



Al- Azhar University - Gaza

Palestine Faculty of Medicine

Vascular Surgery

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N.B/ Many thanks to **Dr. Anas Dalloul** for his contribution in the hard-copy material which was very helpful.

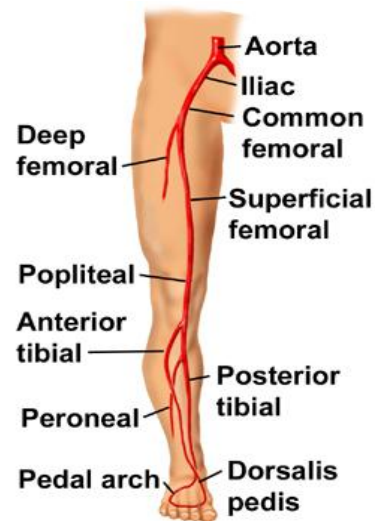
April – 2018

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Peripheral Arterial Diseases

Arterial Occlusion of the limb:

- **Etiology:**
 - Atherosclerosis
 - Emboli
 - Trauma
- **Risk factors:**
 - Smoking
 - Age (90% of cases >70 years)
 - Male sex (estrogen is protective in female)
 - DM & HTN
 - Hereditary hypercholesterolemia
 - High fat diet
 - Obesity
 - Lacking exercise
 - Stress



Note/ PVD: peripheral vascular disease/ disease of the vessels other than the coronary & great vessels of the heart.

- **Atheroma:**
 - Definition: subintimal deposition of cholesterol & fat.
 - It's a systemic disease affect all the vessels
 - Blood vessel layers : from inside to outside: 1) intima 2) media 3) adventitia
 - **Affected areas by atheroma:**
 - Femoro-popliteal area (75%) (The most common affected area by atheroma)
 - Distal (10%)
 - Aorto- iliac region (15%)

Clinical picture of PVD:

- **1) Intermittent claudication/** cramp pain occurring in group of muscle supplied by certain artery that is increase by exercise & relived by rest.
 - Characters of the pain in intermittent claudication.
 - Not present on the 1st steps unlike the osteoarthritis
 - Relived by standing still unlike the nerve compression by disease of the spinal cord
 - Pain occurs at exercise due to accumulation and relived by rest due to wash out of metabolites by venous return.
 - Common sites for felt pain
 - Calf muscles : mostly due to superficial femoral occlusion "femoro-popliteal blockade"
 - Foot due to distal arteries occlusion

Peripheral Arterial Diseases

- Thigh mostly due to deep femoral artery occlusion " profunda femoris blockade" or aorto-iliac occlusion
 - Buttocks due to aorto-iliac artery occlusion
- Claudication distance/ the distance that patient is able to walk before stopping by pain.
- **Leriche's syndrome**
 - Buttocks pain, thigh claudication & erectile dysfunction (impotence) secondary to atherosclerosis occlusion of the bifurcation of aorta into 2 common iliac arteries
 - Diagnosis by: transfemoral/transaortic angiography
- **2)Rest pain (more severe)**
 - Definition/ pain occurring in the periphery "toes, heels"
 - Character of rest pain
 - Continuous, severe, aching in nature
 - Aggravated by exercise, lying flat & elevation of the foot- worse at night & on pressure.
 - Relieved by hanging the foot out the bed or sleeping by hanging the foot or sleeping on the chair.
 - DDx: arthritis, neurological, myositis
- **3)Color changes:**
 - Pale in normal position
 - Bluish (deep purple) on dependency "sunset sign"
 - Limb blanches on elevation
 - Berger angle: normally at 90 degree not blanching, the narrower the angle ;the worse the ischemia (severe if less than 20 degree)
- **4)coldness, numbness, parasthesia**
 - Ischemia – cold & pale
 - Inflamed "cellulitis" – hot & warm
- **5)Pre-gangrene (critical limb ischemia)**
 - ROCH mnemonics(R: rest pain, O:oedema, C:color changes, H:hypo/hyperesthesia)
 - Ankle- Brachial index(ABI) = Systolic Pressure in Ankle/ Systolic pressure in Brachial
 - Normally is 1 (0.9 -1.1)
 - Ischemia is < 0.9 = arterial obstruction results claudication
 - Mild ischemia < (0.7 – 0.6)
 - Critical ischemia < 0.4 and < 0.3 can result necrosis
 - Note/ > 1 indicates calcified non-compressible arteries such as in DM condition.
- **6)ulceration &gangrene**
 - Severe ischemia
 - Between the toes, dorsum of feet, around the malleoli (ulcer, painful)
- **7)sensation &movement**
 - Actually ischemic limb paralysis insensitive
 - Chronic ischemic limb is not paralyzed & sensation is intact

Peripheral Arterial Diseases

- Most sensitive organ to ischemia – nerves
- **8)Pulsation and bruits**
 - Pulsation distal to arterial occlusion is absent or diminished
 - Dominant artery is the posterior tibial artery ; felt 2cm below & behind the medial malleolus
 - Dorsalis pedis artery: continuation of anterior tibial artery
 - Absent in 10-20% of people
 - Felt against navicular & middle cuneiform bone just lateral to extensor hallucis longus.
- **9)Temperature**
 - Usually colder than the other leg
- **10)Trophic changes:** Indicate chronicity
 - Cold feet
 - Loss of hair(skin appendage –hair)
 - Poor nails growth (skin appendage –nail)
 - Fragile shiny skin
 - Loss of sweat response (skin appendage – sweat gland)
 - Atrophy of muscles (muscle layer)
 - Poor healing (blood vessels)
- **11)slow venous refilling**
- **12) slow capillary refill**
 - Normal is 2-3 seconds
- **Some helpful points for Diagnosis:**

Occluded artery name	Signs & symptoms
Distal obstruction	- Femoral & popliteal arteries are palpable - Ankle pulses is absent - Foot/calf claudication
Femoral artery	- Femoral pulse is palpable with absent unilateral distal pulses - Unilateral claudication in calf
Iliac artery	- Unilateral absence of femoral & distal pulses - Unilateral claudication in thigh/ calf & buttock - Bruit in iliac region
Aorto- iliac obstruction (leriche's syndrome)	- Bilateral claudication in buttocks , thigh & both calf - Bilateral absence of femoral & distal pulses - Bruit in aorto-iliac region - Erectile dysfunction (impotence)

Peripheral Arterial Diseases

• Upper limb ischemia

- Etiology:

- Trauma (most common cause)
- Vasospastic condition (e.g Raynaud)
- Atherosclerosis
- Thromboarteries obliterans

- Clinical picture:

- Color changes in the fingers (most common)
- Raynaud's phenomena
- In acute occlusion: sudden feeling of numbness, painful sensation, white or blue fingers
?pulses
- Microemboli: transient, less severe

- Investigation:

General:

- 1) DM :glucose
- 2)lipid abnormalities
- 3)anemia: CBC
- 4)ESR , Auto Abs (Increase viscosity)
- 5)ECG/ exercise ECG
- 6)PFA

Specific:

- 1)Doppler
- 2)Duplex (U/S +Doppler)
- 3)Ankle pressure index
- 4)Treadmill "measure claudication distance"
- 5)Arteriography
- 6)Digital subtraction angiography (DSA)
- 7) MRI, CT angiography

- Notes/ Duplex: measure vessel diameter- vessel in atherosclerosis appears narrowed, irregular lining not straight . DSA: need IV dye (risk of RF/Allergy), very clear picture and accurate.

