Peripheral Vascular Disease

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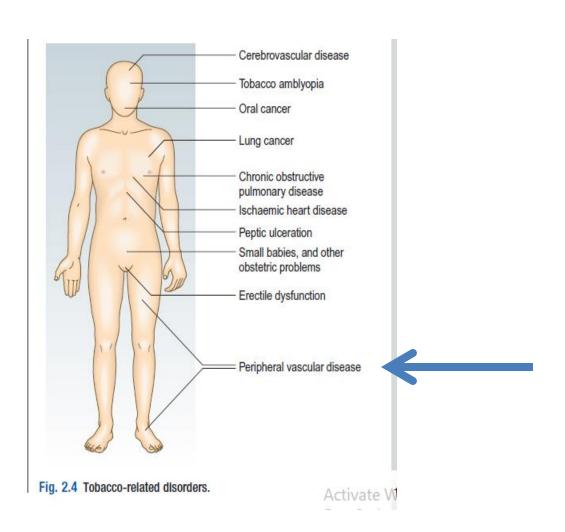
Mutah University - Introductory Course 2023



Greetings

- I request the following in my lectures:
- 1. Silence.
- 2. No use of mobile phones.
- 3. No exit-reentry during lectures.
- 4. You can interrupt me for questions.

What we mean by Peripheral Vascular Disease?



Definition

- Peripheral Vascular disease is slow blood circulation disorder that leads to blockage in blood vessels.
- The blood is unable to circulate properly due to clots in the arteries & veins.

 Claudication, arteriosclerosis obliterans, intermittent claudication, arterial insufficiency of legs are other names to PVD.



Arterial Disease

Venous Disease

- 1- Embolism
- 2- Trauma
- 3- Atherosclerosis

1- Deep Venous Thrombosis

تجلط دموي(DVT)

2- Superficial

Thrombophlebitis

الدوالي 3- Varicose Vein

Risk Factors for PVD

- 1- Hypertension
- 2- DM
- 3- Heart/Kidney Disease
- 4- Obesity
- 5- Smoking
- 6- Unhealthy Lifestyle e.g. Lack of exercise



*Modifying risk factors dramatically improves outcomes

Symptoms

PVD Symptoms depend on the affected artery.

The lack of blood flow in the artery mostly causes **pain** in leg calves, thigh & hips.

SOCRATES

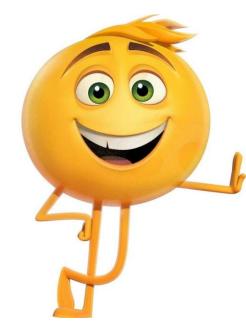


Symptoms

- The pain generally starts, with exercise or regressive physical movements.
- Sometimes pain also occurs when you are walking or climbing a stairs



So, Chief complaint for PVD is Mostly?



