

Arterial disease

Objectives:

- Describe the pathophysiology of atherosclerosis.
- Describe the etiology, clinical features, investigations & managements of chronic lower limb ischemia.
- Explain the differentiating features & significance of critical limb ischemia.
- Describe reperfusion injury & explain its management.
- Describe pathogenesis & management of diabetic foot.
- Describe the etiology, clinical features, investigations & management of carotid artery atherosclerotic disease.

Color index:

Main Text

Males slides

Females slides

42 Doctor notes

Doctor notes

Textbook

Important

Golden notes

Extra

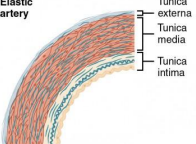
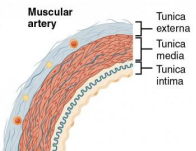
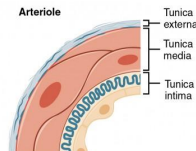
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Arterial System

- ❖ 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



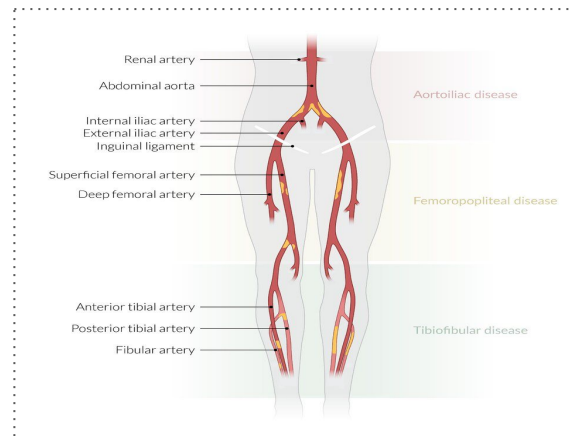
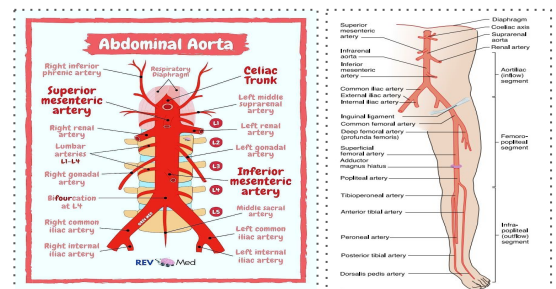
Types of Arteries:

Elastic Arteries	Aorta & beginning of its large branches have predominance of <u>elastic</u> fibers in Media	
Muscular Arteries	<u>Medium</u> sized arteries, distributing arteries exhibit smooth <u>muscles</u> 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)

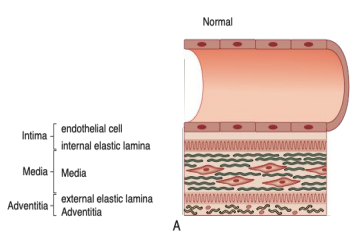
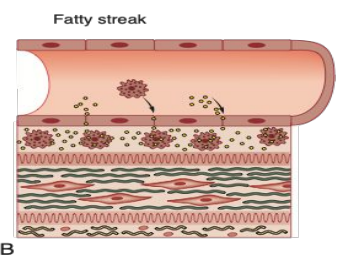