- Management of peripheral arterial diseases in general:

- 1) conservative:

- Explanation & advise
- Adjustment of life style
- Stop smoking
- Diet (weight reduction)
- Lipid abnormalities

Peripheral Arterial Diseases

- DM, HTN
- Care of feet
- Elevation of head of the bed
- Heal rise
- Notes/ collateral need at least 3 months to form after exercise, Care of the feet/ cutting nails.
- **2) Drugs:**
 - Trental (oxypentifylline) – decrease viscosity and increase RBs flexibility
 - Prexiline
 - Prostacyclin, nifedipine
 - Aspirin, clopidogrel
 - Analgesic
 - Notes/ avoid B- blocker as it increases stenosis & claudication
- **3) Sympathectomy:**
 - Surgical or chemical
 - Effect on the skin & subcutaneous blood vessels
 - Not effective on claudication
 - Relieves rest pain by producing vasodilatation
 - Q. why not effective on claudication? Medium sized blood vessels; not under the control of sympathetic system
- **4) Angioplasty (Percutaneous Transluminal Angioplasty – PTA)**
 - Short segment- occlusion
 - Balloon catheter – stent
 - Seldinger technique
 - Easily repeated
- **5) Surgery**
- **What are the complications of angioplasty?**
 - Failure of the procedure
 - Bleeding /bruising- the puncture site
 - Rupture of the artery
 - Blockage of the artery
 - Intimal dissection
 - Re-stenosis
 - A-V fistula
- **What are the indications for the surgery?**
 - Rest Pain (absolute)
 - Acute occlusion to emboli
 - Ulcer not responds to treatment
 - Claudication (relative)
- **Types of Grafts/operations.**

Peripheral Arterial Diseases

- 1) Bypass graft using
Vein :the best, increase the survival rate
Prosthetic: Dacron/ PTFE, gore tex
- 2)Endarterectomy
- 3)Angioplasty
- 4)salvage procedure "prolene suture is used"
(Bantalloon graft: for aorta + iliac arteries)

● Acute Arterial Occlusion

- **Etiology:**
 - 1)thrombosis 80-85%
 - 2)Emboli 15%
 - 3)Trauma
- **Embolic occlusion:**
Embolic(foreign object circulating in the blood from site of origin to distal segment
Any material : circulating in the blood dislodged in the next bifurcation & results occlusion and ischemia.
- **Site/** LL (75%) 6Ps, Brain (stroke), Retina (blindness), spleen, kidney & mesenteric vessels (ischemia), coronary.
- **Types of emboli:**
 - 1) simple (clot): most common 90%, History of AF
 - 2)Air embolism (central line)
 - 3)Fat embolism
 - 4)infective & parasitic embolism (Entamoeba, Echinococcosis -hydatid disease)
 - 5)Amniotic fluid embolism (very rare, death)
 - 6)therapeutic embolism (to stop bleeding)
- **Clinical picture: 5Ps**
 - 1)pain
 - 2)pallor
 - 3)paresis
 - 4)parasthesia
 - 5)pulseless
 - 6)poikolothermia (coldness)
- Ischemia after 6 hours – irreversible – limb loss
- Mottling of the skin: ominous late skin
- **Management:**
 - 1)clinical Diagnosis
 - 2)immediate operation
 - 3)analgesia

Peripheral Arterial Diseases

- 4)early administration of heparin
 - 5)Embolectomy (fogarty catheter)
 - 6)Thromolysis / fibrinolysis
 - 7)Post- Operation anti-coagulation
-
- **Occlusion due to trauma:**
 - 1) In lumen – thrombosis
 - 2)In wall – Dissection
 - 3)Outside the wall – compartment Syndrome

Arterial Disease

- **Arteritis:** large, Medium or small.

- Burger disease
- Temporal (giant cell) arteritis
- Takayasu arteritis (pulseless disease)
- Polyarteritis nodosa (PAN)
- RA/Scleroderma

- **Burger disease/Thromboarteritis obliterans**

- **Epidemiology**

- 1908 discovered by Burger
- Common orient :South-east Asia, India& Middle east
- Young age (20-40) years
- M>F by 10 :1
- Heavy smokers

- **Pathology**

- Small to medium size arteritis (ulnar, radial, tibial arteries)
- Autoimmune inflammation & thrombosis in arteries & veins

- **Clinical picture**

- 1- Ischemia:
 - Claudication in the feet &/or hands associated with pain in these area at rest
 - Pain typically begins in the extremities, but may radiate to other areas(burning/tingling)
 - Skin rash& gangrene at digits (start distally – tip of fingers, then proximally)
- 2-Raynaud's
- 3- Thrombo-phlebitis migrans

- **Diagnosis**

- 1, 2 or 3 together occur
- Arteriography : corkscrew appearance, espically in wrist & ankle
- Duplex imaging: occlusion or stenosis in many areas of both arms & legs

- **Treatment**

- 1- stop smoking immediately and completely
- 2- Patients who continue smoking may require amputation of fingers and/or toes.
- 3-NSAID, anticoagulents & aspirin (very little effect)
- 4-avoid cold (vasoconstriction) and exercise (increase O2 demand).
- 5-sympathectomy may help control pain

- **Vasospastic Disease**

- Raynaud's Disease
- Acrocyanosis

- **Raynaud's Disease**

- Incidence and etiology

Arterial Disease

- 1- young female
- 2- primary raynoud's disease :abnormal sensitivity to cold – idiopathic
- 3- Or secondary to other causes "phenomena"
 - 1- collagen vascular disease :SLE,Scelrodema
 - 2- Drugs: B-blocker
 - 3-Trauma
 - 4-Tools: Vibrating tools
 - 5-arterial occlusion
 - 6-smoking

- Pathology

- Raynoud disease (primary):
 - Most common form of idiopathic disease
 - Being allergic to cold
- Ranynoud phenomena (secondary):
 - 30-40 years
 - Signs and symptoms
 - More aggressive – gangrene /amputation
 - More complicated
 - More serious than the primary
 - Underlying problem

- Clinical picture

- Cold fingers and toes(more in hands> foot)
- Color changes in skin in response to cold/stress : white then blue after that red
 - White: due to spasm
 - Blue: cyanosis & depletion of oxygen supply
 - Red: hyperemia
- Numbness/ pesky feeling
- Pain upon warming/or stress

- Treatment: prevention by:

- 1) Keeping warm by wearing clothes. Gloves & socks
- 2) Avoid cold or frozen objects
- 3) Avoid stress/smoking
- 4) Avoid decongestant/ diet pills "phenylpropunolamine?"
- 5) Drugs: Ca channel blocker
- 6) sympathectomy

● Acrocyanosis:

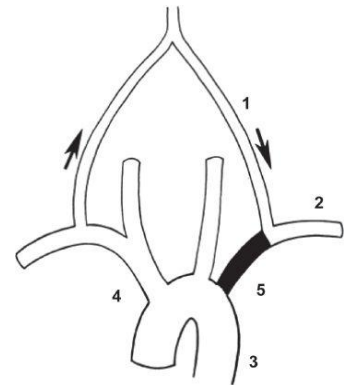
- **Definition/** persistent blue or cyanotic discoloration of the extremities; mostly hands, can be in face or feet or both, due to cutaneous arteriolar spasm due to neuro-hormonal disorders
- **Epidemiology/**
 - Young age, females and children
- **Clinical picture/**

Arterial Disease

- 1) persistent cyanosis: hands, feet, face
- 2) cold and calm extremities
- 3) pulse normal
- 4) sweating of palms and soles
- 5) cold worsen the cyanosis
- 6) normal oxygen saturation
- **Diagnosis:** only by clinical picture
- **Treatment:** no standard medical or surgical treatment
 - 1) avoid cold
 - 2) reassurance benign condition
 - 3) Ca channel blockers have little value
 - 4) Sympathectomy, rarely needed

• Subclavian steal syndrome

- **Pathology**
 - Blockade of 1st part of subclavian artery, involving the vertebral artery
 - Vertebral artery then provide blood supply to the arm through collateral (steal) – reversal of the blood flow – so decrease BF to brain (decline cerebral circulation).
- **Clinical picture**
 - Syncope, visual disturbance, decrease pulse & blood pressure
- **Diagnosis**
 - Doppler
 - CT angio
 - Angiogram
- **Treatment**
 - Angioplasty (stent) or bypass



• Thoracic outlet syndrome / scalene syndrome

- **Etiology**
 - 1) neck trauma (most common)
 - 2) cervical rib
 - 3) abnormal 1st rib
 - 4) abnormal clavicle or scalene muscle
- **Pathogenesis**
 - Angulation of subclavian artery and brachial plexus - stenosis – post stenotic dilatation – stasis then thrombosis and embolization
- **Clinical picture**
 - 1) neck lump
 - 2) claudication and pain
 - 3) cold hand
 - 4) weak pulse and pressure
 - 5) different blood pressure between two arms

Arterial Disease

- 6) Raynaud's phenomenon
- **Investigation:** Chest X-ray, nerve conduction study, arch aortogram
- **Treatment**
- 1) shoulder physiotherapy 3-6 months
- 2) division of scalene anterior muscle and the middle one
- 3) resection of the 1st rib or cervical rib (if there is a cause treat it in the first hand!)

