Arterial disease

Objectives

- Discuss the **pathophysiology** and **epidemiology** of peripheral arterial disease
- Discuss the **anatomy** and **clinical features** of peripheral arterial disease
- Discuss the **diagnosis** and **managements** of peripheral arterial disease
- Recognize the **difference** between the chronic & critical lower limb Ischemia
- Discuss the **etiology** and **classification** of acute limb ischemia
- Discuss the **clinical features** and **diagnosis** of acute limb ischemia
- Discuss the **managements** and **complications** of acute limb ischemia
- Discuss the **pathophysiology** and **epidemiology** of carotid arterial disease
- Discuss the **anatomy** and **clinical features** of carotid arterial disease
- Discuss the **diagnosis** and **managements** of carotid arterial disease

Colour Index

- Main Text
- Males slides
- Females slides
- Doctor notes
- Textbook
- Important
- Golden notes
- Extra

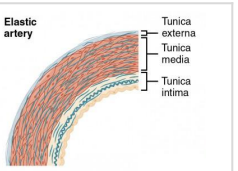
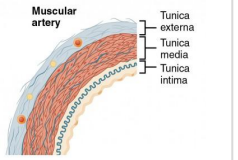
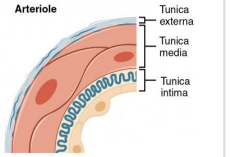
Summary File

Editing File

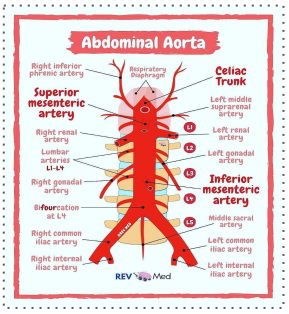
Arterial System

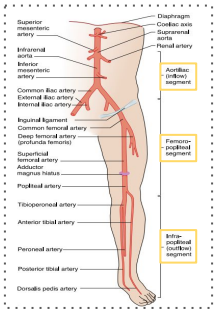
- Contains 30% of blood volume
- Normal systolic pressure <130 mmHg
- Arterial capillary pressure 25 mmHg
- High pressure/low volume system compared to the venous system

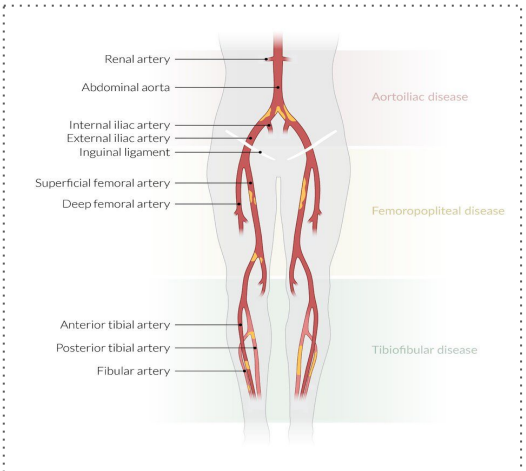
Types of arteries:

Elastic Arteries	Aorta & beginning of its large branches have predominance of elastic fibers in Media	
Muscular Arteries	Medium sized arteries, distributing arteries exhibit smooth muscles in their walls	
Small Arteries	Major site of autonomic regulation of blood flow.	

Anatomy of Peripheral Arteries







- You have to know the main branches of abdominal aorta (Celiac arteries, superior mesenteric, inferior mesenteric, renal arteries and iliac arteries)
- The abdominal aorta extended from the diaphragm till the pelvis then it divides into iliac arteries.
- Atherosclerotic diseases are described as:
  - Inflow disease
  - Outflow disease
- The aortoiliac segment above the inguinal ligament (inflow)
- The femoropopliteal segment (outflow)
- The infrapopliteal segment (outflow)

Atherosclerosis

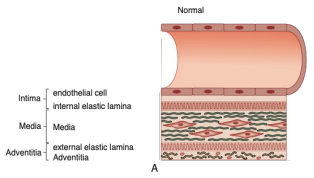
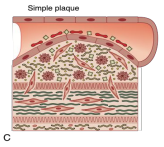
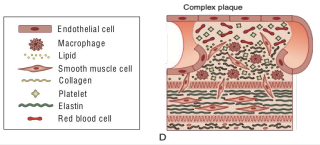
Amboss

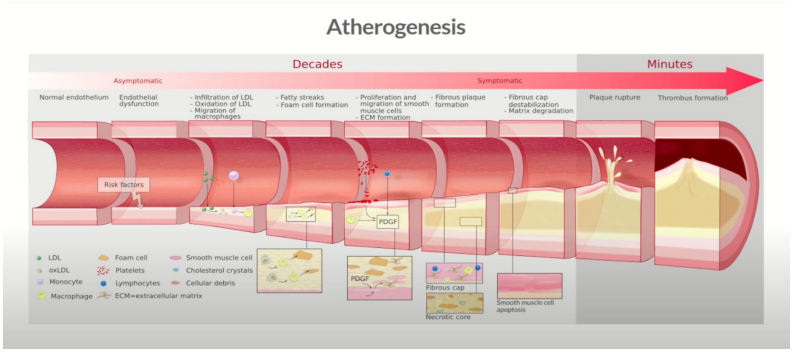
Click here for high yield osmosis flashcards

Risk factors of atherosclerosis

Non Modifiable	Modifiable – Major Factors	Other Risk Factors
• Age (males ≥ 45 years, females ≥ 55 years (postmenopause)) • Male gender • Menopause (Because in perimenopause estrogen acts as a protective factor) • Familial predisposition • Genetic	• Smoking • HTN • DM • Dyslipidemia	• Sedentary lifestyle • Obesity ★ **Elevated homocysteine** which promotes atherosclerosis through increased oxidant stress, impaired endothelial function, and induction of thrombosis. (homocysteine is an intermediate molecule that is derived from the amino acid methionine) • Stressful & competitive lifestyle • Type A personality • High carbohydrate intake

Pathophysiology of atherosclerosis

Endothelial Injury	- Chemical injury (Smoking, Hypercholesterolemia) - Physical injury - Atheroma (a reversible accumulation of degenerative tissue in the intima of the arterial wall) ➢ Hypertension increases this stress "lead to propagation of plaque to larger area"	
Fatty streak	- Increased permeability to lipids and inflammatory cells to leak into sub-endothelial area. - Leukocytes adhere into the subendothelial space and digest lipids to become **foam cells.** - Protease and free radicals liberated. - Cytokines attract more leukocytes and smooth muscle cells.	
Simple Plaque	- Smooth muscle cells exit the media. - Proliferate, take on the **characteristics of fibroblasts** and **produce collagen**, raising the atheroma.	
Complex Plaque	- Proliferation forms an endothelial cap, which may rupture, ensuing further endothelial Injury. - this results in thrombosis and distal embolization.	