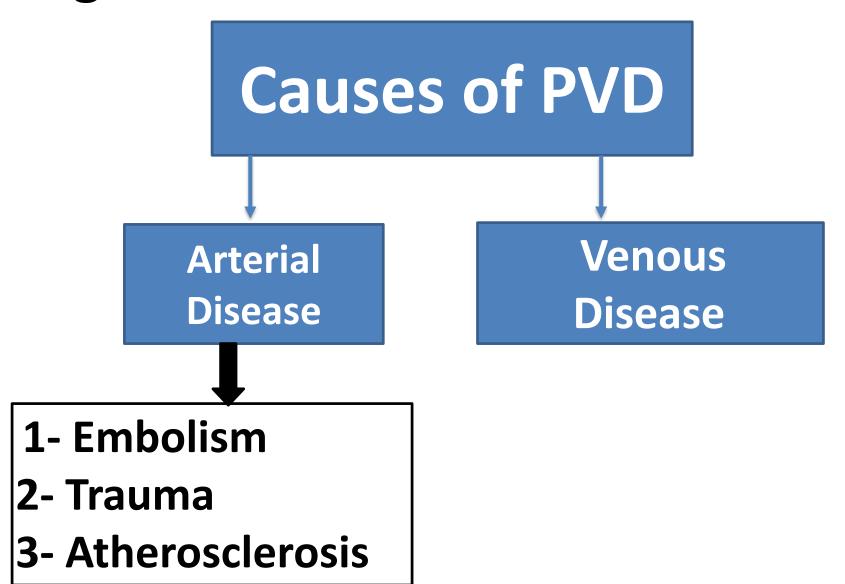
Diagnosis

- Diagnosis may be done for measuring pusles in your legs & feets, blood pressure readings in your arm & leg.
- ☐ Tests could be:
- MRI
- X-Ray
- CT –Scan
- Angiography

Treatment

- Treatment could be:
- Lifestyle changes e.g jogging, losing weight & Balanced diet.
 - **Medication-** To reduce pain symptoms & increase blood flow.
- Surgical procedure

Again.....



PERIPHERAL ARTERIAL SYSTEM

PERIPHERAL ARTERIAL SYSTEM

See Figures 6.9 and 6.35.

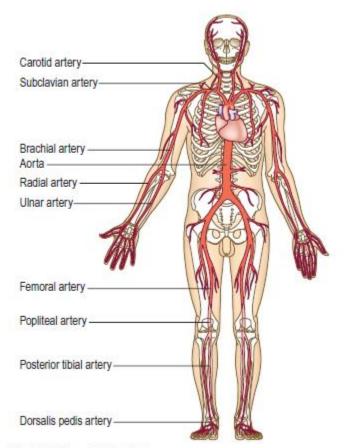


Fig. 6.35 The arterial system.

*Pulses



Peripheral Arterial Disease (PAD)

- Approximately 20% of people aged >60 years in developed countries have PAD but only a quarter of these are symptomatic. The underlying pathology is usually atherosclerosis (hardening of the arteries) affecting large and medium-sized vessels.
- PAD, even if asymptomatic, is a powerful marker for premature vascular death.

 The first manifestation of PAD may be a life- or limbthreatening complication, e.g. stroke, acute limb ischaemia or ruptured AAA.

Peripheral Arterial Disease (PAD)

- PAD affects the legs <u>eight times</u> more commonly than the arms for the following reasons:
- 1- The arterial supply to the legs is less well developed in relation to the muscle mass
- 2- The lower limb is more frequently affected by atherosclerosis.

There are four stages of lower limb lack of blood supply (ischaemia)

ĭ	6.33 Classification of lower limb ischaemia	
I	Asymptomatic	
II	Intermittent claudication	
Ш	Night/rest pain	
IV	Tissue loss (ulceration/gangrene)	

Asymptomatic ischaemia

- Ischemia: lack of blood supply
- Haemodynamically significant lower limb ischaemia is defined as an ankle to brachial pressure index (ABPI)
 <0.9 at rest.
- Most of these patients are asymptomatic, either because they choose not to walk very far, or because their exercise tolerance is limited by other comorbidity.
- Although asymptomatic, these patients are at high risk of 'vascular' complications and should be assessed and treated medically as if they have intermittent claudication.

Intermittent claudication

- Intermittent claudication is pain felt in the legs on walking due to arterial insufficiency and is the most common symptom of PAD.
- The pain typically occurs in the calf secondary to femoro-popliteal disease but may be felt in the thigh and/or buttock in proximal (aorto-iliac) obstruction.

Intermittent claudication

 Patients describe tightness or 'cramp-like' pain which develops after a relatively constant distance; the distance is often shorter if walking uphill, in the cold and after meals.

 The pain disappears completely within a few minutes of rest but recurs on walking. The 'claudication distance' is how far patients say they can walk before the pain stops them from walking.