• Mesenteric Artery stenosis :

- It's a part of atherosclerosis
- Commonly at the origin of superior mesenteric artery
- Acute occlusion, thrombosis or embolism
- **Clinical picture**
 - 1) post prandial? central abdominal pain/ upper left side
 - 2) patient afraid to eat
 - 3) acute occlusion (sudden onset and acute abdomen with marked distension associated with metabolic acidosis and leukocytosis)
- **Treatment:** by bypass surgery

• Renal artery stenosis

- **Etiology:**
 - 1) atherosclerosis (most common cause)
 - 2) fibromuscular dysplasia in female < 50 years
 - 3) risk factors
- **Clinical picture:**
 - 1) most are asymptomatic
 - 2) hypertension at young age, high BP that suddenly gets worse and difficult to control
 - 3) rapid deterioration of renal functions
- **Pathogenesis:**
 - Decrease blood flow, kidney responds as hypotension, retention of salt and water the HTN is the result.
- **Investigation:**
 - 1) RFT
 - 2) Duplex
 - 3) renal arteriogram
 - 4) MRI
 - 5) CT angiography
- **Treatment:**
 - Treatment is medical, unless renal failure is likely to happen.
 - Bilateral direct intervention is indicated
 - PTA & stent is the method of choice
 - Surgery

• Carotid artery stenosis

Arterial Disease

- **Etiology:** atheroma, systemic disease
- **Clinical picture:**
 - 1) recurrent transient ischemic accident often followed by severe permanent strokes
(TIA: short lived ministroke with complete resolution within 24 hours –usually within minutes.)
 - 2) Amaurosis Fugax: fleeting blindness caused by minute embolus in central retinal artery
 - 3) less common symptoms, bruits
- **Investigation:**
 - o 1) U/S Doppler (gold standard)
 - o 2) CT or MRI angiogram
- **Treatment**
 - o if the stenosis less than 70% then the treatment is medically:
 - life style modification
 - control risk factor
 - aspirin and warfarin
 - o if the stenosis more than 70% the treatment is surgically:
 - stent or carotid endarterectomy (removal intima and media- 2/3 thickness of artery.)

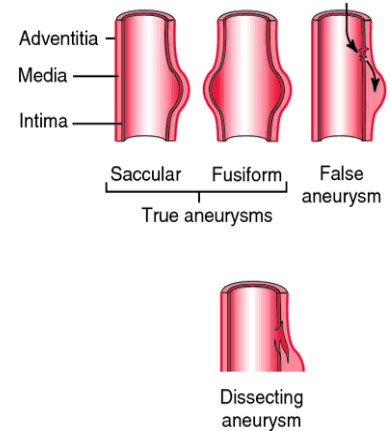
• Blue toe syndrome

- **Definition**
 - o Is a situation of atheroembolic microemboli causing transient focal ischemia, occasionally with minor apparent tissue loss, but without diffuse fore foot ischemia.
- **Incidence**
 - o 1) M > F
 - o 2) Age > 40 years
- **Aetio-pathogenesis**
 - o 1) blockade of distal arteries by microembolies often cholesterol, from proximal sources (aortoiliac)
 - o 2) may follow invasive vascular procedure
 - o 3) may follow abdominal surgery
 - o 4) Diabetic foot
 - o 5) connective tissue disease
- **Clinical picture**
 - o 1) painful cyanotic discoloration of toes
 - o 2) palpable pulses
- **Why is the Diagnosis often missed?** As it Transient
- **Treatment :**
 - o 1) proloTEX FIP socks
 - o 2) Anticoagulant
 - o 3) analgesic
 - o 4) bypass? – angioplasty
 - o 5) amputation if gangrene develop

Aneurysm

- **Definition:** localized dilatation in arterial wall involving 3 layers"
- **Types & classifications:**

Shape:	1) Fusiform: AAA 2) Saccular: ascending thoracic aorta 3) Dissecting: intimal tear "double lumen"
Pathology :	1) True: involving 3 layers of arterial wall 2) False: Not involving 3 layers & consider a hematoma connecting arterial wall
Cause/etiology:	1) Atheromatous 2) Mycotic "bacterial rather than fungal" 3) collagen vascular disease (CVD) 4) Trauma



Note/ In CT: dissecting aortic aneurysm appear as septum "Double lumen"

- **Clinical picture:** symptoms due to:
 - Expansion (back pain/pressure on uterus)
 - Thrombosis (shower of emboli)- DVT due to arterial embolism.
 - Rupture
 - Release of emboli: detachment from the hematoma

• Abdominal aortic aneurysm (AAA)

Dilatation one & half of its size (upto 3 cm is normal)

Incidence:

- The most common aneurysm
- 2% of subjects at biopsy(autopsy)
- M:F is 4:1 due to smoking
- 9th most common cause of death in male >55 years
- 1% of male 55-64 years have AAA, incidence increase with age
- 1st degree relatives have 15-19% chance to have AAA

Etiology:

- Atherosclerosis: most common cause
- Media degeneration
- Inflammatory (poly arteritis nodosa "PAN")
- Infection "syphilis or fungal"
- Genetics: Ehler D. syndrome, Marfan, Polycystic kidney disease
- Cigarette smoking in >90% of patients

Note / cigarette smoking: increase the risk of expansion (rupture), Syphilitic aneurysm: most of them is saccular aneurysm in ascending thoracic aorta.

Pathogenesis:

- Aortic medial generation
- Matrix metalloproteinase & their inhibitors
- Aneurysm tend to expand over time
-

Aneurysm

Laplace law: tension(of wall) = pressure *radius

wall tension is directly related to BP & the radius of the artery.

- Gradual/ sporadic expansion of aneurysm & accumulation of mural thrombi
- When wall tension > tensile strengths rupture occurs.

Definition of AAA:

- Aneurysm more than 3 cm of abdominal aorta
- Most of cases >95% below renal arteries L1 L2
- Average small aneurysm <5.5 cm, grows at a rate 2.3- 3.2 mm/year
- Large aneurysm grows at a faster rate as did tobacco smoking >5mm/year
- No identified risk factor to assess which aneurysm will enlarge except smoking
- If >5.5 cm then surgery is indicated, if <5.5 cm only observation
- Types of AAA: 1) infra-renal mostly 2) supra-renal 3) juxta-renal

Clinical picture (AAA)

- 70% asymptomatic
- Abdominal discomfort / pulsatile mass
- Back /groin/ loin pain if inflamed (compress nerve vertebra)
- Release of emboli distally – trash foot?
- Sudden severe pain – rupture/expansion
- Compression: ureteric obstruction, hydronephrosis, renal failure
- Fistula: a duodenal or caval? Yet fistula develop more commonly after surgery repair
- Rupture.

On physical examination

- Epigastric pulsatile mass
- Peripheral pulses: normally not affected
- Note/ Aorta ends below umbilical 1 -2 cm & to the left

Diagnosis:

- 1) by Hx & P/E – central abdominal pulsatile mass, tender
- 2)U/S
- 3) CT or CT angio
- 4)MRI

Ruptured AAA

- 1) Anterior into peritoneal cavity (20%) – free bleeding – inevitable death (die within hour due to hemodynamic circulation collapse)
- 2) Posterior into retroperitoneal cavity (80%) (tamponade effect? – need time, called leaking rupture: back pain / hypotension / tender/ pulsatile mass)
- 3) less than 50% of patient with (R.AAA) survive to reach the hospital

Aneurysm

- 4) presents in 50s male with tender, pulsatile mass, back pain & hypotension= rupture AAA unless proven otherwise
- 5) DDx: Pancreatitis, so do amylase before exploration
- Note/ in AAA amylase increase in about 3 times in contrast to pancreatitis where amylase can reach thousands.

Screening of AAA

- **WHY ?** - to identify pt with high risk of rupture
 - elective repair decline mortality to <5%
 - 9th cause of death (high mortality rate)
- **WHOM?** – those with family Hx as it increase the risk to 10 folds
 - male > 55 years & smokers
 - patients with peripheral artery disease
- **HOW?** - clinical examination
 - U/S 100% sensitivity, simple, cheap cost, safe, no restrictions
 - CT
 - MR

Management of AAA:

Medical treatment/ 1) stop smoking 2) exercise 3) statin 4) diet 5) control (BP-B.Blocker – ACEi).