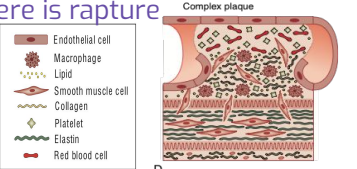


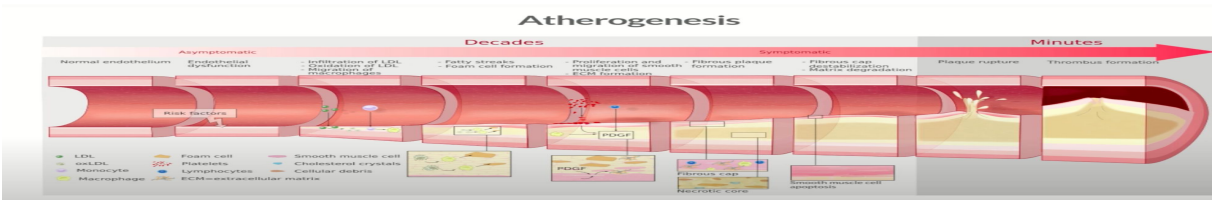
Atherosclerosis

Risk Factors of Atherosclerosis

Non Modifiable	Modifiable – Major Factors	Other Risk Factors (very little, or have effect on the modifiable risk factor)
<ul style="list-style-type: none"> ❖ Age (males \geq 45 years, females \geq 55 years (postmenopause)) ❖ Male gender ❖ Menopause (Because in perimenopause estrogen acts as a protective factor) ❖ Familial predisposition ❖ Genetic it's non-modifiable until we have genetherapy. 	<ul style="list-style-type: none"> ❖ Smoking ❖ HTN ❖ DM ❖ Dyslipidemia 	<ul style="list-style-type: none"> ❖ 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	<ul style="list-style-type: none"> - Chemical injury (Smoking, Hypercholesterolemia) - Physical injury (stress with blood pressure) - 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	<ul style="list-style-type: none"> - 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. causing chain reaction - Cytokines attract more leukocytes and smooth muscle cells. 	
Simple Plaque	<ul style="list-style-type: none"> - Smooth muscle cells exit the media. into the intima space - Proliferate, take on the characteristics of fibroblasts and produce collagen, raising the atheroma. 	
Complex Plaque	<ul style="list-style-type: none"> - Proliferation forms an endothelial cap, which may rupture, ensuing further endothelial injury. - (causing inclusions due to rough surface) this results in thrombosis and distal embolization. 	<p>there is rupture</p> 



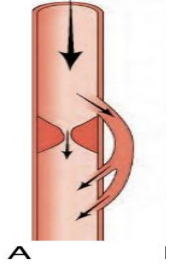


Atherosclerosis cont..:

Mechanisms of injury in atherosclerotic disease

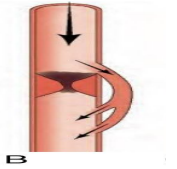
(A) Critical stenosis compensated by collateral vessels

- ❖ 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.



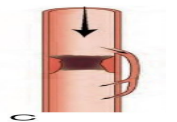
(B) Acute thrombosis of a critical stenosis

- ❖ 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



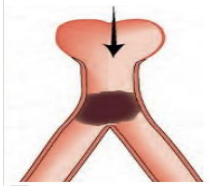
(D) Atheroembolism from ruptured plaque

- ❖ "cholesterol" emboli.



(E) Thromboembolism

- ❖ "thrombus" emboli,
- ❖ Atrial fibrillations are the most common cause
- ❖ Severe ischemia because of lack of collateral supply (Acute ischemia due to the absence of collaterals, usually caused by heart-related conditions).
- ❖ 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





Peripheral Arterial Disease (PAD)



Chronic	Acute
<ul style="list-style-type: none"> • <u>Slow gradual</u> luminal stenosis secondary to plaque • <u>Collateral development</u> compensate • Symptoms proportional to disease burden For example: <ul style="list-style-type: none"> - One area of stenosis → asymptomatic - Multiple → claudication - More → peripheral ischemia • Exertional symptoms appear first <i>الشخص عرض و طلب: لما يمشي الطلَب (الدم) يزيد فالعرض يزيد [الأعراض- للمرض]</i> ○ Leads to: <ul style="list-style-type: none"> - Intermittent Claudication (IC) - Critical limb ischemia (CLI) - 10% of Diabetic foot (DF) 	<ul style="list-style-type: none"> • Emergency PADs • <u>Sudden</u> occlusion in the <u>absence</u> of adequate <u>collaterals</u>. • Caused by <ul style="list-style-type: none"> - Embolism - Thrombosis - Injury (either the vessels themselves are injured or something compressing the vessels, leading to acute limb ischemia) ○ Leads to: <ul style="list-style-type: none"> - Acute Limb ischemia (ALI)



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.

Epidemiology



- 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

-Claudication pain is a muscular pain (affecting muscle groups) (no numbness or burning sensations)

-Not present at rest (Because at rest there is enough blood to meet the tissue demand)

-The pain comes on after walking a particular distance, which is known as the **claudication distance**¹. e.g. a patient says after walking 500m my muscle starts to hurt.

-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.

-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".

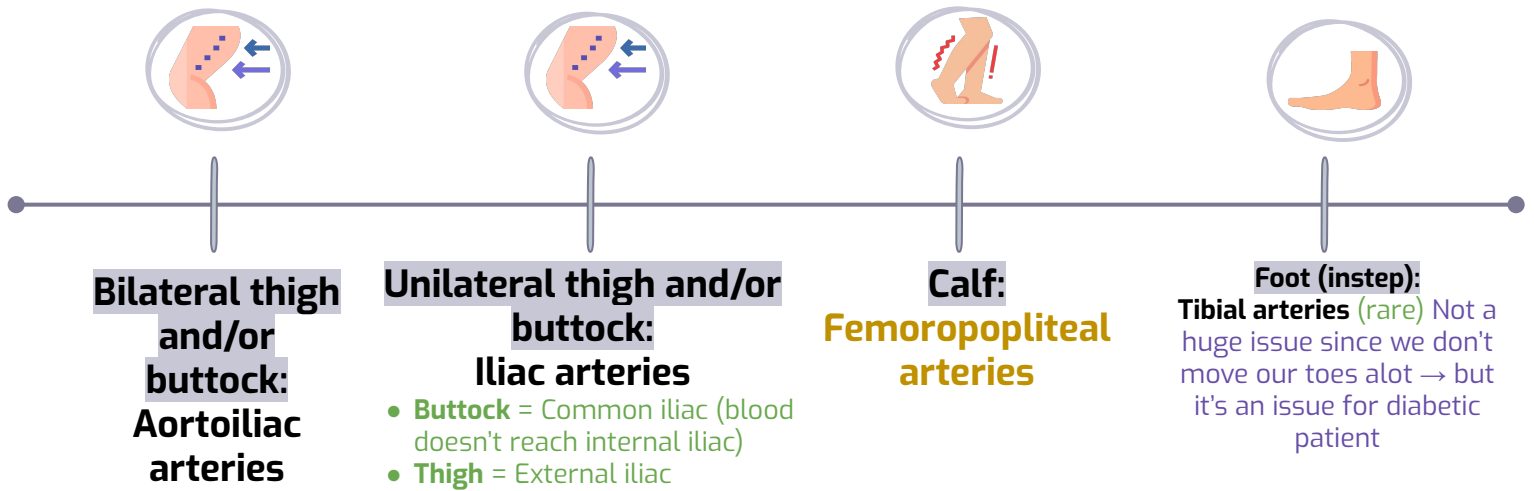
- **Other symptoms:** Due to low blood supply
 - Impotence (leriche syndrome) due to internal iliac occlusion - aortoiliac occlusion.
 - Weakness / decreased mobility
 - Skin changes (dry and thin)
 - Toe nail changes
 - Muscle wasting (Muscle atrophy affecting the legs usually, caused by a disease of iliac arteries)