Peripheral Arterial Disease (PAD)

Amboss

Click here for a helpful table from the textbook

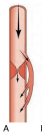
Chronic	Acute

• Slow gradual luminal stenosis secondary to plaque
• Collateral development compensate
• Symptoms proportional to disease burden For example:
  - One area of stenosis → asymptomatic
  - Multiple → claudication
  - More → peripheral ischemia
• Exertional symptoms appear first
○ **Leads to:**
  - Intermittent Claudication (**IC**)
  - Critical limb ischemia (**CLI**)
  - 10% of Diabetic foot (**DF**)
	• Emergency PADs
	• Sudden occlusion in the absence of adequate collaterals.
	• Caused by
	  - Embolism
	  - Thrombosis
	  - Injury
	
	○ **Leads to:**
	  - Acute Limb ischemia (**ALI**)

Mechanisms of injury in atherosclerotic disease

(A) Critical stenosis compensated by collateral vessels

- Symptomatic **on exercise**
- A plaque starts to slowly develop over years until there's critical stenosis. But because the plaque is developing over years **there's compensation by collaterals**. Therefore the patient is usually **asymptomatic**, however, pain can be triggered by vigorous exertion. stenosis decrease blood flow **distal** to it.



(B) Acute thrombosis of a critical stenosis

- Little change in symptoms due to collateral development
- A thrombus is formed. Which can cause critical stenosis or total occlusion. But because there's collaterals, the patient may be asymptomatic or the symptoms may be minimal

(C) Acute thrombosis of non-critical stenosis

- Severe symptoms either severe claudication or rest pain due to poorly developed collaterals

(D) Atheroembolism from ruptured plaque

- "cholesterol" emboli.

(E) Thromboembolism

- "thrombus" emboli,
- Atrial fibrillations are the most common cause
- Severe ischemia because of lack of collateral supply
- As in aortoiliac - femoral bifurcation - popliteal bifurcation

- A,B & occasionally C = chronic, Collaterals compensate, pain only after walking 300m-1km.
- D & E = Acute manifestation "emergencies-pain on rest"
- **Speed of occlusion onset is one of the major determinants of clinical manifestations of arterial diseases**
  1. **Slow development:** E.g. atherosclerosis plaque building up = give chance for collateral development → needs months or years. (e.g. profunda [deep] femoral artery collaterals around a diseased superficial femoral artery in patients with intermittent claudication).
  2. **Rapid Occlusion:** Rapid occlusion of previously normal artery = no collaterals = severe ischemia

Chronic Peripheral Arterial Disease (PAD)

1 Intermittent Claudications (IC) partial occlusion

- Up to 5% of people >60 years
- 1–2% of patients will deteriorate if they comply with best medical treatment (BMT)
- The emphasis is on the preservation of life. then limb, then function.

- The annual mortality rate is 5–10% per year,
  - 2–3-times higher than non-claudicant
  - **Marker of atherosclerosis** , (once you have one plaque, by default you have also another atherosclerotic plaques in your vessels.) and most of these patients succumb to myocardial infarction (MI), stroke and limb loss

Clinical Features

01

- **Claudication** pain is a muscular pain (affecting muscle groups)
- **Not present at rest** (Because at rest there is enough blood to met the tissue demand)
- The pain comes on after walking a particular distance, which is known as the **claudication distance**[1].

02

- It is quickly **relieved by resting** because oxygen demand of the specific muscle groups is reduced by rest or if blood flow to the muscles increases, which is achieved by lowering the affected limb due to the effect of gravity.
- It is **repetitive** (always the same distance), the patient will develop the pain after walking the claudication distance.

03

- **Typical complaining:** "When I go to the mosque, I stop by our neighbour house due to calf pain (same house every time). I stop for few minutes (relieve), & after resting I can complete my way to mosque without issue".

04

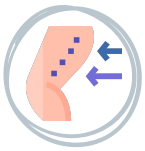
- **Other symptoms:** Due to low blood supply
  - Impotence (leriche syndrome) due to internal iliac occlusion - aortoiliac occlusion.
  - Weakness / decreased mobility
  - Skin changes
  - Toe nail changes
  - Muscle wasting

Claudications site

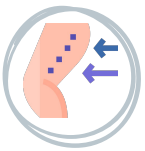
- The site of claudication gives a clue to site of arterial disease:

★ To differentiate and locate the site of occlusion. check the most proximal muscle pain, as the pain occurs distal to the occlusion.

- EXTRA: Peripheral artery disease at the level of the aortic bifurcation or bilateral occlusion of the iliac arteries that leads to the classic triad of bilateral buttock, hip, or thigh claudication; erectile dysfunction; and absent/diminished femoral pulses.



Bilateral thigh and/or buttock:
Aortoiliac arteries



Unilateral thigh and/or buttock:
Iliac arteries

- **Buttock** = Common iliac (blood doesn't reach internal iliac)
- **Thigh** = External iliac

Calf:
Femoropopliteal arteries

Foot (instep):
Tibial arteries (rare)

1. Arterial occlusion reduces oxygen supply to the muscles during exercise, oxygen demand increases but can't be met because of reduced perfusion. This imbalance leads to pain

Chronic Peripheral Arterial Disease (PAD)

2 Critical Limb Ischemia (CLI) Complete Occlusion

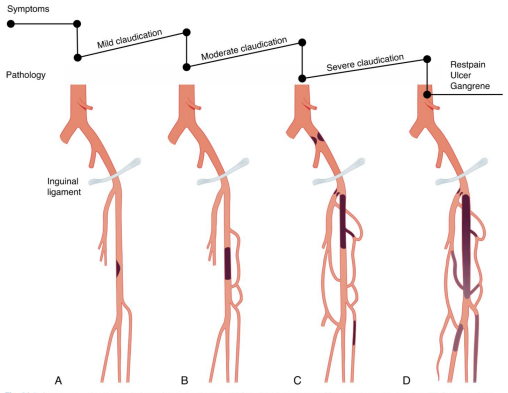


Fig. 21.5 **Symptoms and pathology in intermittent claudication.** (A) Superficial femoral artery (SFA) stenosis at adductor canal. (B) Occlusion of the SFA and development of a collateral circulation between the deep femoral (profunda femoris) artery (PFA) and the popliteal artery. (C) Iliac artery and PFA stenosis leading to worsening symptoms of intermittent claudication and further collateralisation. (D) Eventually critical limb ischaemia characterised by ischaemic rest pain and tissue loss develops due to multilevel disease affecting tibial arteries and collateral supply.

Sequence of events

A) Superficial femoral artery (SFA) stenosis "start of claudications"

B) Complete occlusion of SFA, collaterals developed from deep femoral artery (PFA)

C) Stenosis of PFA & common iliac, worsening symptoms.

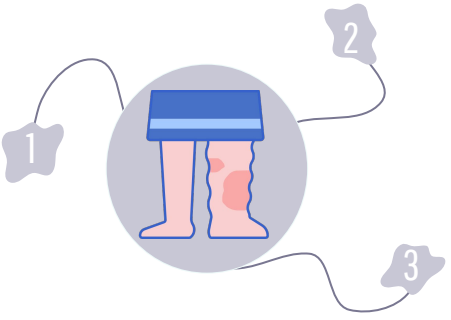
D) Critical Limb Ischemia (CLI) - **pain at rest, gangrene, ulcer.**

CLI is caused by **<u>multiple</u> lesions** affecting different arterial segments in the affected limb - When its occurs fast or in more than one area . These patients usually have:

Clinical Features

★ **Rest pain** (Continuous Pain)

- **Exacerbated by lying down** or elevation of the foot, because the patient is depending on the gravity to help deliver more blood.
- Classically felt at night and is **relieved by sleeping** with feet **hanging over the bed** or sleeping on a chair.