- **Indication of surgery:**
 - 1) all symptomatic patients
 - 2) fit patients
 - 3) asymptomatic if with – rapid growth > 0.5 cm/year
 - >5.5 on U/S(increase incidence of rupture)
- Notes / 1) without surgery: 80% of those symptomatic die in 1 year
- 2) with surgery: 80% will live!
- 3) incidence of rupture is 1% or less in aneurysms <5.5 cm but if the diameter >5.5 the incidence will raise to 75%

Management of rupture AAA:

- 1) Do not keep the pt in the ER for vital signs stabilization in case of rupture
- 2) immediate resuscitation(O₂, IV fluids & central line insertion)
- 3) Monitor BP not more than >100mm Hg
- 4) urinary catheter
- 5) Cross match 6 units of blood
- 6) rapid transfer into operation theatre
- 7) ask for an IUC bed

Note/ for every liter of transfused blood give either: 1) 1 liter of hemocrite 2) 3-4 liter ringer lactate (Hartman solution)

- Surgical operation:
 - 1) Open surgery – bypass (high mortality)
 - 2) Endoluminal procedures: for – elderly whose not fit for an open surgery,

Aneurysm

minimal invasive procedure, less complication, less hospital stay, through femoral artery by: Dacron or PTEE? "metal integrated"

Contraindication of the surgery:

- Severe COPD
- Severe cardiac disease
- Acute infection
- Advanced cancer- metastasized

Complication of the surgery:

- 1) respiratory: consolidation 5%, ARDS
- 2) Hemorrhage
- 3) MI, CHF or arrhythmia
- 4) wound dehiscence 10-20%
- 5) Death 1.8-5% in elective, within the surgery if ruptured happen 50%
- 6) bowel ischemia: as IMA(L3) is ligated (1% elective, 15-20% if rupture)
- 7) distal emboli: ischemic limb/ trash foot?
- 8) fistula – common aorto-duodenal fistula which results severe hematemesis
- 9) renal failure
- 10) infection of the graft
- 11) neurological: sexual dysfunction
- 12) anterior spinal artery syndrome
- 13) paraparesis/ quadriparesis
- 14) impaired pain & temp sensation (proprioception & vibration- preserved)

• **Popliteal Aneurysm**

- Most common peripheral aneurysm
- 70% of the peripheral all aneurysm
- 70% Bilateral
- 70% complication within 5 years
- 30% associated with AAA

Clinical picture

- 1) swelling pulsatile behind knee
- 2) symptoms following complications:
 - Following thrombosis- limb ischemia, DVT
 - Following emboli – Distal ischemic ulcer, gangrene toe
- Treatment: surgery if > 25 mm (2.5cm)

• **Femoral Aneurysm**

- Uncommon, affect elderly male
- Most cases are due to iatrogenic in 2% of people who perform surgery at this region
- (like: Cardiac catheterization can result such false femoral aneurysm 'hematoma')
- +/- AAA may be associated with complicated arterial surgery

Aneurysm

- Usually false aneurysm
- Treatment: usually conservative, resolve usually spontaneously, in elective repair complication <3%
- If infected increase incidence of rupture

• Aorto- Venous Fistula

- Definition "abnormal communication between artery & vein"
 - Causes: -1) congenital, 2) iatrogenic – renal dialysis, 3) Trauma
- Pathology/**
- Veins enlarged and dilated , tortuous, thick (arterialization of the veins)
 - Raise CO2 result LVH increase cardiac output which can result heart failure.

Clinical picture/

- pulsatile swelling if superficial
- continuous machinery murmur on AV fistula
- ischemia distal to it
- chronic venous insufficiency
- increase skin temp. (dilated, tortuous, warm vein)
- thrill & bruits

Treatment /

- Embolization (closing the vein)]
- Excision

• The normal size of some arteries.

Aorta	<3cm
Infra-diaphragmatic aorta	17 -20mm
Common iliac artery	10 -12 mm
External iliac artery	8-10 mm
Common femoral artery	7-9mm
Superficial femoral artery	6-8mm
Popliteal	4-6mm

Varicose Veins

- **Classification according to the site:**

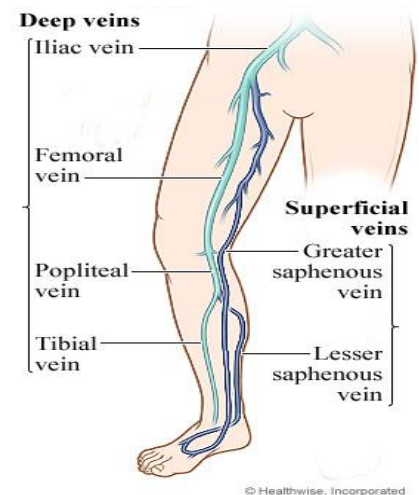
- lower limbs: varicose veins
- esophagus: varices
- spermatic: varicocele
- umbilicus: caput medusa
- anal region: hemorrhoids

- **Anatomy of the lower limb vein system:**

- 2 system : superficial and deep system are connecting together by perforating small veins.
- **Great saphenous vein:** arise from the dorsum of the foot then goes anterior to medial malleolus after that ascend in the medial aspect of the leg where ascends above knee anteriorly to thigh to connect the femoral vein by **sapheno-femoral junction (SFJ)** – at fixed point, 1.5 inch below & lateral to pubic tubercle.
- **Small saphenous vein:** arise from the lateral aspect of the dorsum of the foot then ascends along the posterior-lateral aspect of the leg where drains into popliteal vein below the knee (in variable regions)

Note: valves allow only unidirectional flow (from superficial to deep system)

- Leg & thigh perforators:
 Hunterian: in the proximal thigh
 Bodd: inferior 1/3 of the thigh
 Boyd: at the level of the knee
 Cockett: inferior 2/3 the leg consists of 3 divisions according to position from the medial malleolus
 I: 5 cm, II: 10cm, III: 15cm



- **Varicose vein (lower limb)**

- Definition: Dilated, tortuous, palpable, subcutaneous veins, larger than 3 mm in diameter.
- Reticular veins: Dilated, non palpable, subdermal veins, 1-3 mm in size
- Telangiectasia: Dilated, intradermal venules, less than 1mm in size (spider naevi)

Etiology:

- 5% of the adults
- F:M 2:1
- Erect position
- Wall weakness & valve destruction
- Hormonal factor- pregnancy
- Hereditary FOXC2 gene

Pathology:

- Primary: congenital weakness/destruction of the valves
- Secondary: to other causes: portal hypertension, pregnancy
- Which system? (trunk varices)
- Normal blood flow (compromised)
- Reticular veins or telangiectasia

Varicose Veins

- Thrombosed , calcified or rupture

CEAP Classification:

- CEAP system: used to describe finding in patients with different stages of venous insufficiency:
C: clinical picture, E: Etiology, A: Anatomic, P: Path-physiology

Clinical picture:

- VV rarely cause severe symptoms.
- Cosmetic
- Heaviness & fullness in the leg (most common)
- Ankle swelling at the end of the day
- Itching(stasis-RBCs extravasation – hemosiderin – irritant)
- Night cramps (while sleeping)
- Bleeding superficial phlebitis

Signs

- Cough impulse on SFJ
- Visible vein in the legs – localized blow out
- ?Ankle edema
- Pigmentation, indurations, eczema (lipodermatosclerosis)
- ? Venous ulceration – specially above the ankle in gaiter area(the highest venous pressure?)

Saphena varix

- Large groin varicosity, which presents as painless lump, emerges when standing but disappear while lying flat.
- Positive cough impulse: feeling thrill during palpation
- DDx: inguinal hernia, femoral hernia, lipoma or lymphadenopathy

Complication of VV:

- Thrombophlebitis (inflammation of superficial vein)
- Hemorrhage
- Calcification & pigmentation
- Eczema
- Lipodermatosclerosis
- Venous ulcer
- Equine deformity
- Periostitis

(Slide) Picture of inverted champagne bottle: post thrombotic syndrome.

(Slide) Picture of chronic venous ulcer in medial malleolus. Why venous? According its site besides its size which shallow and wide in contrast to arterial which is deep & round ulcer

Investigation

Varicose Veins

- Standard Doppler examination.
Uniphasic signal: flow in one direction (normal)
Biphasic signal: forward & reverse flow which indicate valve incompetence
- Duplex U/S: mainly for short saphenous

What are the indication for duplex scan?

A- should be done in all patients

B-In case of limited resources:

- o 1- Short saphenous incomplete, confirm the disease & site of SPJ as it variable between population.
- o 2- Recurrent varicose
- o 3- Complicated veins (ulcer/perforated)
- o 4- History of DVT

Management

1- Conservative

- Exercise
- Elevation periodically during the day
- Elastic support (compression)
- Elevate the foot of the bed
- Avoid long standing
- Don't sleep on chairs
- Don't take shower in the morning- to prevent the vasodilatation that may occur

2-Injection sclerotherapy:

- Few varicose veins
- sodium tetradecyl sulphate STD is preferred
- Egan method (very fine needle)
- Elastic support after injection
- U/S guided foam sclerotherapy for both LSV, SSV using polydecene

Complications of sclerotherapy.

- Necrosis or gangrene if the agent goes out the vessels
- DVT
- Severe headache

3-Surgery:

For gross varicosities

- 1-) Trendelenburg surgery
 - Flush ligation (sapheno-femoral disconnection & ligation of the tributaries of LSV at their exact connection to LSV)
 - Ligation of the tributaries of the LSV at the saphenous opening include:
1- Superficial inferior epigastric vein, 2- superficial circumflex iliac vein, 3- superficial external pudendal vein, 4- anterolateral thigh vein.