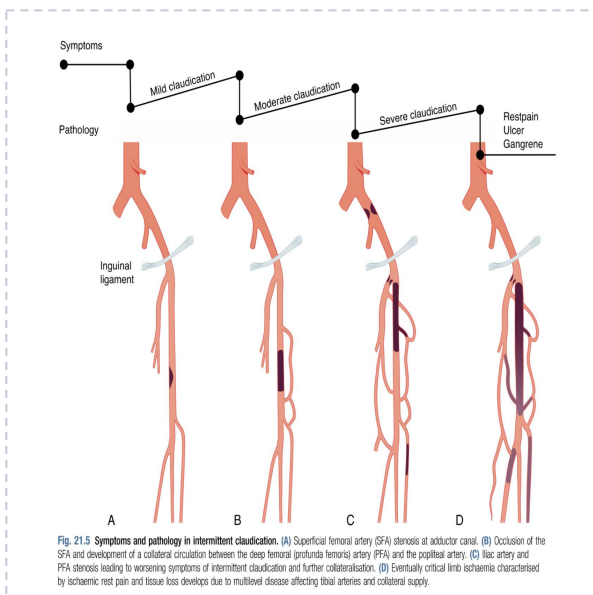


Claudication Site, (IC) cont.:

- 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.



Critical Limb Ischemia (CLI): Complete occlusion



Sequence of events

- Superficial femoral artery (SFA) stenosis "start of claudications" Usually asymptomatic
- Complete occlusion of SFA, collaterals developed from deep femoral artery (PFA) develops symptoms when walking → goes to the mosque.
- Stenosis of PFA & common iliac, worsening symptoms. Patients usually in the house.
- Critical Limb Ischemia (CLI) - **pain at rest, gangrene, ulcer.**

- العرض أقل من الطلب = يسبب ألم.
- العرض أكثر من الطلب = يسبب جرح فا يزيد الألم

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





Critical Limb Ischemia (CLI) cont.: Complete occlusion



★ **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. Patient can't sleep on bed because of the discomfort.
- 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

Physical Examination	
Skin 	<ul style="list-style-type: none"> ● Skin is thin and dry. ● Reduced temperature. ● Pallor, particularly on elevation (If the leg been affected the foot might turn pale, white when it's raised called elevation pallor): <ul style="list-style-type: none"> ○ 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).
Vein	<ul style="list-style-type: none"> ● 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.
Nails and Muscles	<ul style="list-style-type: none"> ● Brittle Nails ● Muscle wasting <div style="text-align: right;">  </div>





Physical Examinations

<p>Pulses</p>	<ul style="list-style-type: none"> All patients must have their pulse status recorded <ul style="list-style-type: none"> This includes: carotid, subclavian, brachial, radial, ulnar, femoral, popliteal, posterior tibial and dorsalis pedis (it's important for the treatment and the diagnosis) <ul style="list-style-type: none"> (examine all pulses) The pulses are recorded as normal, weak or absent (comment on the absence or the presence of the pulses only- weakness isn't IMP) The presence of a thrill and/or bruit denotes turbulent flow Ankle/brachial pressure index should be recorded (refer to vascular investigation lecture) 	
<p>Tissue loss in (CLI)</p>	<p>Arterial Ulcers:</p> <ul style="list-style-type: none"> Often located on toes or foot Pale and with necrotic floor Irregular margins Painful Surrounding ischemic features "Pinkish but non erythematous" "مافيه شعر" 	



Ulcer Examination:



Inspection

- Site** (location)
- Number**
- Size**
- Shape** (triangle, circular, oval, or irregular)
- 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", such as bone and tendon
- Exudate** (Discharge)
- Surrounding area**
- Margin** - Line of demarcation between normal and abnormal



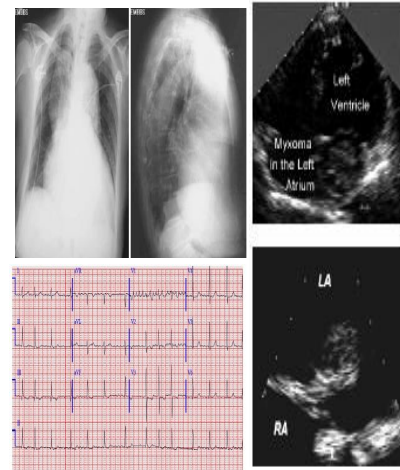
Palpation

- 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. What can I feel? Can I feel the bone or the tendon? How far is tendon or bone? mm or cm? → this indicates the severity of the condition (does it require an immediate action or not?)
- Relation** with Deeper structures
- Examination of Surrounding Area**
- Examination of Lymph Nodes**
- Examination of the pulse.**



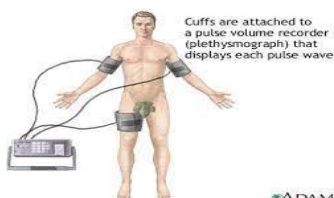
PAD Investigations

- 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 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 single 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 = Multiple stenosis or occlusion**
- **CT Angiogram & MRA** (same as duplex)
- **Invasive Vascular Investigations**

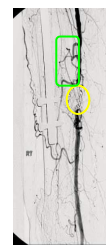
MRI
Tibial occlusion and popliteal occlusion.



CT Scan
Reconstruction



Angiogram



Green: collaterals.
Yellow: occlusion

PAD Prevention & Medical Management



Primary Prevention

- Modifiable risk factors-lifestyle changes: (BP/DM/dyslipidemia) If you have a patient with atherosclerotic 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.

Secondary Prevention - Best Medical Treatment

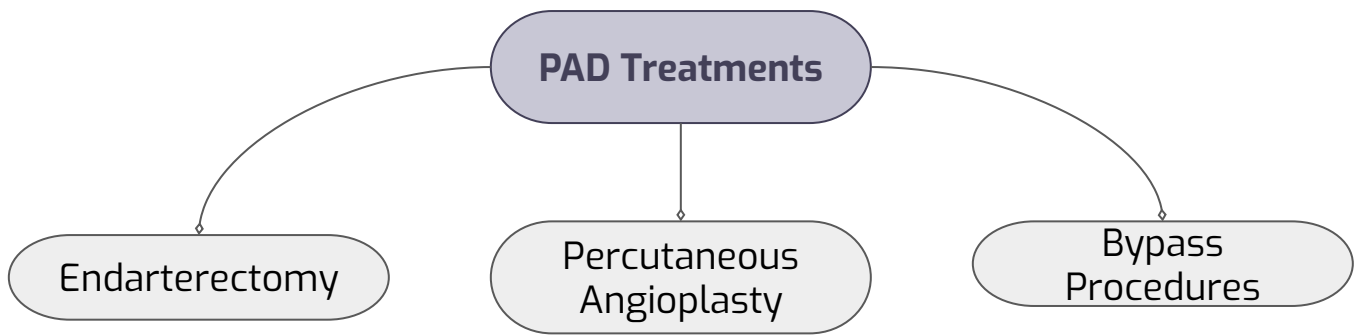
BMT	<ul style="list-style-type: none"> • All patients should be strongly urged to comply with Best Medical Therapy (BMT): <ul style="list-style-type: none"> ○ Cessation from smoking (most important) ○ Control of hypertension (ACE Inhibitors) ○ Prescription of a statin despite the absence of dyslipidemia - 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 : aspirin¹ (81 mg daily), or clopidogrel² (75mg daily) ○ Regular exercise ○ Control of obesity ○ The identification and treatment of patients with diabetes (HbA1c<7%)
Compliance	<ul style="list-style-type: none"> • 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: <ul style="list-style-type: none"> ○ 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
Walking Exercise Program	<ul style="list-style-type: none"> • You ask the patient to walk on a flat surface for 3 months, 3 days/week, 30 Minutes/day. Tell the patient 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: <ul style="list-style-type: none"> ○ Improve the collaterals ○ Train muscles to use less O₂ (anaerobic respiration)

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



PAD Treatment




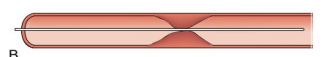
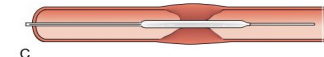
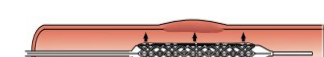

Endovascular and Surgical Interventions:

Indications for Interventions Are:	
<ul style="list-style-type: none"> Disabling for intervention (Does the patient have pain at rest or wound? yes, interven. No, BMT.) 	<ul style="list-style-type: none"> CLI
Interventions Includes:	
<ul style="list-style-type: none"> Balloon angioplasty, with or without stenting 	<ul style="list-style-type: none"> Surgery



Endovascular Intervention (Balloon Angioplasty): less

invasive and the patient can walk the next day.