Intermittent claudication

- There are two other types of claudication
- Neurogenic claudication is due to neurological and musculoskeletal disorders of the lumbar spine
- <u>Venous claudication</u> is due to venous outflow obstruction from the leg, following extensive DVT.
- Neurogenic and venous claudication are much less common than arterial claudication, and can be distinguished on history and examination

Night/rest pain

- The patient goes to bed, falls asleep, but is then woken 1–2 hours later with severe pain in the foot, usually in the instep.
- The pain is due to poor perfusion resulting from the loss of the beneficial effects of gravity on lying down and the reduction in heart rate, BP and cardiac output that occurs when sleeping.

Night/rest pain

 Patients often obtain relief by hanging the leg out of bed or by getting up and walking around. However, on return to bed, the pain recurs and patients often choose to sleep in a chair. This leads to dependent oedema, increased interstitial tissue pressure, a further reduction in tissue perfusion and ultimately a worsening of the pain.

• Rest (night) pain indicates severe, multilevel lower limb PAD and is a 'red flag' symptom that mandates urgent referral to a vascular surgeon as failure to revascularise the leg usually leads to the development of critical limb ischaemia with tissue loss (gangrene, ulceration) and amputation.

- In diabetic patients it may be difficult to differentiate between rest pain and diabetic neuropathy as both may be worse at night.
- Neuropathic pain is not usually confined to the foot, is associated with burning and tingling, is not relieved by dependency and is associated with dysaesthesia (pain or uncomfortable sensations sometimes described as burning, tingling or numbness).
- Many patients cannot even bear the pressure of bedclothes on their feet

IMPORTANT!

6.34 The clinical features of arterial, neurogenic and venous claudication					
	Arterial	Neurogenic	Venous		
Pathology	Stenosis or occlusion of major lower limb arteries	Lumbar nerve root or cauda equina compression (spinal stenosis)	Obstruction to the venous outflow of the leg due to lilofemoral venous occlusion		
Site of pain	Muscles, usually the calf but may involve thigh and buttocks	III defined. Whole leg. May be associated with numbness and tingling	Whole leg. 'Bursting' in nature		
Laterality	Unilateral if femoropopiiteal, and bilateral if aortolliac disease	Often bilateral	Nearly always unliateral		
Onset	Gradual after walking the 'claudication distance'	Often immediate on walking or standing up	Gradual, from the moment walking starts		
Relieving features	On stopping walking, the pain disappears completely in 1–2 minutes	Bending forwards and stopping walking. May sit down for full relief	Leg elevation		
Colour	Normal or pale	Normal	Cyanosed. Often visible varicose veins		
Temperature	Normal or cool	Normal	Normal or Increased		
Oedema	Absent	Absent	Always present		
Pulses	Reduced or absent	Normal	Present but may be difficult to feel owing to oedema		
Straight-leg raising	Normal	May be limited	Normal		

Tissue loss (ulceration and/or gangrene)

• In patients with *severe lower limb PAD*, even trivial injuries to the feet fail to heal. This allows bacteria to enter, leading to gangrene and/or ulceration. This usually progresses rapidly and, without re-vascularisation, often leads quickly to amputation and/or death.

Signs of lower limb PAD

- Ischaemic signs include:
- 1- absence of hair
- 2- thin skin
- 3- brittle nails.

- The presence of foot pulses does not completely exclude significant lower limb PAD but they are almost always diminished or absent.
- If the history is convincing but pulses are felt, ask the patient to walk until the claudication pain stops him and then recheck the pulses; if they have disappeared then PAD is very likely.

Signs of lower limb PAD



6.35 Signs of acute limb ischaemia

Soft signs

- Pulseless
- Pallor

Perishing cold

Hard signs (indicating a threatened limb)

- Paraesthesia
- Paralysis

Pain on squeezing muscle





 The classical features of acute limb ischaemia are the 'six Ps' (Box 6.35). Paralysis (inability to wiggle the toes/ fingers) and paraesthesia (loss of light touch sensation over the forefoot/dorsum of the hand) are the most important and indicate severe ischaemia affecting nerve function.