- The patient may present with foot swelling.
- **Tissue loss** in the form of:
  - **Ulceration**
  - **Gangrene**

Due to low blood perfusion

Chronic Peripheral Arterial Disease (PAD)

Physical Examinations

Skin

- Skin is thin and dry
- Pallor, particularly on elevation (If the leg been affected the foot might turn pale, white when it's raised called elevation pallor)
- Reduced temperature
- Upon **dependency** "postural changes", the foot becomes **bright red**; this is known as dependent rubor or 'sunset foot', and is due to **reactive hyperaemia (Buerger's test)**
- **Reactive hyperemia** is the transient increase in organ blood flow that occurs following a brief period of ischemia

- **Buerger's test** is used to assess the adequacy of the arterial supply to the leg. It's performed by positioning the patient in a supine position, then both of the patient's feet are raised to 45° for 1-2 minutes. The development of pallor indicates that peripheral arterial pressure is unable to overcome the effects of gravity, resulting in loss of limb perfusion. If a limb develops pallor, note at what angle this occurs (e.g. 25°), this is known as Buerger's angle. In a healthy individual, the entire leg should remain pink, even at an angle of 90°. Examination for venous guttering is done during Buerger's test. The patient is asked to hang their legs down over the side of the bed: Gravity should now aid reperfusion of the leg, resulting in the return of color to the patient's limb. The leg will initially turn a bluish color due to the passage of deoxygenated blood through the ischemic tissue. Then the leg will become red due to reactive hyperaemia secondary to post-hypoxic arteriolar dilatation (driven by anaerobic metabolic waste products).

Muscles & Nails

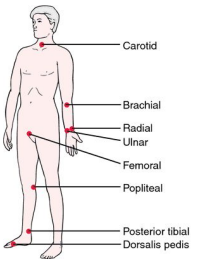
- Brittle Nails
- Muscle wasting

Veins

Superficial veins that fill sluggishly in the horizontal position and empty upon minimal elevation **(venous guttering)** (common) the body compensate the obstruction by vasodilation but it only happens in the vein and the capillaries while the arteries obstructed and the tissue still ischemic.

Pulses

- All patients must have their pulse status recorded
  - This includes: carotid, subclavian, brachial, radial, ulnar, femoral, popliteal, posterior tibial and dorsalis pedis
  - (examine all pulses)
- The pulses are recorded as normal, weak or absent
- The presence of a thrill and/or **bruit** denotes turbulent flow
- **Ankle/brachial pressure index** should be recorded (refer to vascular investigation lecture)



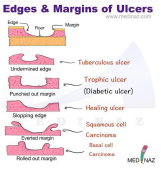
Tissue loss in (CLI)

Arterial Ulcers:

- Often located on toes or foot
- Pale and with necrotic floor
- Irregular margins
- Painful
- Surrounding ischemic features "Pinkish but non erythematous" مافيه شعر"

Chronic Peripheral Arterial Disease (PAD)

Inspection



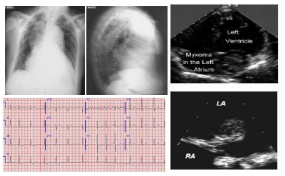
- **Site** (location)
- **Number**
- **Size**
- **Shape**
- **Floor** - The exposed part of an ulcer (Inspection)
- **Edges** – Part between the margin and the floor of an ulcer (Undermined, Punched out, Sloping, Rolled, Raised)
- **Depth** - in mm, or if you see deep structures sat its deep, if not say " i don't see deep structures so i assume it's not deep because i don't see structures"
- **Exudate** (Discharge)
- **Surrounding area**
- **Margin** - Line of demarcation between normal and abnormal

Palpation

- **Tenderness**
- **Base** - the structure on which the ulcer rests (Felt on palpation) - What's the difference between floor and base? The floor is what you see, and the base is what you feel on palpation
- **Relation** with Deeper structures
- **Examination of Surrounding Area**
- **Examination of Lymph Nodes**
- **Examination of the pulse.**

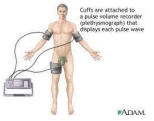
PAD Investigation

- CBC, Electrolytes, Creatinine - changes to the renal arteries which increases the risk of having a chronic kidney disease
- **Coagulation profile:**
  - aPTT "activated partial thromboplastin time": It measures how long it takes your blood to form a clot
  - INR "international normalized Ratio": calculation based on results of a PT and is used to monitor individuals who are being treated with the blood-thinning medication (anticoagulant) warfarin (Coumadin®). It helps to find out if your blood is clotting normally. It also checks to see if a medicine that prevents blood clots is working the way it should.
- Type and screen
- Lipid profile
- **Hemoglobin A1c** **(modifiable risk factors)** don't trust the patient when it comes to diabetes it's important to measure HB_{A1c})
- Chest X Ray
- ECG
- Echocardiogram (By default, any patient with PAD already have coronary disease, but the question "how severe is it?")



Intermittent claudications (IC):

- ★ **Ankle Brachial Index (ABI) =** 0.8-0.4
  - ★(Normal≥0.9) (>1.3 is considered false positive, in patient with DM their vessels are calcified so it can't be be compressed enough to read the pressure.)
- **Toe pressures =** <50 mmHg
- **Segmental pressure** (pressure difference e.g: between thigh and leg) = 20 mmHg reduction
- **Volume Plethysmography** = Measures arterial volume changes
- **Duplex Ultrasound =** Stenosis or <u>single</u> occlusion
- **CT Angiogram & MRA** (same as duplex)
- **Invasive Vascular Investigations**



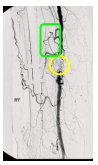
Critical Limb Ischemia (CLI): (worse)

- **Ankle Brachial Index (ABI) =** <0.5
- **Toe pressures =** <30 mmHg
- **Segmental pressure**
- **Volume Plethysmography**
- **Duplex Ultrasound =** <u>Multiple</u> stenosis or occlusion
- **CT Angiogram & MRA** (same as duplex)
- **Invasive Vascular Investigations**

MRI
Tibial occlusion and popliteal occlusion.

CT Scan
Reconstruction

Angiogram



Green: collaterals.
Yellow: occlusion

Chronic Peripheral Arterial Disease (PAD)

IC & CLI Management:

1 Primary Prevention

- Modifiable risk factors-lifestyle changes: (BP/DM/dyslipidemia) If you have a patient with atherosclerostic arterial disease, you have to consider primary prevention of PAD as the most important step in your management. **best treatment)**
- This can happen by reducing weight, being active.