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

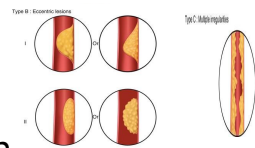


Endovascular Intervention (Balloon Angioplasty) cont.:

Endovascular Intervention

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

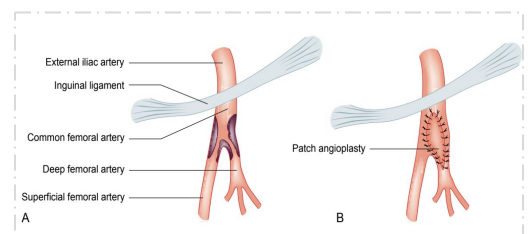
- Some plaques comes back and reclude the lumen, in such cases we need to mount a stent.
 - The benefit of stents is to keep a radial force on the vessels to prevent reclusion
- Drug-eluting (**Paclitaxel** → chemo drug which has local effect) reduce inflammatory reaction balloons and stents reduce the 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



Surgical Interventions:

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.
- منسويها بأماكن محددة very easy to access and has no muscle





Surgical Interventions cont...:

b. Bypass Grafting

Just understand the idea. You don't have to know the differences

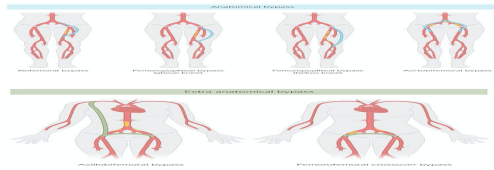
For a **bypass** operation to be successful in the long term, **three** conditions must be fulfilled:

- 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 mimics 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.

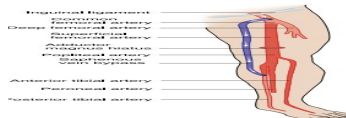
Two Main Types of Conduit Are Available "Anatomical":

Autogenous Material

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 man-made materials. Vein is also much more resistant to infection and less expensive.

Picture: Femoro-distal bypass

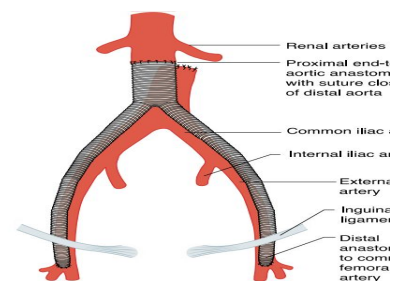


Prosthetic Material

Most commonly expanded polytetrafluoroethylene (ePTFE) or Dacron.

Prosthetics grafts are prone to infections.

Picture: Aorto-bifemoral bypass



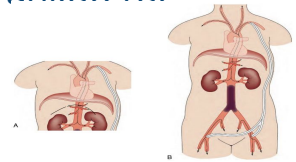


Surgical Interventions cont.:

c. 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-



Choice of Treatment.:

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



Diabetic Foot (DF-PAD)

- 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 diabetic patients are very susceptible to sepsis, ulceration and gangrene.
- Bypass grafting is not an option since there is no outflow.

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 **يصير يمشي 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



Diabetic Foot Prevention and Management



- **Diabetic foot diagnosis is similar to PAD:**
 - **History Taking.**
 - **Physical Examination.**
 - **Investigations.**

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**

1 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 treat the infection (foot care), and then 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 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	Embolism
<ul style="list-style-type: none"> ● Thrombosis in situ may arise from: <ul style="list-style-type: none"> ○ Acute plaque rupture ○ Hypovolemia ○ Increased blood coagulability (e.g., in association with sepsis, malignancy) ○ Pump failure (e.g., cardiac event) 	<ul style="list-style-type: none"> ● More than 70% of peripheral emboli are due to Atrial Fibrillations ● ER



ALI Classifications: On the basis of onset and severity:

<p>Incomplete Acute Ischemia</p>	<ul style="list-style-type: none"> ● 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.
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ALI Classifications cont.: On the basis of onset and severity:

Complete Ischemia	<ul style="list-style-type: none"> Usually due to embolus. Normally result in extensive irreversible tissue injury within <u>6 hours</u> unless the limb is revascularized. <i>Acute, the patient presents to the emergence with a pain that suddenly appeared.</i>
Irreversible Ischemia	<ul style="list-style-type: none"> Mandates early amputation or, if the patient is elderly and unfit, end-of-life care. <i>such as stroke patients and dementia patients</i>



ALI Clinical Features: **6 Ps**

2 distinctive features of acute ischemia:		
<u>Paralysis</u>	<ul style="list-style-type: none"> inability to wiggle toes or fingers 	Both indicate the 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.
<u>Paresthesia</u>	<ul style="list-style-type: none"> loss of light touch over the dorsum of the foot or hand 	
4 additional features of acute ischemia that assist the diagnosi.:		
Pain	<ul style="list-style-type: none"> May be absent in complete acute ischemia & severe pain in chronic ischemia 	
Pallor	<ul style="list-style-type: none"> feature of chronic ischemia also 	
Pulselessness	<ul style="list-style-type: none"> <u>Perishing cold</u>: (Cold foot is Unreliable, as the ischaemic limb takes on the ambient temperature). 	
Poiklothermia	<ul style="list-style-type: none"> feature of chronic ischemia also. 	