 Muscle tenderness is a grave sign indicating impending muscle infarction. A limb with these features will usually become irreversibly damaged unless the circulation is restored within a few hours.

- The commonest causes of acute limb ischaemia are:
- Thromboembolism: usually from the left atrium in association with atrial fibrillation

Thrombosis in situ: thrombotic occlusion of an already narrowed atherosclerotic arterial segmen

Acute limb ischaemia: embolus (Arterial) VS thrombosis(Venous)

	Embolus	Thrombosis
Onset and severity	Acute (seconds or minutes), ischaemia profound (no pre-existing collaterals)	Insidious (hours or days), ischaemia less severe (pre-existing collaterals)
Embolic source	Present (usually atrial fibrillation)	Absent
Previous claudication	Absent	Present
Pulses in contralateral leg	Present	Absent
Diagnosis	Clinical	Angiography
Treatment	Embolectomy and anticoagulation	Medical, bypass surgery, thrombolysis

 Patients with critical limb ischaemia (rest pain, tissue loss) typically have an ankle BP <50 mmHg and a positive Buerger's test.



 Acute arterial occlusion produces intense spasm in the arterial tree distal to the blockage. The limb appears 'marble white'. Over a few hours, the spasm relaxes and the skin microcirculation fills with deoxygenated blood, leading to light blue or purple mottling, which has a fine reticular pattern and blanches on pressure.

 As ischaemia progresses, blood coalesces in the skin, producing a coarser pattern of mottling which is dark purple, almost black, and does not blanch. Finally, large patches of fixed staining lead to blistering and liquefaction.



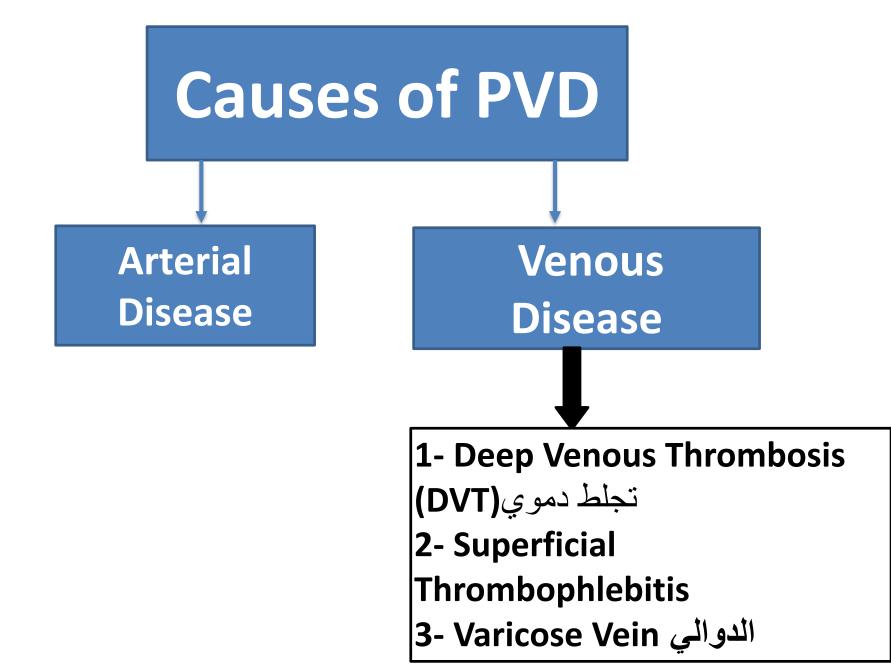
- As ischaemia progresses, blood coalesces in the skin, producing a coarser pattern of mottling which is dark purple, almost black, and does not blanch. Finally, large patches of fixed staining lead to blistering and liquefaction.
- Fixed mottling of an anaesthetic, paralysed limb, with muscle rigidity and turgor, indicates irreversible ischaemia; amputation or end-of-life care is the only option

Compartment syndrome

 Compartment syndrome occurs where there is increased pressure within the fascial compartments of the limb, most commonly the calf, which compromises perfusion and viability of muscle and nerves.