2 Secondary Prevention - Best Medical Treatment

1- BMT

- **All** patients should be strongly urged to comply with **Best Medical Therapy (BMT):**
  - Cessation from **smoking (most important)**
  - Control of **hypertension** (ACE Inhibitors)
  - Prescription of a **statin** despite the absence of dyslipidemia - Because statins have an anti-inflammatory effect that inhibits the migration of inflammatory cells, which slows the progression of the disease and improves the plaque's morphology.
  - Prescription of **antiplatelet agent** : aspirin[1] (81 mg daily), or clopidogrel[2] (75mg daily)
  - Regular **exercise**
  - Control of **obesity**
  - The identification and treatment of patients with **diabetes (HbA1c<7%)**

2- Walking Exercise Program

- Along with BMT
- You ask the patient to walk on a flat surface for 3 months, 3 days/week, 30 Minutes/day. Tell the patent to push themselves when they have the pain, and once its excruciating stop for a rest.
- If patient can't walk (E.g: more than 5 minutes). Ask him to walk these 5 minutes, and push himself for additional 1 or 2 minutes, all these are included in the 30 minutes.
- This can help:
  - Improve the collaterals
  - Train muscles to use less O2 (anaerobic respiration)

3- Compliance

- **Many patients fail to comply** (For many causes, E.g: feel it's hard to walk in hot weather)
- **Compliance with BMT & walking exercise program increases:**
  - Walking distance, Affords protection against cardiovascular events, Improves the quality of life and life expectancy, BMT reduces the overall intervention risks and increases the likely success

1. Irreversible cyclooxygenase inhibition → decreased thromboxane A2 synthesis → decreased platelet aggregation.
2. Inhibition of the P2Y12 ADP receptor → decreased platelet activation and platelet-fibrin crosslinking

Chronic Peripheral Arterial Disease (PAD)

3 Treatment: Endovascular and surgical interventions

- Endarterectomy
- Percutaneous Angioplasty
- Bypass procedures

Indications for intervention

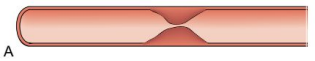
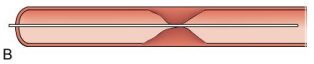
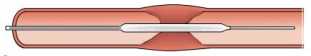
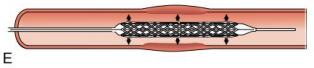
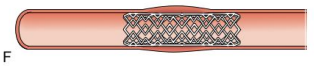
1. **Disabling claudication pain**, Depends:
   - Not needed in Elderly >70 y/o presented with claudication for 400m (High risk, low benefit of surgery)
   - In the other hand, It's needed in patient who is 40 y/o & work as security (needs to walk 1 km or more daily) if presented with the same claudication for 400m .
   - Of course, you need trying to improve his condition by BMT & Exercise firstly.

2. **CLI** - Always, due to tissue loss (necrosis and gangrene)

Intervention Includes

- **Balloon angioplasty** ± stenting
- **Surgery**

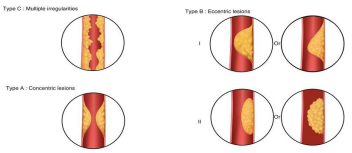
1) Balloon-Angioplasty

A. The lesion is identified (Critical arterial stenosis)	 A
B. **Lesion** crossed with a wire	 B
C. A balloon "angioplasty catheter" is inserted	 C
D. And inflated, This enlarges the lumen by disrupting the plaque	 E
E. In patients with occlusions and complex disease, stents may be deployed	 F

(Some plaque will disappear once you insert the balloon and some need a stent to keep it open)

a. Some plaques comes back and recluse the lumin, in such cases we need to mount a stent.
b. The benefit of stents is to keep a radial force on the vessels to prevent reclusion

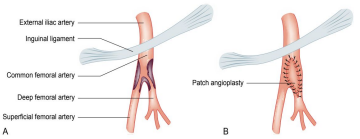
- Drug-eluting (**Paclitaxel** → chemo drug which has local effect) reduce inflammatory reaction balloons and stents reduce the neointimal hyperplasia that can lead to restenosis and occlusion
  - Balloon are either normal (conventional balloons)
  - Or drug coated Balloons that is coated by chemotherapy agent to prevent neointimal hyperplasia (Arterial scarring)
- **Favorable** lesions - **short** concentric stenosis
- **Unfavorable** lesions (it means three months later you need another angioplasty)- **long** eccentric stenosis or occlusion



Chronic Peripheral Arterial Disease (PAD)

3 Endovascular and surgical interventions

2) Surgery



a. Endarterectomy

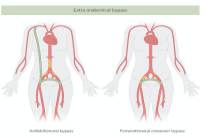
- Direct removal of atherosclerotic plaque and thrombus, for patients who have plaques at the site of bifurcation usually done at the **carotid and femoral bifurcations.**
- The surgeon will make a cut in the blocked part of the artery and remove the plaque that is blocking the blood flow.
- Then the artery will be closed by performing either a primary closure or patch angioplasty (patch made out of either synthetic material or bovine pericardium). **Patch angioplasty** (Patch because you opened the artery) is the preferred technique. Why? Patch angioplasty reduce the risk of restenosis due to hyperplasia and scar tissue formation and, therefore, reduce the risk of recurrent blockage and consequent stroke or death.
- نسويها بأماكن محددة very easy to access and has no muscle

b. Arterial reconstruction: Bypass grafting

For a **bypass** operation to be successful in the long term, **three** conditions must be fulfilled:

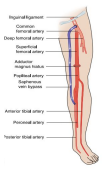
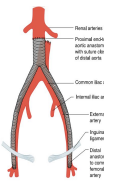
- There must be **high-flow**, high-pressure blood entering the graft **(inflow)**
- The **conduit** must be suitable, (vein or prosthetic)(must be healthy e.g. no varicose)
- The blood must have somewhere to go when it leaves the graft **(outflow or run-off)**

We do it in long occlusion:
1- You failed the angioplasty
2- or you can't do the angioplasty
The anatomical is better because it mimic the effect of the healthy anatomy



Types of conduit:

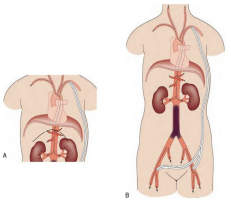
- **Anatomical conduit:** Blood follows the original artery it flows with the anatomy
- **Extra-anatomical conduit:** any bypass graft that is placed outside of the normal anatomic vascular pathway.

Two main types of conduit are available "Anatomical"	
Autogenous Material	**Prosthetic Material**
Most commonly a vein (most commonly the ipsilateral great saphenous vein GSV) (either by flipping the vein upside down or removing the valves) The main advantage of vein is that it is lined by endothelium that is actively antithrombotic and profibrinolytic, and therefore much less liable to induce coagulation than even the most inert of manmade materials. Vein is also much more resistant to infection and less expensive. **Picture:** Femoro-distal bypass 	Most commonly expanded polytetrafluoroethylene (ePTFE) or Dacron. Prosthetics grafts are prone to infections. **Picture:** Aorto-bifemoral bypass 

Extra-anatomical Bypass:

When you can't open the abdomen (70 year old, EF very low)

- Lesser procedures and preferred in high-risk patients or those that **have a limited life expectancy**. -those who cannot tolerate laparotomy-
  - Fem-Fem (femoro-femoral) crossover for patients with unilateral occluded iliac
  - Axillobifemoral if both iliac arteries are occluded
- Do not have as good long-term patency as anatomic -Maximum 3-4 years-



4 Choice of treatment

Choice of treatment decided depending on :
Patient symptoms, Comorbidities, Life expectancy, Risk and benefits, Anatomy of the disease, Prior interventions

Chronic Peripheral Arterial Disease (PAD)

3 Diabetic Foot (DF)

- Approximately 40% of patients with CLI have diabetes
- Combination of **ischemia**, **neuropathy** and **immunocompromised patient**
- **Diabetic neuropathy affects the motor, sensory and autonomic nerves**
- Diabetic **neuropathy** may lead to foot ulceration in its own, and also complicates peripheral ischemia
- Arteries are often calcified
- These patients usually have very Severe multisystem arterial disease (CAD, CVD and PAD)
- Diabetic vascular disease has a tendency for the infrapopliteal vessels
    - "Tibial clots", remember the 3 conditions the must be fulfilled for a bypass operation to be successful? These patients have poor outflow due to diseased small vessels etc..
- The feet of diabitc patients are very susceptible to sepsis, ulceration and gangrene.