Varicose Veins

- 2-)Stripping only above the knee(because below the knee the vein attach to nerve which can result numbness and parasthesia if the stripping happens below the knee)
- 3-)Ligation of the communicating veins

Complication of the surgery

- Recurrence
- Deep veins and arteries injury
- Skin bruising
- Nerve injury : LSV, Saphenous nerve- SSV, sural & common pudendal nerves

4- Laser treatment (endovascular laser ttt)

- The most common type of leg varicose treatment
- Vein occlusion by direct thermal ablation energy
- Takes about 30-60 minutes
- Based on the spontaneous laser technique
- Minimally invasive
- Minimal bruising , swelling
- No pain

5-RadioFrequency ablation (RFA)

- Less-invasive method
- Increase patient satisfaction
- Performed as outpatient basis
- Does not require using of operation theatre
- Does not require using general anesthesia
- As effective as other methods

• Spider veins (telangectasia)

- Definition: dilated, intradermal venules 0.1-1mm in diameter.
- Small dilated blood vessels near the surface of the skin or mucosa membranes
- Separate from the saphenous vein system

Site:

- They can develop anywhere, but commonly seen in face, nose, cheeks, chin,limp(upper thigh) below the knee or around the ankle – progressive.

Etiology:

- Also: called thread veins/ hyphen webs.
- Result from local skin injury or "sun damage"
- Obesity , increase in age, thinning of the skin mostly in females.

Clinical picture:

- Can cause self negative image
- Alteration in skin texture, discoloration or itching.
- Rarely, bulge above the level of the skin.
- Drain into deeper veins called reticular veins
- If irritated – they rupture & causes bleeding.

Treatment: topical laser.

• Reticular veins:

- Commonly called 'feeding veins'

Varicose Veins

- 1-3 mm wide blue veins ,that can be seen in a curve linear fashion under the skin surrounding the spider complexes.
- Often not protrude on the skin surface
- Have incompetent valve
- On inner part of the thigh
- Treatment/ telangectesia &reticular veins can be treated with combination of sclerotherapy and topical laser.

- **Coroo phlebectesia**

- Fan-shaped, pattern of small intradermal veins on the medial and lateral aspect of the ankle &the foot.
- It's also called "levoid retivcularis" / "mallearb? Or ankle flare"
- Sign of chronic venous insufficiency or indicate complixon?
- Early sign of advanced venous disease
- Treatment/ topical laser.

Compartment Syndrome & Lymphedema

• Compartment syndrome:

- Definition

- Compression of organs or structures e.g: nerves, blood vessels, muscles inside a closed space (compartment) within the body. This leads to tissue death from lack of oxygen due to blood vessels being compressed by high hydrostatic pressure within the compartment (pressure – pressure on veins – pressure in arteries – no blood supply – ischemia!).

- Incidence

- M > F
- Male 7.5: 100,000
- Female 0.7 : 100,000
- 70% due to trauma (most common cause)
- 36% due to fracture tibia
- 9.8 due to fracture of distal radius
- 23% due to soft tissue injury without fracture
- 10% of cases of anticoagulants
- High energy trauma > low energy trauma

- Causes

- 1- Fracture and muscle injuries :most common cause 70%
 - Fracture in long bones; tibia and forearm bones
- 2- Vascular injury : 30% (2nd to revascularization)
 - Ischemia: causes damage to cellular basement membrane that results in edema
 - Within reestablishment of flow, fluids leak into the compartment results high pressure

N.B especially arterial injury so prophylactic fasciotomy

- 3- Crush injury
- 4- Burns
- 5- High injury trauma
- 6- Penetrating injuries (unlikely)
- 7- Iatrogenic causes
 - 1)military Anti-shock trouser which decrease the compartment size
 - 2)tight splints, casts, dressing (most important one) cut venous supply.
 - 3)Malfunctioning sequential compression device
 - 4)IM, IA, IO infusion?
 - 5)massive hypertonic IV fluid infusion
 - 6)pressurized IV infusion of parental hypertonic contrast agent
 - 7)attempts on cannulating? Veins and arteries of the arm in patients on systemic anticoagulants.

N.B cast is contraindicated in fracture of forearm and leg – apply backslap ? for 2 days – then after swelling subsides then put cast.

- Where does it occur?

Anywhere, if there is an enclosed space, typically restricted by fascia

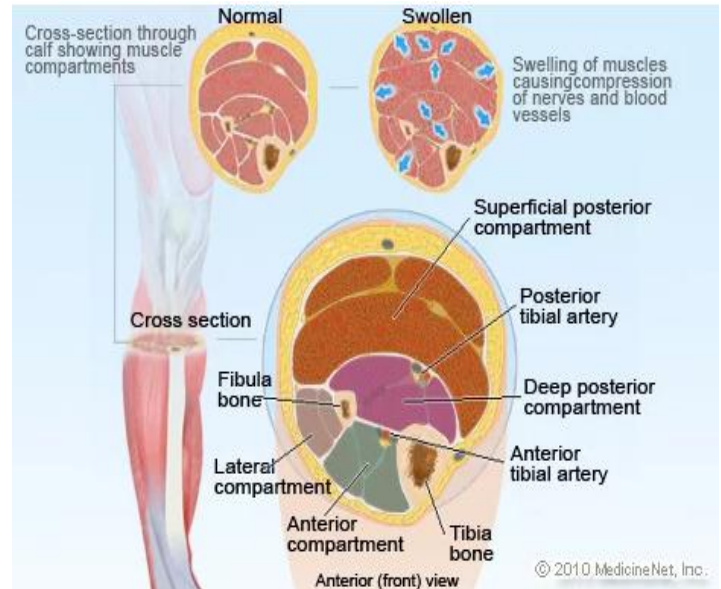
- 1)upper arm

Compartment Syndrome & Lymphedema

- forearm: especially volar compartment (deep flexor area)
- Hand
- 2) Lower limb
 - Leg: especially Deep post. and ant. Compartments
 - Thigh
 - Foot
- 3) Abdomen
- **Compartments of the legs**
 - Posterior compartment :
 - Flexor of the feet and toes
 - Tibial nerve
 - 2/3 of the blood supply to the lower limb by posterior tibial artery
 - Results:
 - 1) weakness of flexors
 - 2) pain extension
 - 3) weakness in inversion of ankle
 - Anterior compartment:
 - Extensors of the feet "tibialis anterior, extensors hallucis, digitalis longus & peronus"
 - Deep peroneal nerve
 - 1/3 of the blood supply to lower limb by dorsalis pedis and anterior tibial artery.
 - Results:
 - 1) foot drop
 - 2) loss of sensation of skin between 1st and 2nd toes
 - 3) weakness of toe extension, pain in flexion
- N.B/ Anterior distal compartment of the leg is the most common site of the compartment syndrome.
 - Lateral compartment
 - Peroneus longus & brevis + superficial peroneal nerve "eversion" so in lateral compartment syndrome – difficult eversion
- **Pathophysiology**
 - Tissue ischemia – tissue reperfusion – tissue injury
 - Ant. compartment of leg is more susceptible to ischemia due to stronger fascia

The underlying mechanisms:

 - 1) increase hydrostatic pressure within compartment
 - 2) increase volume within space
 - 3) decrease space for contents
 - 4) combination of both
 - 5) irreversible damage of tissue
- **Why it so serious?**



Compartment Syndrome & Lymphedema

- Normal pressure within the compartment is 4 – 6 mmHg but upto 15mmHg during exercise
- If pressure > 30 mmHg – prevents perfusion of tissue – ischemia and ultimately death of tissue
- **Tissue threshold for ischemia**
 - 1) muscle 4 hrs
 - 2) nerve 8 hrs
 - 3) fat 12 hrs
 - 4) skin 24 hrs
 - 5) bone 72 – 96 hrs

N.B for a viable limb, the upper threshold is about 6 hours

 - **Muscle ischemia:**
 - 4 hrs: reversible damage
 - 4 – 8 hrs: variable
 - 8 hrs: irreversible damage
 - Myoglobinuria after 4 hrs: leads to Renal failure so we have to maintain good hydration and alkalization of the urine
 - Cell death initiate a vicious cycle: increase capillary permeability and muscle swelling
 - **Nerve ischemia**
 - 1 hrs: normal conduction
 - 1 – 4 hrs neuropraxia damage (temporally loss of nerve function), reversible
 - 8 hrs: irreversible damage
- **Clinical picture:**
 - 1) History of
 - Fracture long bone
 - High energy trauma
 - Vascular injury
 - Penetrating injury – rarely
 - Soilders, ? athletes
 - 2) deep aching pain out of proportion to injury
 - 3) Incredible pain on passive movement due to stretch ischemic muscles
 - 4) tight tense swollen limb
 - 5) 5Ps may or may not present – late sign (pulseless, pallor, parasthesia, paresis, persistent cold "poikilothermia" & paralysis)
 - 6) congestion of the digits
 - 7) prolonged capillary refill, the redness, mottling,
- **Diagnosis**
 - Diagnosis is CLINICALLY
 - Passive stretches result severe pain
 - Support clinical suspicious:
 - 1) manometer to calculate pressure > 30 mmHg