ALI Clinical Features cont..:

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)





ALI Management:

1 Complete Ischemia

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

2 Incomplete Ischemia 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 (PAD)



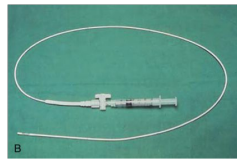
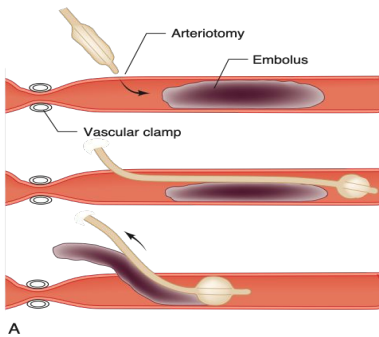
- Femoral embolus is associated with ischemia to the upper thigh.
- Acute embolic occlusion of the aortic bifurcation (saddle embolus) leads to absent femoral pulses and having white or mottled waist & legs.
 - May also present with paraplegia due to ischemia of the cauda equina.





Acute Embolism Management:

- 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.



Thrombosis in Situ (PAD)



- **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:

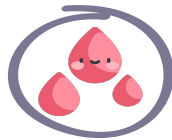




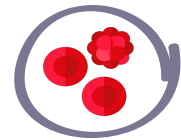
Causes of Thrombosis in Situ:



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



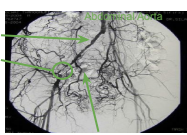
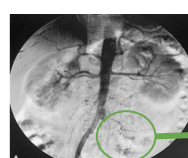
Hypovolaemia, which may be associated with widespread thrombosis



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

1	<ul style="list-style-type: none"> • Many patients can be managed medically -Especially in the early consequences. Give them anti-coagulants & follow-up
2	<ul style="list-style-type: none"> • If the limb remains threatened after one day or two of follow-up then it may be possible to clear thrombus by: <ul style="list-style-type: none"> ○ 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
3	<ul style="list-style-type: none"> • If an urgent intervention is required, the in-hospital limb loss rate may approach 30%, with an in-hospital mortality rate of 10–20%

History and Clinical Findings Differentiating the Etiology of Acute Ischemia

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
<div style="display: flex; align-items: center;"> <div style="margin-right: 10px;"> <p>External iliac occlusion</p> <p>Common iliac</p> </div>  <div style="margin-left: 10px;"> <p>(Acute Thrombosis) lots of collaterals</p> </div> </div> <p style="text-align: center; margin-top: 5px;">Internal iliac</p>	<div style="display: flex; align-items: center;">  <div style="margin-left: 10px;"> <p>Common iliac occlusion</p> </div> <div style="margin-left: 10px;"> <p>(Acute embolus) no collaterals</p> </div> </div>



Post-Ischemic Syndrome

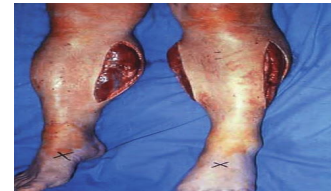
Compartment Syndrome (Local):

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



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 stunning¹
 - 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	<ul style="list-style-type: none"> • Symptoms last for < 24 hours • E.g: "Patients had a slurred speech for half a day, then it got back to normal." 	<ul style="list-style-type: none"> • 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"