01

Sensory Neuropathy:

- Patient incapable of **feeling pain.**
- It affects **proprioception** such that, when walking, pressure is applied at unusual sites.
    - Abnormal walking leads to joint disruption as you see in the picture
- This leads to ulcer formation and joint destruction (**Charcot's Foot).**

02

Autonomic Neuropathy:

- Dry foot deficient in the sweat that normally lubricates the skin and contains antibacterial substances.
- Causing scaling and fissuring.
- Abnormal flow in the bones due to loss of autonomic control may also contribute to osteopenia and bony collapse which causes Charcot's foot.

03

Motor Neuropathy:

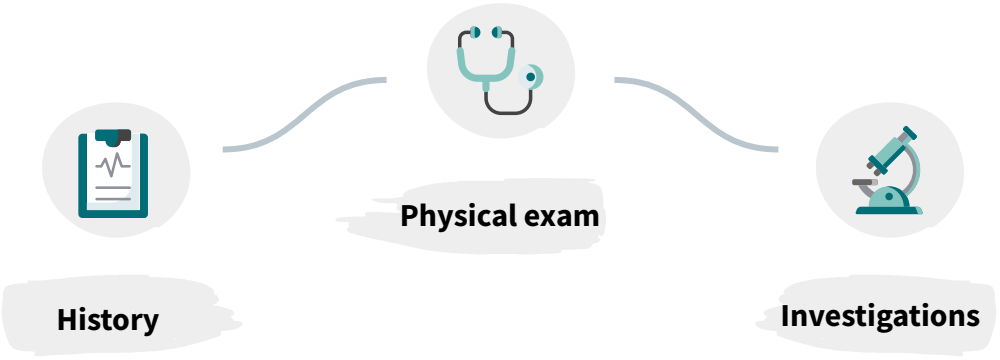
- The flexors are affected more than the extensors.
- The extensors are unopposed and the toes become dorsiflexed بصير بمسي واطراف اصابعه مرفوعه.
- This dorsiflexion exposes the metatarsal heads to abnormal pressure, and they are a frequent site of callus formation (thickened skin) and ulceration. - as in the picture

Callus diagram located under second, third and fourth metatarsal head

Chronic Peripheral Arterial Disease (PAD)

DF Management:

- **Diabetic foot diagnosis is similar to PAD:**



1 Diabetic Foot prevention

- **Diabetic control (Hb_{A1c} <7%)** most important approach to prevent DF
- Comprehensive **behavioral foot care education**
  - **Washing** the feet with soap daily and dry it thoroughly -Dry it more than once per single wash
  - **Use a file** to shape the nails (not a clipper)
  - Keep the **skin moisturized** the cream should be unscented and small in quantity. A socks should be worn after the cream dries.
  - **Don't walk barefoot**
  - Change daily into **clean soft socks** -Must be cotton socks
  - **Daily foot inspection** for injuries
  - Therapeutic **footwear**

2 Diabetic Foot Management

- **If the blood supply to the foot is adequate**
  - Excise dead tissue -Considering that it will grow back again
  - Control the Infection (antibiotics)
  - Protected the foot from pressure (off-loading) - By either a cast or boots, this is orthopedic job
- **If there is ischemia, the priority is to revascularize the foot, if possible** -This is our job, we treat him as any CLI condition "if possible" " you can't revascularize below the ankle"
- Many patients present late, with extensive tissue loss and unreconstructable disease accounting for the **very high amputation rate**

Acute Peripheral Arterial Disease (PAD)

Acute Limb Ischemia (ALI)

- **Acute limb ischemia is caused most frequently by**
  - Acute **thrombotic** occlusion of a preexisting stenotic arterial segment (60%)
  - **Embolism** (30%)
  - Trauma
- Distinguishing between **thrombosis** and **embolism** is important because investigation, treatment and prognosis are different.
- In thrombosis we can wait on the patient but in embolism its considered an emergency.

Thrombosis

- **Thrombosis in situ may arise from:**
  - Acute plaque rupture
  - Hypovolaemia
  - Increased blood coagulability (e.g., in association with sepsis, malignancy)
  - Pump failure (e.g., cardiac event)

Embolism

- More than **70% of peripheral emboli are due to Atrial Fibrillations**
- **ER**

On the basis of onset and severity:

Incomplete acute ischemia

01

- Usually due to **thrombosis** in situ.
- Can often be treated medically, at least in the first instance
  - Patient with CLI, presented with acute rest pain. There's a pain, but the patient is able to move his foot. Such patient can wait on, give him heparin, confirm diagnosis by investigations, then treat him.

Complete ischemia

02

- Usually due to **embolus.**
- Normally result in extensive irreversible tissue injury within 6 hours unless the limb is revascularized.

Irreversible ischemia

03

- Mandates early amputation or, if the patient is elderly and unfit, end-of-life care.

Acute Peripheral Arterial Disease (PAD)

2 **distinctive** features of acute ischemia

- **P**aralysis (inability to wiggle toes or fingers)
- **P**aresthesia (loss of light touch over the dorsum of the foot or hand)

Both indicate loss of function which is the most important feature of acute limb ischaemia and denotes a threatened limb that is likely to be lost unless it is revascularized within a few hours.

(ALI) Signs & Symptoms (6Ps)

4 additional features of acute ischemia that assist the diagnosis

- **P**ain (May be absent in complete acute ischemia & severe pain in chronic ischemia).
- **P**allor (feature of chronic ischemia also)
- **P**oikilothermia - Perishing cold: (Cold foot is Unreliable, as the ischaemic limb takes on the ambient temperature)
- **P**ulselessness (feature of chronic ischemia also)

ALI Early Stage

- Acute complete ischemia is associated with intense distal arterial spasm and the limb is **'marble' white** as shown in the picture below
- As the spasm relaxes over the next few hours and then fills with deoxygenated blood, **mottling appears**
  - **This appears light blue or purple** has a fine reticular pattern, and on pressure, so-called **non-fixed mottling** when you push it will blanch (color will change)
- At this stage, the limb is salvageable you can mostly save the limb by surgery.

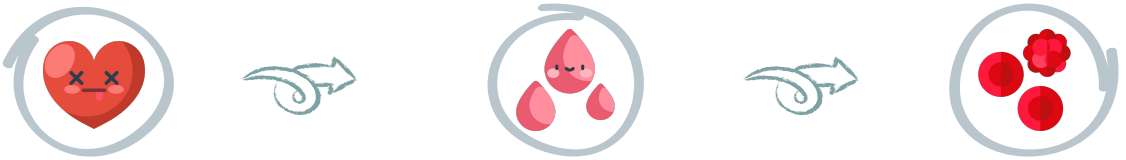
ALI Late Stage

- As ischemia progresses, **blood coagulates in the skin**, leading to **fixed mottling** that is **darker in colour and does not blanch on pressure**
- Blistering and **liquefaction** - beyond the skin, occurs after skin coagulation.
- Treatment: amputation. Because attempts at revascularization are futile and will lead to life-threatening reperfusion injury (will be discussed later on this lecture)

Acute Peripheral Arterial Disease (PAD)