Compartment Syndrome & Lymphedema

- 2) Doppler to measure blood flow
 - 3) plain X-ray – if there is a fracture
- **Lab:**
 - CPK (1000 – 5000 u/ml)
 - Renal function studies: urine analysis and urine myoglobin
- **What are the consequences if not recognized and treated**
 - 1) Myoneural necrosis: due to decrease tissue pH from (anaerobic necrosis and lactic acidosis plus release of potassium)
 - 2) Rhabdomyolysis which leads to ATN and ARF
 - 3) Volkmann contracture
 - Supracondylar fracture – brachial artery – necrosis of flexor muscle of forearm – permanent flexion deformity
 - 4) metabolic acidosis and hyperkalemia plus sepsis
- **Treatment**
 - 1) surgical emergency
 - 2) all bandages and casts should be removed, releasing one side of the cast can decrease compartment pressure by 30%
 - 3) fasciotomy: to allow the pressure to return to normal
 - 4) following fasciotomy; fracture reduction or stabilization and vascular repair if needed
 - 5) 48hrs after procedure, patient should be brought back to OR for further debridement
 - 6) delayed skin closure or skin graft 3 – 7 days after the fasciotomy
- **Complications** usually < 25%
 - 1) chronic swelling
 - 2) chronic pain
 - 3) muscle weakness
 - 4) iatrogenic nerve injury
 - 5) cosmetic concerns
 - N.B management depends on 1) time of injury- intervention 2) diagnosis, complete recovery of intervention done within 6 hours.

• Lymphedema

- **Definition:** abnormal limb swelling caused by the accumulation of increased amount of high protein interstitial fluid secondary to defective lymphatic drainage in the presence of near normal net capillary filtration
- **Incidence:** 0.1-2.0 %
- **Risk factors:**
 - 1) surgeries: axillary LN, post varicose vein surgery or others
 - 2) radiotherapy to breast and axilla
 - 3) trauma in at risk arm e.g: veinopuncture
 - 4) obesity
 - 5) chronic skin disorders/ inflammation
 - 6) immobilization and prolonged limb dependency

N.B bone healing in lower limb takes 8- 12 weeks

Compartment Syndrome & Lymphedema

- **Etiology:**
 - 1)infection – cellulitis, erysipelas, lymphadenitis, TB, filariasis and pedunculosis.
 - 2)malignant obstruction of LV(Breast, prostate, gut)
 - 3)iatrogenic lymphedema(LN dissection, Radiotherapy)
 - 4) chronic disease (rheumatoid, psoriasis, sarcoidosis, dermatitis)
 - 5)chronic venous insufficiency
- **Pathology**
 - 1)affect subcutaneous tissue
 - 2)decrease lymph return – gradual swelling of the limb start distally usually LL
 - 3)Transudation, pitting edema (**early**)
 - 4) accumulation, coagulation – non-pitting edema (**later**)
- **Classification**
 - Secondary/ there is underlying cause: filariasis: larva of w. bancrofti, malignancy
 - Primary / problem in the lymph vessel itself, either hypo/hyperplasia
 - 1) L. edema congenita (0-2) years
 - 2) Milroy disease, meigs disease
 - 3)L. edema praecox <35 years most common 90%
 - 4)L. edema tarda >35 years 10%
 - Familial/ hereditary lymphedema
 - 1)milroy disease: usually 2 limbs – onset at birth or before puberty
 - 2)Meigs disease: usually one or both limbs – onset between puberty and middle age.
- N.B/ post-thrombotic syndrome: spare foot but, Lymphedema: involve foot
- **Severity of unilateral limb lymphedema**
 - Mild: <20 % of excess limb volume
 - Moderate: 20-40 % of excess limb volume
 - Sever: >40% of excess limb volume
- **Clinical classification of lymphedema (Brunner Grade)**
 - 1) subclinical: no clinical evidence of edema
 - 2) I : edema pits on pressure, disappear on elevation
 - 3) II: edema not pits on pressure, not disappears on elevation
 - 4) III: irreversible skin changes (fibrosis, papillae)
- Skin changes: eczema, papillae, pigmentation
- **Clinical picture**
 - 1)constant, dull aching, even severe pain
 - 2)generalized tiredness and debility
 - 3)hyperesthesia (pins/needles)
 - 4)increase sensitivity to heat
 - 5)immobility – obesity and muscle wasting
 - 6)backache and joint problem
 - 7)athlete foot (fungal infection)
 - 8)enlargement of the draining lymph nodes
 - 9)cramps (non-pitting edema)

Compartment Syndrome & Lymphedema

- 10) skin damages: plaques, cellulitis, chronic eczema, fissuring, onychomycosis, dermatophytosis?
- **Investigation**
 - 1) lymphangiography, lymphangioscintigraphy: agenesis, hypo/hyperplasia, increase collateral, filling defects.
 - 2) CT, MRI, U/S, Biopsy
 - 3) Routine tests: CBC, Cr, LFT, KFT, total protein and albumin, FBG, CRP, UA, Blood smear, CXR
- **Complications**
 - 1) Recurrent cellulitis in upto 50% of cases due to S.pyogen/fungi
 - Affect patient life, 10% of which change careers
 - Long-term Abs may be needed
 - Indications of AB?
 - 2) Malignancy
 - Lymphangiosarcoma, BCC, malignancy melanoma, SCC, Fibrous histiocytoma, Kaposi sarcoma
 - 3) Limb heaviness, dysfunction, back and joint pain
- **Management of lymphedema** HINT/2016 short-note question.
- **Firstly** medical treatment – treatment of lymphedema is mostly conservative one
 - 1) manual lymphatic drainage MLD
 - 2) elastic stocking/ bandage class I, II
 - 3) elevation
 - 4) exercise, short term of diuretics
 - 5) faradic stimulation
- Standard treatment now is " Combined Decongestive Therapy (CDT)"
 - 1) MLD (50 min/ 2 times/ day to 2 weeks)
 - 2) Multilayer lymphatic bandages MLLB
 - 3) skin care/ exercise
 - 4) Patient education
 - 5) compression garments? (high P)
- **MLD**
 - 1) gentle manual ttt which improve the function of lymphatic system
 - 2) re –flow the lymphatics around blocked area to healthy channel
 - 3) by trained specialist
- **Compression therapy**
 - 1) applied between MLD
 - 2) improve efficacy of the muscle
 - 3) 1st stocking – then apply MLD
- Exercise preferred with compression bandages in place to increase muscle and joints mobility
- **Skin care**
 - 1) to keep skin in function?
 - 2) sometime long term AB?
 - 3) by skin hygiene

Compartment Syndrome & Lymphedema

- Patient education
- Pressure garments/ not part of CDT, but it is applied after MLD- By vibration device
- **Secondly** Surgical option:
 - Indication
 - 1) gross swelling
 - 2) recurrent infection
 - Aim: reduce weight of the affected part
 - Expensive
 - Results not satisfactory
 - Operations:
 - Bypass procedure
 - Liposuction
 - Limb reduction procedures
 - Charless op, Thompson op, swiss roll op
- In addition to the previous treatment options, these are also important issues to remember!
 - 1) Control DM
 - 2) Antibiotics (Culture& sensitivity)|
 - 3) Good surgical debridement and drainage if pus?

N.B: peripheral pulses(D.pedis/ P.tibial) are felt normal, Yet the problem is the digital arteries (microangiopathy)

Venous disorders

• Venous thrombosis

- **Virchow Triad**
 - Change in vessel wall (endothelial injury)
 - Diminished blood flow(stasis)
 - Increase blood coagulability (viscosity)
- Changes in vessel wall
 - Blunt trauma ,Bruises, not cut (prolonged operation)
 - Smoking – endothelial injury
 - External pressure eg: popliteal aneurysm
- Diminished blood flow
 - Immobility (E.thrombosis) Travelers
- Increase coagulability
 - Cancer. Dehydration, shock, post op.