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



- 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

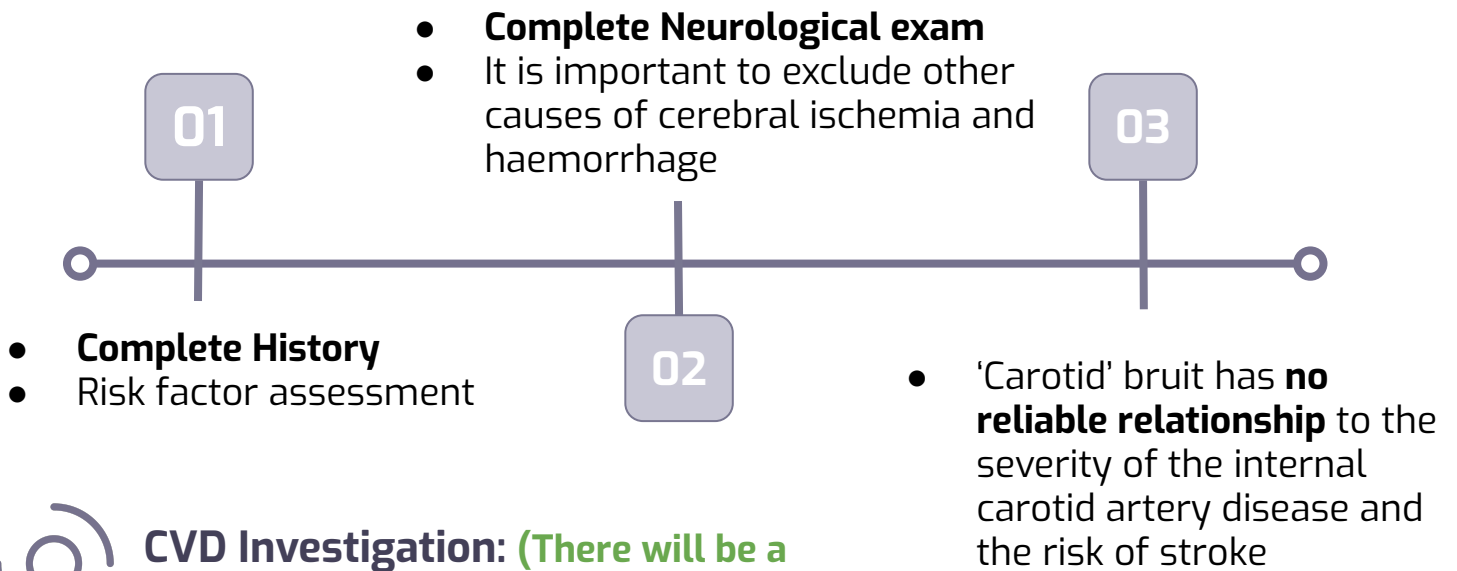
- Emboli entering the ophthalmic artery leads to amaurosis fugax or permanent monocular blindness on the same side (**ipsilateral**)

- If they enter the middle cerebral artery they may cause **hemiparesis and hemisensory** loss on the **opposite side (contralateral)**





CVD Clinical Assessment:



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

<ul style="list-style-type: none"> • 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 	
<ul style="list-style-type: none"> • Magnetic resonance angiography (MRA) 	
<ul style="list-style-type: none"> • Computed tomographic angiography (CTA) 	
<ul style="list-style-type: none"> • 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:

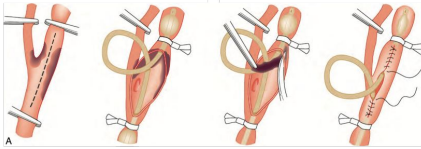
- **Asymptomatic patients are treated with BMT (Best Medical Therapy)**
 - Because, the risk of developing TIA/stroke are low (< 10% at 5 years)
 - The **Relative Risk Reduction (RRR)** is 50%, the **Absolute Risk Reduction (ARR)** would be only 1% per year
 - The number needed to prevent one TIA or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



Carotid Endarterectomy (CEA) & Carotid Artery Stenting (CAS)



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



CAS:



- The role of (CAS) remains controversial -Not preferred
- **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.



Quiz!



Explanation

Q1: A 53-year-old Asian woman comes to the physician because of a 2-month history of **severe pain in her right leg while walking**. She used to be able to walk a half-mile (800 m) to the grocery store but has been **unable to walk 200 meters without stopping** because of the pain over the past month. She can continue to walk after a break of around 5 minutes. She has hypertension, atrial fibrillation, and type 2 diabetes mellitus. She has smoked one pack of cigarettes daily for the past 32 years. Current medications include metformin, enalapril, aspirin, and warfarin. Vital signs are within normal limits. Examination shows an irregularly irregular pulse. The right lower extremity is cooler than the left lower extremity. The skin over the right leg appears shiny and dry. Femoral pulses are palpated bilaterally; **pedal pulses are diminished on the right side**. Which of the following is the most appropriate next step in management?

1. Duplex ultrasonography
2. CT angiography
3. Digital subtraction angiography
4. Ankle-brachial index

Q2: A 62-year-old man comes to the physician for a follow-up examination. For the past year, he has had **increasing calf cramping** in both legs when walking, especially on an incline. He has hypertension. Since the last visit 6 months ago, **he has been exercising on a treadmill 4 times a week**; he walks until the pain starts and then continues after a short break. He has a history of hypertension controlled with enalapril. He smoked two packs of cigarettes daily for 35 years but **quit 5 months ago**. His temperature is 37°C (98.6°F), pulse is 84/min, and blood pressure is 132/78 mm Hg. Cardiopulmonary examination shows no abnormalities. The calves and feet are pale. Femoral pulses can be palpated bilaterally; pedal pulses are absent. His ankle-brachial index is 0.6. Which of the following is the most appropriate next step in management?