Thrombosis in-situ

- **Thrombosis-in-situ** Generally occurs in vessels affected by **pre–existent atherosclerosis**
- Ischemia is often **less severe** than with acute embolism
- Location of occlusion may play a role in the severity of limb ischemia
- **Causes of** exacerbation acute-on-chronic attack include:



Pump failure (e.g., silent or overt MI)

Hypovolaemia, which may be associated with widespread thrombosis

Increased blood coagulability (e.g. sepsis, malignancy)

01

- **Many patients can be managed medically** -Especially in the early consequences. Give them anti-coagulants & follow-up

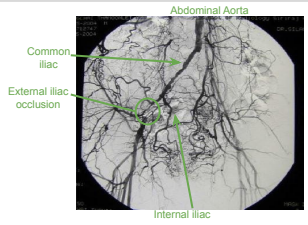
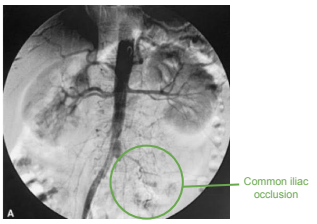
02

- If the limb remains threatened after one day or two of follow-up then it may be possible to clear thrombus by:
  - Thrombectomy
  - Endoluminal techniques -E.g: Balloon catheter angioplasty
  - Thrombolysis Takes time, therefore it's not used for patients with acute ischemia due to embolus. It's not used for all cases because it may cause hemorrhage
  - Bypass

03

- If an urgent intervention is required, the in-hospital limb loss rate may approach 30%, with an in-hospital mortality rate of 10–20%

Treatment

Thrombosis-in-situ	Embolism
Previous claudication	No previous symptoms of arterial insufficiency
No source of emboli	Obvious source of emboli (atrial fibrillation, myocardial infarction)
Long history (days to weeks)	Sudden onset (hours to days)
Less severe ischemia	Severe ischemia
Lack of pulses in the contralateral leg	Normal pulses in the contralateral leg
Positive signs of chronic ischemia	No signs of chronic ischemia
 (Acute Thrombosis) lots of collaterals	 (Acute embolus) no collaterals

Acute Peripheral Arterial Disease (PAD)

(ALI) management

- Must be discussed immediately with a vascular surgeon
- Blood work, ECG, and cross-match
- If there are no contraindications, **IV heparin** (5000-8000 IU) is administered
  - To limit propagation of thrombus and protect the collaterals.

01

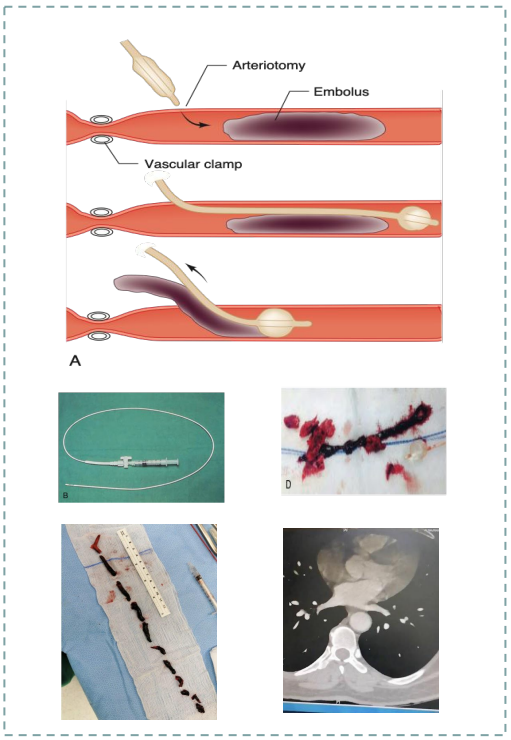
★ If ischemia is **complete**
- The patient proceeds for embolectomy -we have to remove this embolism as fast as we can.

02

- If ischemia is **incomplete** - patient can wait
  - Preoperative **imaging** is obtained (simple embolectomy or thrombectomy is unlikely to be successful they already have a chronic plaque, they need bypass or angioplasty)
  - Preoperative optimization

Acute Embolus

- Femoral embolus is associated with ischemia to the upper thigh.
- Acute embolic occlusion of the aortic bifurcation (saddle embolus) leads to absent femoral pulses and having white or mottled waist & legs.
  - May also present with paraplegia due to ischemia of the cauda equina.



- Embolectomy (using Fogarty Catheter) can be performed under Local Anesthesia or General Anesthesia
- Postoperatively, the patient should continue on IV heparin
- Warfarin reduces the risk of recurrent embolism but is associated with an annual risk of significant bleeding of 1–2%
- In-hospital mortality from cardiac death or recurrent embolism,e.g. stroke, is 10-20%
- Embolectomy:
  - Balloon embolectomy is done by inserting a catheter with a small inflatable balloon attached at the end into the artery and past the clot. The balloon is then inflated and slowly pulled back out of the artery, removing the clot with it.
  - We calculate the distance between the embolism & toe, then insert the catheter as far as we can.
  - We do the procedure in opposite “insert the catheter distally” in those who have embolism in the aortic bifurcation.

Post-Ischemic Syndrome

01

Compartment Syndrome (Local)

Pathophysiology

- Endothelial cell injury leads to increased permeability **Postoperative or post treatment Calf muscles swelling**
- The calf muscles are confined within tight fascial compartments -Which compress the swollen muscles
- The increase in interstitial tissue pressure leads to muscle necrosis despite adequate arterial inflow: **compartment syndrome (>25mmHg)**
- There is swelling and pain on squeezing the calf muscle or moving the ankle
- Palpable pedal pulses **do not** exclude compartment syndrome
  +ve Pedal pulse indicates good perfusion of large vessels, compartment syndrome affects the small vessels that supply the individual muscle.

Management

- **Prevention** through expeditious **revascularisation**
- ★ Low threshold for **fasciotomy** to relieve the pressure
  - Medial incisions = for **posterior** compartment (both deep & superficial)
  - Lateral incisions = for **anterior** and **lateral** compartment

02

Reperfusion Injury (Systemic)

Pathophysiology

- **Caused by activated neutrophils, free radicals, enzymes, hydrogen ions, carbon dioxide, potassium and myoglobin released from reperfused tissue -Washed out due to reperfusion of necrotic tissue**
- Leads to:
  - Acute respiratory distress syndrome (ARDS)
  - Myocardial stunning[1]
  - Endotoxemia
  - Acute Tubular Necrosis
  - Multiple organ failure and death

Management

- **Hydrate** the patient
- **Communication** with the anesthesiologist and intensivist
- Protect the heart with **calcium**
- **Prevent** and treat hyperkalemia before reperfusion Or produce hypokalemia until reperfusion is accomplished
- **Correct acidosis and produce alkalosis** in anticipation to reperfusion
- Use **inotropic** support liberally
  - if patient had myocardial stunning and low blood pressure. He may need inotropic agent to keep the BP high enough to preserve the vital organs

1. Myocardial stunning or transient post-ischemic myocardial dysfunction is a state of mechanical cardiac dysfunction that can occur in a portion of myocardium without necrosis after a brief interruption in perfusion, despite the timely restoration of normal coronary blood flow.