Note/ Phlebothrombosis means Deep vein thrombosis, Thrombophlebitis means superficial vein thrombosis

- **Etiology** of venous thrombosis (thrombophlebitis)
 - IV canula
 - Infusion of hyperosmolar? Solution
 - Varicose veins
 - Burgers disease
 - Visceral cancer
 - Blood disease(SCD or Polycythemia)
- Venous cut down: Incision in the skin, over the veins , to reveal the veins to provide IV access
- Note : Canula in lower limb only allowed up to 48 hours
- **Clinical Picture:**
 - Pain , Tenderness, redness, cork like veins
- **Treatment:**
 - Elastic bandage for 2 weeks if not infected
 - NSAID
 - Antibiotics?
 - Excision if thrombus infected

• Deep Vein Thrombosis (DVT)

- **Incidence**
 - 5-10,000 in hospitalized patient
 - 50% if no prophylaxis procedures (ortho)
 - 30% bilateral
 - 50,000 death/year due to
- **Aetiology**
 - Virchow triad
 - Post op
 - Trauma , rare under 40 years

Venous disorders

- Varicose veins, obese, pregnancy, ...
- E.thrombosis and long flights
- Malignancy and OCD
- Post delivery
- **Clinical picture**
 - Local distension, discoloration, fever, pain, tenderness? Homman's sign? PE ?
 - May affect (calf muscle 90% below the knee), axillary veins.
 - Phlegmasia alba dollens 10% of cases
 - Phlegmasia cerulea dollens
- **Phlegmasia alba dollens (white milky leg)**
 - 10% of cases
 - 40% have underlying malignancy
 - Ilio-femoral veins and lymphatics
 - Thrombosis involve only major deep veins channels of the extremity: therefore the collateral veins
 - The veins drainage is decreased, but still present - interstitial edema – appear white
 - Lack of venous congestion (x-cerulea dollens)
 - May turn into phlegmasia cerulea dollens
 - No ischemia
- **Phlegmasia cerulea dollens**
 - More dangerous
 - Extreme case of DVT
 - Edema, agonizing pain, cyanosis
 - Massive fluid sequestration, may lead to bleb and bulla formation.
 - Thrombosis of deep, superficial and collateral veins (50% of capillaries)
 - Irreversible ischemia (vasospasm)
- **Diagnosis**
 - C/P in 50% of the cases
 - Test of choice duplex imaging
 - Gold standard venogram
 - Radioactive: fibrinogen reuptake test
 - Fibrin degradation products (FDP), may also increase in the followings: pregnancy, thrombosis, surgery, trauma, vacuities.
- **DDx of DVT (2015 short essay question)**
 - Rupture backer cyst
 - Calf muscle hematoma –how to differentiate with DVT is very important as the line of management is totally different.
 - Ruptured plantris muscle
 - Thrombosed popliteal aneurysm
 - Arterial ischemia

Yet, the duplex scan differentiate most of them
- **Prevention:**
 - (Pre&post op)
 - TEDs (thromboembolism deterrent hose)
 - Pills? Decrease weight?
 - Extended- pneumatic pressure

Venous disorders

- Early ambulation
- Low dose heparin or LMWH
- **Treatment**
 - Bed rest for resolving pain and edema
 - Exercise in bed, elevation foot of bed
 - Elastic bandage to enhance venous return
 - Full dose heparin /LMWH& Warfaren to achieve INR(2.5-3.5)
 - Fibrinolytic treatment
 - Greenfeild filter : precutaneously below renal veins in IVC (I2)in high critical & recurrent cases.
 - Plication of IVC
 - Thrombolysis
- **Treatment of Phlegmesia:**
 - Cercula- surgical management if untreated necrosis and gangrene are the result
 - Conservative medical treatment(limb elevation, IV heparin, fluid resuscitation)
If no response: surgical thromboectomy preferred through a femoral venotomy allow instant decompression.

• **Post thrombotic syndrome syndrome(PTS):**

- **Chronic venous insufficiency**
- Medical continuum where the veins cannot pump enough oxygen -poor blood back to the heart, due to muscle pump dysfunction? Incompetent valve?
- **Definition of PTS** "is the signs and symptoms that may occur as long term complication of DVT"
- **Incidence:** 20-25% of varicose vein patients.
- **Risk factors:** (2016 short essay question)
 - Proximal DVT
 - Age<65 years, obesity
 - Recurrent ipsilateral DVT
 - Persistent DVT symptoms, 1 month after DVT diagnosis
 - Poor control by anti-coagulants in the first 3 months
- **Pathology**
 - Same as varicose veins plus more fibrous tissue which will destroy lymphatics - fibrous rings & edema.
 - Leg ulcer in >75% of cases.
 - Signs & symptoms of PTS & DVT are quite similar, thus the diagnosis of PTS should be delayed for 3-6 months after DVT diagnosis
- **Clinical picture**
 - Leg swelling (foot sparing)
 - Calf firmness and enlargement (inverted champagne sign)
 - Dermatoliposclerosis: eczema, itching, indurations.
 - Brown pigmentation of the leg
 - Ulceration in 50% of cases, after 5-10 years
 - Significant disability
- **Treatment:**

Venous disorders

- Elastic support class III
- Skin care
- If the symptoms are severe, bypass surgery & valve reconstruction should be considered using the saphenous vein from the opposite limb as a cross over of free bypass.
- If the thrombosis recurs, patient may need life long anticoagulant.
- **Class of elastic support stocking:**
 - **Class I** (20-30) mmHg
Indication: slight varicose, edema, prevention of varicose during pregnancy
 - **Class II** (30-40) mmHg
Moderate varicosities, prevention of ulcer recurrence & management, moderate edema, lymphedema, prevention on PTS.
 - **Class III** (40-50) mmHg
Severe varicosities, severe edema, lymphatic edema, management of venous ulcer, post phlebitis syndrome, chronic venous insufficiency
- **Initial therapy for DVT**
 - Warfarin & heparin
Heparin/ bolus dose around 80U/Kg, and 18 U/kg as maintenance dose for 24 hour.
Warfarin is given as the following ,
 - In the First day – 10 mg
 - In the 2nd day – 10 mg
 - In the 3rd day – 5 mg
 - Then order INR, if INR>2, discontinue warfarin.

Gangrene

• Infective gangrene

- Etiology
 - o Mixed pattern of organisms: coniform?, staph, bacteroids spp, strept, peptosreptococcus
- Patients are almost always immunocompromised. DM
- Subdermal extensions of gangrene is always more extensive than it appears from initial examination
- Rapid spread and if untreated – MSOF
- For example:
 - 1) necrotizing fasciitis
 - 2) Fournier's gangrene (external genitalia)
 - 3) meleny's ? synergistic gangrene (post op)
 - 4) stomatitis gangrene (face)
 - In central part of Africa
 - Infective gangrene in oral cavity and around the mouth
- On x-ray: gas in tissue, not necessarily to be distal, it's according to site of infection.

• Traumatic Gangrene

- Never deals with tissue/skin flap with toothed forceps/ instruments- use hands
- In PVD patients, not normal patients

• Bed sores/ Pressure or Decubitus ulcer

- Etiology:
 - Occurs over bony prominence: anemia, malnutrition, injury, moisture? In immobilization patient
- Site:
 - Sacrum, heels (m.c.c?)
- Treatment:
 - Best way is prevention
 - Special care, plastic surgery
- Prevention way:
 - 1- change the position regularly
 - 2- Air- Water sth? (equalize the pressure)
 - 3- powder(over the pressure area)
 - 4- straight blanket under the pt (50% raise the risk)

• Frost bite

- 1- caused by exposure to cold
- 2- it is seen climbers at high altitude and in elderly

Gangrene

- 3- source cold- damage of blood vessels— sever burning pain- waxy appearance- blister formation – gangrene
- 4- treatment
 - Gradual re-warming
 - Delayed conservative amputation after demarcation of deritalized tissue
 - Analgesia

• Dry and Wet gangrene:

Differences between Dry and Wet Gangrene

Dry Gangrene	Wet Gangrene
▪ Clear line of demarcation is seen.	▪ Line of demarcation is vague.
▪ Dry, shriveled, mummified.	▪ Edematous, putrefied, discolored
▪ Slow, gradual loss of blood supply.	▪ Sudden loss of blood supply.
▪ Separation is by aseptic ulceration.	▪ Septic ulceration causes separation.
▪ Limits to the demarcation.	▪ Can extend proximally rapidly.
▪ Causes are- atherosclerosis, TAO (Thromboangitis obliterans).	▪ Emboli, trauma are the causes.
▪ Limited amputation is sufficient.	▪ Major higher amputation is often needed.

leg ulcers

• Leg Ulcer

- What are the cause of the leg ulcer?
 - 1) **Vascular**: venous, arterial, lymphatics and vasculitis.
 - 2) **Neuropathic**: DM, Peripheral neuropathy "usually foot"
 - 3) **Hematological**: polycythemia, sickle cell disease
 - 4) **Traumatic**: Burn and cold injures, radiation or pressure sores
 - 5) **Neoplastic**: Basal or squomus cell carcinoma , melanoma, marjoline ulcer or bowen's disease
 - 6) **Others**: sarcoidosis, tropical ulcers, pyoderma gangrenous
 - N.B/ ulcer: discontinuation of the epithelial tissue and covering.

