAGNOSTICS

Trendelenburg test

- Person laid back on flat surface, leg raised above heart, blood will flow towards heart → compress upper thigh with tourniquet (not too tightly) → lower leg onto flat surface → person stands, refilling times assessed
 - Normal: superficial saphenous vein fills < 30 - 35s
 - Faster filling → valvular incompetence below compressed area → deep/ communicating veins
 - \circ Slower filling \rightarrow tourniquet released \rightarrow if filling sudden → incompetent superficial veins

TREATMENT

SURGERY

- Radiofrequency/laser ablation
- Sclerotherapy
- Ambulatory phlebectomy: removal of surface vein through slits in skin

OTHER INTERVENTIONS

Preventative: compression stockings/ devices, avoid prolonged standing



Figure 26.7 An X-ray image demonstrating varicose veins of the left leg.

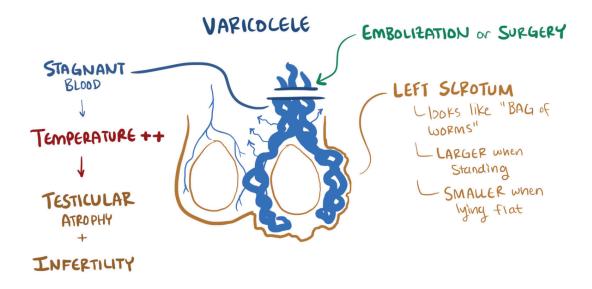


Figure 26.8 Illustration of a varicocele in the left testicle.