Cerebrovascular Disease (CVD)

Stroke	Transient ischaemic attack (TIA)	Amaurosis fugax
An episode of **focal** neurological dysfunction lasting **> 24 hours**, of vascular etiology	• Symptoms last for **< 24 hours** • E.g: "Patients had a slurred speech for half a day, then it got back to normal."	• **Transient incomplete -sometimes complete- unilateral loss of vision,** NEVER synchronously bilateral • A veil or curtain coming across the eye • E.g: "patient feel like there is a curtain closing in front of his eyes"

Clinical Assessment:

01

- **Complete History**
- Risk factor assessment

02

- **Complete Neurological exam**
- It is important to exclude other causes of cerebral ischemia and haemorrhage

03

- 'Carotid' bruit has **no reliable relationship** to the severity of the internal carotid artery disease and the risk of stroke

Pathophysiology:

- Approximately **80% of strokes are ischemic**
- **About half** of these are thought to be due to **atheroembolism** from the **carotid artery** -Both cranial and sub-cranial.

★ The origin of the internal carotid artery is most common site of **atheroma** formation -comparing to the middle internal carotid and MCA-"middle cerebral artery" -

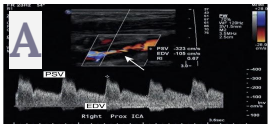
- The tighter the degree of stenosis, the more likely it is to cause symptoms

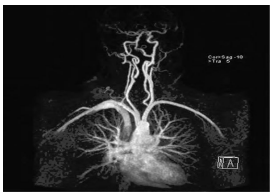
- Emboli entering the ophthalmic artery leads to amaurosis fugax or permanent monocular blindness on the same side **(ipsilateral)**
- If they enter the middle cerebral artery they may cause hemiparesis and hemisensory loss on the opposite side **(contralateral)**
- If the dominant hemisphere is affected there may also be dysphasia (language disorder marked by deficiency in the generation of speech, and sometimes also in its comprehension, due to brain disease or damage)
- If someone was left handed, and get a stroke in right side, he will have:
  - Dysphasia
  - Ipsilateral vision loss
  - Contralateral weakness

Cerebrovascular Disease (CVD)

CVD Investigations: (There will be a separate lecture for investigations)

- **Doppler (duplex) ultrasound**
  - We check the peak systolic volume (PSV), End Diastolic Volume (EDV)
  - You can see also the narrowing in picture A "arrow", this most likely is the stenosis
- **Magnetic resonance angiography (MRA)**
- **Computed tomographic angiography (CTA)**
- Intra-arterial digital subtraction **angiography** is associated with risk of TIA/stroke as it is an invasive procedure
  - Catheter or contrast may disrupt the plaques.

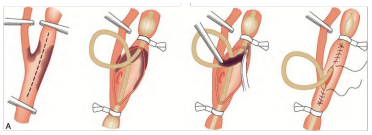




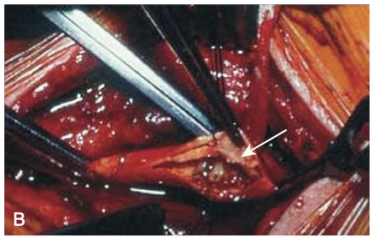
CVD Management:

- **Asymptomatic patients are treated with BMT (Best Medical Therapy)**
  - Because, the risk of developing TIA/stroke are low (< 10% at 5 years)
  - The Relative Risk Reduction (RRR) is 50%, the Absolute Risk Reduction (ARR) would be only 1% per year
  - The number needed to prevent one TIA or stroke is at least 20–30 (Carotid endarterectomy)
  - While, the number needed to treat for symptomatic disease is less than 10

1 Carotid Endarterectomy (CEA)



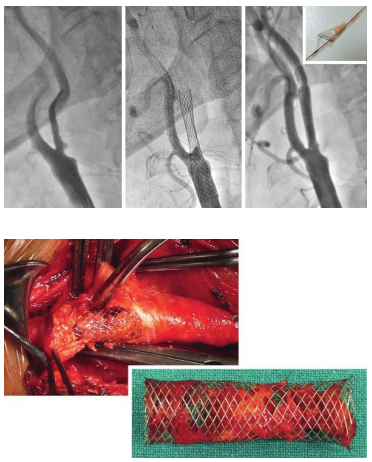
- Here, We insert a shunt -the yellow pipe- to pass blood into brain during surgery.
- We also make the carotid wider than its normal size to compensate neointimal hyperplasia



- CEA with BMT is associated with a significant reduction in recurrent stroke, compared with BMT alone
- **Patient must fulfill this criteria to be suitable for CEA**
  - ICA stenosis (> 50%)
  - Life expectancy of at least 2y
  - Undertaken with a stroke and/or death rate of <5%
  - The intervention can be performed soon
- The sooner the better
- Performed under GA or LA (General or Local anaesthesia)
- Patients with major stroke and little in the way of recovery are not candidates for carotid intervention -they can't tolerate the surgery
- Patients with an occluded Internal carotid are not candidates for carotid intervention -The risk of opening the artery is higher the benefits

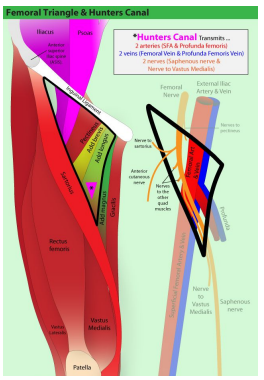
Cerebrovascular Disease (CVD)

2 Carotid Artery Stenting (CAS)



- The role of (CAS) remains controversial -Not prefered
- **Have 2 benefit only:**
  - Avoids a neck wound and the risks of cranial nerve injury
  - Reduces the risk of MI
- **Short-term risks of clinical and subclinical strokes are greater than CEA**
- CAS should be **reserved for**
  - Patients where **CEA is not possible or desirable** because of anatomic and clinical factors (e.g., **recurrent** stenosis after previous surgery or **radiation arteritis)**
- You can see In picture B this patient who undergoes CAS, the stent precipitated strokes as you see in the second picture due to the neointimal hyperplasia and the plaques formation.

Recall



Q1:What must be present for a successful arterial bypass operation?
Answer:
1. Inflow (e.g., patent aorta)
2. Outflow (e.g., open distal popliteal artery)
3. Run off (e.g., patent trifurcation vessels down to the foot)

Q2: What is the most common site of arterial atherosclerotic occlusion in the lower extremities?
Answer: Occlusion of the SFA in Hunter's canal

Q3: What is intermittent claudication?
Answer: Pain, cramping, or both of the lower extremity, usually the calf muscle, after walking a specific distance; then the pain/cramping resolves after stopping for a specific amount of time while standing; this pattern is reproducible.