1. Rest and orthotic braces
2. Operative vascular reconstruction (e.g. bypass surgery)
3. clopidogrel and simvastatin
4. Percutaneous transluminal angioplasty and stenting

Q3: A 63-year-old woman comes to the physician with a 3-month history of progressively worsening right **calf pain**. She reports that the pain occurs **after walking** for about 10 minutes and resolves when she rests. She has **hypertension** and **hyperlipidemia**. She takes lisinopril and simvastatin daily. She has **smoked two packs of cigarettes daily for 34 years**. Her pulse is 78/min and blood pressure is 142/96 mm Hg. Femoral and popliteal pulses are 2+ bilaterally. Left pedal pulses are 1+; right pedal pulses are absent. The remainder of the examination shows no abnormalities. **Ankle-brachial index (ABI) is 0.65 in the right leg and 0.9 in the left leg**. This patient is at greatest risk for developing which of the following?

1. Critical limb ischemia requiring amputation
2. Deep vein thrombosis
3. Acute myocardial infarction
4. Lower extremities lymphedema

⌂ (E) ⌂ (Z) ⌂ (L)





Quiz!

Q4: A 49-year-old man comes to the physician because of increasing difficulty achieving an erection for 6 months. During this period, he has had to reduce his hours as a construction worker because of pain in his lower back and thighs and a progressive lower limb weakness when walking for longer distances. His pain resolves after resting for a few minutes, but it recurs when he returns to work. He also reports that his pain is improved by standing still. His last visit to a physician was 25 years ago. He is sexually active with 4 female partners and uses condoms inconsistently. His father has coronary artery disease and his mother died of a ruptured intracranial aneurysm at the age of 53 years. He has recently taken sildenafil, given to him by a friend, with no improvement in his symptoms. His only other medication is ibuprofen as needed for back pain. He has smoked one pack of cigarettes daily for 35 years. He is 172.5 cm (5 ft 8 in) tall and weighs 102 kg (225 lb); BMI is 34 kg/m². His temperature is 36.9°C (98.4°F), pulse is 76/min, and blood pressure is 169/98 mm Hg. A complete blood count and serum concentrations of electrolytes, urea nitrogen, and creatinine are within the reference ranges. His hemoglobin A1c is 6.2%. Which of the following is the most likely finding on physical examination?

1. Decreased bilateral femoral pulse
2. Internuclear ophthalmoplegia
3. papular rash of the palms
4. Jugular venous distention

Q5: A 25-year-old woman comes to the physician because of intermittent painful double vision for the past 3 days. Her symptoms occur only when looking sideways. She has myopia and has been wearing corrective lenses for 10 years. Ten days ago, she lost her balance and fell off her bike, for which she went to a hospital. A CT scan of the head at that time showed no abnormalities and she was released without further treatment. Her only medication is an oral contraceptive. Vital signs are within normal limits. The pupils are equal and reactive to light. Her best corrected visual acuity is 20/40 in each eye. She has an adduction deficit in the right eye and horizontal nystagmus in the left eye when looking left; she has an adduction deficit in the left eye and horizontal nystagmus in the right eye when looking right. Convergence testing shows no abnormalities. Fundoscopy shows bilateral disc hyperemia. Which of the following is the most likely cause of this patient's findings?

1. Caudal displacement of the cerebellar vermis
2. demyelination of the medial longitudinal fasciculus
3. Antibodies against acetylcholine receptors
4. A compressive tumor in the pons

Q6: A 65-year-old man comes to his primary care physician with a 6-month history of **bilateral calf pain**. The pain usually occurs **after walking his dog a few blocks** and is more severe on the right side. He has coronary artery disease, essential hypertension, and type 2 diabetes mellitus. He has smoked two packs of cigarettes daily for 43 years and drinks two alcoholic beverages a day. Current medications include metformin, lisinopril, and aspirin. He is 183 cm (5 ft 11 in) tall and weighs 113 kg (250 lb); BMI is 35 kg/m². His temperature is 37.0°C (98.6°F), pulse is 84/min, and blood pressure is 129/72 mm Hg. Cardiac examination shows a gallop without murmurs. The legs have shiny skin with reduced hair below the knee. Femoral and popliteal pulses are palpable bilaterally. Dorsal pedal pulses are 1+ on the left and absent on the right. Ankle-brachial index (ABI) is performed in the office. **ABI is 0.5 in the right leg, and 0.6 in the left leg.** Which of the following is the most appropriate initial step in management?

1. structured exercise therapy
2. Propranolol therapy
3. Vascular bypass surgery
4. cilostazol therapy

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Quiz!

#439

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



القادة

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في الدوسري

رزان المهنا

وعد أبو نخاع

نوف الضلعان

الأعضاء

ميمي مخلص سقيان

شكرًا جزيلاً ل 439 و ل 438 على
عملهم الرائع!

حسبي الله لا إله إلا هو عليه توكلت وهو رب العرش العظيم.
اللهم إني أستودعك ما قرأت وما حفظت وما تعلمت فرده لي عند حاجتي إليه إنك على كل شيء قدير.



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