- Venous Ulcer:

- Incidence:
 - 1- Most common type of ulcer 65-75% of leg ulcer
 - 2- affect 1-2/1000 capita/year
 - More in female than male, middle age
- Etiology (predisposing factors)
 - 1- mostly as a sequelae to DVT
 - 2- long term varicosities
 - 3- local trauma
 - 4- poor hygiene
- Pathogenesis
 - Valve destruction in communicating and deep veins – stasis – increase venous pressure – extravasation – fibrous reaction - thickening of small vessel wall – decrease in O2 & nutrients – tissue necrosis, minimal trauma – ulceration .
- Clinical picture
 - 1- in goiter area, any size
 - 2- shallow, sloping edge, unhealthy granulation in the floor, edema around
 - 3- pigmentation, eczema & induration around
 - 4- Painless and less infected as it shallow
 - 5- varicose veins are often visible as well
 - 6- rest of the foot and heels look normal
 - Note/ Goiter area is the most common site? Due to high volume pressure in this area
- CEAP classification of venous disease:
 - C: clinical picture
 - E: etiology
 - A: anatomy
 - P: pathophysiology
- Investigations:
 - 1- All patients should have duplex scan

leg ulcers

- 2- ABI to exclude arterial disease which can be presented in 30% of cases
- 3- the addition of disability score to the CEAP classification is important in clinical practice as many patients have work related problems
- 4- patients with advanced lipodermatosclerosis have tissue O₂ deficits despite their normal macrovascular flow, so Transcutaneous Po₂ should be measured
- 5- Assessment of microvascular disease
- **Management of the venous ulcer**
 - **A- Conservative management:**

(Aim to heal ulcer and prevent recurrence)

 - 1- Elevation of the foot of the bed to release of venous pressure
 - 2- Clean dry saline dressing (best)
 - 3- Hydrocolloid dressing combined with elastic compression in day time, most patients require class II , III
 - Hydrocolloid wound dressings
 - Made of adhesive gum like material such as pectin covered with water resistance films
 - Advantages :
 - 1- excellent for the ttt and management of the skin wounds/ ulcer/ pressure sores/ donor sites/ surgical incision/ and burns
 - 2- convenient & flexible – increase level of the control
 - 3- highly comfortable / non-irritable
 - 4- Doesn't stic? And water proof
 - 4- bisgaards regimens (4Es)
 - Patient education, elevation, elastic stockings, evaluation
 - 5- pentaxifylline?:
 - 1- Effective adjunct to compression bandages
 - 2- Works by decreasing platelet aggression and thrombus formation
 - 6- Antibiotics:
 - only if there are signs of infection "cellulitis": fever, pus, discharge, increase in WBCs
 - note/ don't ever make culture of venous ulcer because it's open injury.
 - **B – Surgical treatment**
 - ✓ Superficial venous surgery has been shown to improve ulcer healing in the patients with **only** superficial venous incompetence
 - ✓ excision of ulcer site and grafts
 - ✓ excision of incomplete veins
 - ✓ Elastic support post op
 - Berthes test : to check whether need a surgery or not
 - Assess patency of the deep femoral vein
 - Newer technique: TIRS – sclerotherapy under U/S guided
 - Terminal interruption of reflux resource
 - Reduce high venous pressure
 - Heals within 4- 8 weeks
 - Injection of sclerotherapy in roflux ?
 - **Complication of venous ulcer**

leg ulcers

- 2ry infection, recurrent cellulitis
- Stuffiness of ankle joint (due to dressing)
- Phlebitis
- Malignant changes "majolin ulcer (SCC)"
 - Biopsy should be considered as part of the work up if wounds
 - are greater than >6 months to 1 year in duration
 - have irregular appearances
 - or when concerns exist for underlying inflammatory conditions or carcinoma
 - NB/ long standing (>5 years) venous ulcer: marjolin SCC (more in edge)
- **Prognosis:**
 1. Treatment is costly
 2. Recurrence 30% of venous ulcers will recur by 5th year after healing
 3. Cellulites/ gangrene: amputation without proper care
 4. Venous eczema due to topical drugs used
- **Atrophie Blanchae:**
 - Whitish discoloration in healed ulcer
 - Localized white atrophic skin frequently surrounded by dilated capillaries and hyperpigmentation usually seen around ankles
 - Sign of severe venous disease

- Arterial Ulcer

- Incidence: 10% of the leg ulcer
- Etiology:
 1. PVD: atherosclerotic, AV malformation
 2. Vacuities: autoimmune destruction of blood vessel: SLE, RA, PAN, Scleroderma
 3. DM, HTN, RF
 4. Limited joint mobility
 5. Old age
 6. trauma
- site: usually on the foot
 - mostly the heels(pressure area)
 - tip of toes,
 - between toes where toes rub against each other or any where the bones may protrude & rub against bed sheets, socks & shoes
 - nail beds, if the toe nail cut into skin or if the patient had recent aggressive toe nail or ingrown toe nail trimming or ingrown toenail removed
- clinical picture:
 - very painful; specially at night and elevation
 - cold limb, No pedal pulses, No callus
 - Description: ulcer is usually
 - Deep: may reach muscles and tendons
 - Floor: necrotic tissue, grey, black – no blood
 - Edge: panned off?

leg ulcers

- Site: pressure area
- Treatment of arterial ulcer:
 - Goals of treatment are the followings:
 1. Prevent new ulcer
 2. Provide a good? protection to the surface of skin
 3. Remove contact irritation to the existing ulcer
 4. Monitoring the s&s of the infection
 - Arterial ulcer ttt: varies according to the severity of arterial disease
 - **Non- invasive** ttt: vascular tests to assess the potential for healing (TCo2) pressure- transcutaneous o2, If the TCo2 is low then debridement should performed.
 - **Invasive** ttt: Depending on the pt condition; endovascular therapy or bypass surgery to restore circulation to legs

	Venous Ulcer	Arterial Ulcer
1- Base	Ruddy? color	Pale on elevation
2- Skin	Brown staining	Shinny, taut
3- Depth	Shallow wound	Deep wound
4- Edge	Irregular wound ? in sloping	Pouch out appearance, well defined
5- Exudates	Moderate to heavy	Minimal
6- Temp	Warm skin	Cold
7- edema	Pitting/ non-pitting edema	Variable edema
8- granulation tissue	G.T present	Rare
9- infection	Critical, colonization may lead to infection (rare)	Frequently infected
10- Pain	Minimal to sever pain	Pain at rest and exercise
11- Capillary refill	Normal capillary delay	Capillary delayed
12- Pulses	Pedal pulses present	Absent/ diminished
13- site	Gaiter area	Tip of toes

N.B/ in arterial ulcer more than one vessels are affected – multiveessel disease

- Neuropathic (Diabetic) Ulcer

- Incidence
 - In 20 – 20 % over patient's life time
 - Occur primarily in diabetics, but can occur in anyone who has impaired sensation in foot
- Contributing factors
 - 1- sensory/ motor/ automatic neuropathy
 - 2- PVD with poor microcirculation
 - 3- poor control of glucose level
 - 4-reputitive trauma, unperceived pressure
- Description

leg ulcers

- Site/ usually located at the increased pressure points on the bottom of the feet – head of metatarsal bones is the commonest site .
- Callus ring (hyperkeratosis) around edge & healthy granulation tissue in the center – typically.
- Edge/ pinched out
- Skin around it almost normal
- Summary of characteristics
 - 1-Dry cracked skin
 - 2-Warmed skin
 - 3-Decrease sensation
 - 4-Regular margins
 - 5-Callus around the wound
 - 6-charcot joint: one or more joints with the foot can collapse & causes serious deformity & instability. (it is a chronic process and it can affect elbow joint)
- Treatment of neuropathic ulcer:
 - 1- Avoid pressure & weight bearing on the affected leg
 - 2- regular debridement (removal of infected tissue) is usually necessary before healing of neuropathic ulcer
 - 3- special shoes or orthotic devices must be worn
 - 4-control DM / sterile dressing

- Lymphatic Ulcer

- Ateiology : venous edema, injury in the presence of lymphedema
- Site: most common in ankle area, but may develop in any trauma site
- Description:
 - Shallow oozing moist or blistered
 - Surrounding skin: firm fibrotic & thickened by edema
 - Cellulites may present
 - Feeling of heaviness
- Treatment: compression bandages and wound care

Malignant leg ulcer is uncommon

| If you don't dare to think you might, you won't!
Except in such material you have just to memorize :* |

Best of Luck