Q4: What is the site of a PVD ulcer versus a venous stasis ulcer?
PVD ulcer answer: PVD arterial
Venous stasis ulcer Answer: insufficiency ulcer—usually on the toes/foot
Venous stasis ulcer—medial malleolus (ankle)

Q5: What ABIs are associated with normals, claudicators, and rest pain? Normal ABI:
Answer:
≥1.0
Claudicator ABI: <0.6
Rest pain ABI: <0.4

Summary

Recall

Q6: What are the indications for surgical treatment in PVD?
Answer: Use the acronym **"STIR":**
Severe claudication refractory to conservative treatment that affects quality of life/livelihood (e.g., can't work because of the claudication)
Tissue necrosis
Infection
Rest pain

Q7: How can the medical conservative treatment for claudication be remembered?
Answer: Use the acronym **"PACE":**
Pentoxifylline **A**spirin
Cessation of smoking **E**xercise

Q8: What is the risk of limb loss with claudication?
Answer: 5% limb loss at 5 years (Think: 5 in 5), 10% at 10 years (Think: 10 in 10)

Q9: What is the risk of limb loss with rest pain?
Answer: >50% of patients will have amputation of the limb at some point

Q10: What is a FEM-POP bypass?
Answer: Bypass SFA occlusion with a graft from the **FEM**oral artery to the **POP**liteal artery

Q11: What is a FEM-DISTAL bypass?
Answer: Bypass from the **FEM**oral artery to a **DISTAL** artery (peroneal artery, anterior tibial artery, or posterior tibial artery)

Q12: What graft material has the longest patency rate?
Answer: Autologous vein graft

Q13: What type of graft is used for below-the-knee FEM-POP or FEM- DISTAL bypass?
Answer: Must use vein graft; prosthetic grafts have a prohibitive thrombosis rate

Q14:What is DRY gangrene?
Answer: Dry necrosis of tissue without signs of infection ("mummified tissue")

Q15: What is WET gangrene?
Answer: Moist necrotic tissue with signs of infection

Q16: What is blue toe syndrome?
Answer: Intermittent painful blue toes (or fingers) due to microemboli from a proximal arterial plaque

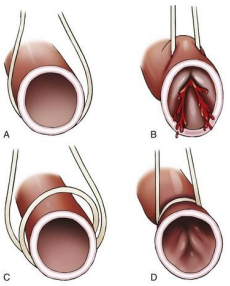
Summary

Recall

Q17: What is atherosclerosis?
Answer: Diffuse disease process in arteries; atheromas containing cholesterol and lipid form within the intima and inner media, often accompanied by ulcerations and smooth muscle hyperplasia

Q18: What is the common theory of how atherosclerosis is initiated?
Answer: Endothelial injury → platelets adhere → growth factors released → smooth muscle hyperplasia/plaque deposition

Q19: What are the common sites of plaque formation in arteries?
Answer: Branch points (carotid bifurcation), tethered sites (superficial femoral artery [SFA] in Hunter's canal in the leg)



Q20: What is the major principle of safe vascular surgery?
Answer: Get proximal and distal control of the vessel to be worked on!

Q21: What does it mean to "POTTS" a vessel?
Answer: Place a vessel loop twice around a vessel so that if you put tension on the vessel loop, it will occlude the vessel

Q22: What is the suture needle orientation through graft versus diseased artery in a graft to artery anastomosis?
Answer: Needle "in-to-out" of the lumen in diseased artery to help tack down the plaque and the needle "out-to-in" on the graft

Q23: Which arteries supply the blood vessel itself?
Answer: Vaso vasorum

Q24: What is a true aneurysm?
Answer: Dilation (>2 × nL diameter) of all three layers of a vessel

Q25: What is a false aneurysm (a.k.a pseudoaneurysm)?
Answer: Dilation of artery not involving all three layers (e.g., hematoma with brous covering) Often connects with vessel lumen and blood swirls inside the false aneurysm

Q26: What is "ENDOVASCULAR" repair?
Answer: Placement of a catheter in artery and then deployment of a graft intraluminally

438's Quiz

Q1: 63 year old patient known to be smoker and have HT & DM presented with 500m claudications, what is the proper first step in management?

A) Smoking cessation
B) Administer heparin
C) Start aspirin
D) Balloon Angioplasty

Q2: 63 year old patient presented with pain in his lower limbs, the pain is presented during rest & relieved with feet hanging over the bed, what is the most likely diagnosis?

A) Chronic Limb Ischemia
B) Acute Limb Ischemia
C) Chronic Venous Insufficiency
D) Critical Limb Ischemia

Q3: Patient have claudications in the left thigh, which of the following arteries is the most likely to be occluded ?

A) Femoral artery
B) Tibial artery
C) Popliteal artery
D) External Iliac Artery

Q4: A 51 year old who known to have CLI and got an operation done, postoperative his leg is swollen and tender with fever, What is the proper management for this patient?

A) Embolectomy
B) Fasciotomy
C) Balloon Angiopathy
D) Bypass grafting

Q5: A 68 year old patient diagnosed with CVD, he presented with dysphasia, right sided vision loss, Left-sided weakness. What is the site of lesion ?

A) Right common carotid
B) Right Internal carotid origin
C) MCA
D) Left Internal carotid origin

Answers

Q1	A	Q4	B
Q2	D	Q5	B
Q3	D		

439's Quiz

Q1: A 60-year-old woman has been diagnosed as having claudication of the lower limbs which does not impair her lifestyle. The patient is a smoker and has hyperlipidaemia for which she is taking a 'statin'. You are asked to discuss with the patient the treatment options available to her. From the list below, choose the recommended treatment option for this patient.

A) Angioplasty
B) Amputation
C) Lower limb bypass
D) Start an antiplatelet, increase exercise and quit smoking

Q2: You see a 60-year-old man with a history of coronary heart disease, diabetes and hyperlipidaemia in your clinic. The patient has found it increasingly hard to walk due to the gradual increase in intensity of the cramping pain he experiences in his right leg on walking, which is relieved by resting a few minutes. In addition, he tells you that cramps have started to occur at night when he is sleeping. On examination of the right leg, you notice that there is a 'punched out' ulcer on the right heel. The right posterior tibial and dorsalis pedis pulses are weak. You suspect that this patient has critical limb ischaemia. What is the most appropriate next line investigation that would support your diagnosis?

A) CT angiography
B) Ankle–brachial pressure index
C) Radiograph the lower limbs
D) None of the above

Q3: Which of the following statements are true?

A) Intermittent claudication may be present at rest.
B) Intermittent claudication is commonly relieved by getting out of bed.
C) Intermittent claudication is most commonly felt in the calf.
D) Intermittent claudication distance is usually inconsistent on a day-to-day basis for a given patient

Q4: You assess a patient with a plantar ulcer who has poorly controlled diabetes. From the list of options below, select the most likely management plan.

A) Optimise glycaemic control
B) Reduce plantar pressure by ensuring good footwear
C) Ensure podiatry input
D) Assess vascularity of the limb
E) All of the above

Q5: You are asked to see a 67-year-old woman admitted with severe limb ischaemia. Your senior colleague asks you to examine the patient and report your findings. What are the two most likely clinical features that suggest the patient has severe limb ischaemia?

A) Pulselessness and pain
B) Pallor and pain
C) Paraesthesia and paralysis
D) Paraesthesia and pallor

Q6: You are in the vascular surgery outpatient clinic explaining the indications for undergoing carotid endarterectomy to a patient. From the list below, select the most likely scenario where carotid endarterectomy is likely to be indicated.

A) Symptomatic carotid artery stenosis of greater than 50%
B) Asymptomatic carotid artery stenosis of between 70% and 80%
C) Symptomatic carotid artery stenosis of less than 50%
D) None of the above

Answers

Q1	D	Q4	E
Q2	B	Q5	C
Q3	c	Q6	A

Extra Questions

Good Luck!

Team leaders:

Reem Alqahtani

Shayma Alghanoum

Sarah AlQuwayz

Mona Alomirainy

This lecture was done by:

- Yara Alasmari
- Raina Almutairi
- Aljoharah albnyan
- Yara Alasmari
- Lama alahmadi
- Ghada Alothman

Note taker Reviser

Feedback