Compartment syndrome

- The two commonest causes are lower trauma, e.g. fractured tibia, and reperfusion following treatment of acute lower limb ischaemia.
 Failure to recognise and treat compartment syndrome may require limb amputation.
- The key symptom is severe pain often unrelieved by opioids and exacerbated by active or passive movement. Peripheral pulses are usually present.

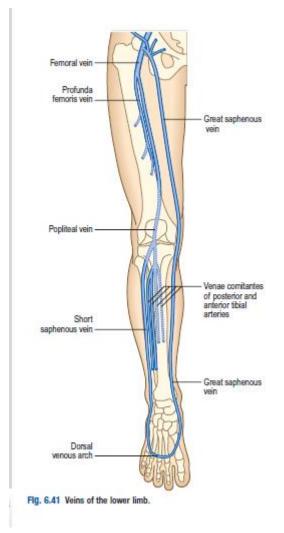


Venous Disease

 Its due to Obstruction in the venous outflow of the leg due to iliofemoral venous occlusion e.g. Thrombosis

Venous Disease

- Backward flow (reflux) is prevented by valves which divide the long column of blood from the foot to the right atrium into a series of short low-pressure segments.
- As a result, the 'ambulatory venous pressure' in the feet in health is usually <20 mmHg. The great majority of lower limb venous symptoms and signs are due to failure of the muscle pump and/or valves and the resulting 'ambulatory venous hypertension'.
- Deep veins follow the course of the main arteries; are often paired; and may be affected by primary or postthrombotic (following DVT) valvular insufficiency



- The great (long) saphenous vein passes anterior to the medial malleolus at the ankle, up the medial aspect of the calf to behind the knee, then up the medial aspect of the thigh to join the common femoral vein in the groin at the saphenofemoral junction (Fig. 6.41).
- The lesser (short) saphenous vein passes behind the lateral malleolus at the ankle and up the posterior aspect of the calf. It commonly joins the popliteal vein at the saphenopopliteal junction, which usually lies 2 cm above the posterior knee crease.

Venous Disease

 There are numerous intercommunications between the long and short saphenous, and between the deep and superficial venous (via perforating or communicating veins) systems; and the venous anatomy of the leg is highly variable.

Clinical Presentation

- Lower limb venous disease presents in four ways:
- Varicose veins
- Superficial thrombophlebitis
- DVT
- Chronic venous insufficiency and ulceration.

Pain

 Patients with uncomplicated varicose (dilated, tortuous, superficial) veins often complain of aching leg discomfort, itching and a feeling of swelling. Symptoms are aggravated by prolonged standing and are often worse towards the end of the day. Once established, DVT causes pain and tenderness in the affected part (usually the calf).

Pain

 Superficial thrombophlebitis produces a red, painful area overlying the vein involved. Varicose ulceration may be surprisingly painless; if it is painful, this may be relieved by limb elevation (but exclude coexisting arterial disease). Bandaging for a leg ulcer is contraindicated unless there is documented evidence of adequate arterial circulation. Do this by feeling the pulses or by measuring the ABPI.

Swelling

 Swelling (or oedema), or a 'feeling of swelling', may be associated with lower limb venous disease.

Discoloration

 Chronic venous insufficiency is associated with lipo-dermatosclerosis, which results from the deposition of haemosiderin (from the breakdown of extra-vasated blood) in the skin. Lipodermatosclerosis varies in colour from deep blue/black to purple or bright red and usually affects the medial aspect of the lower third of the leg, although it may be lateral if superficial reflux predominates in the lesser saphenous vein.

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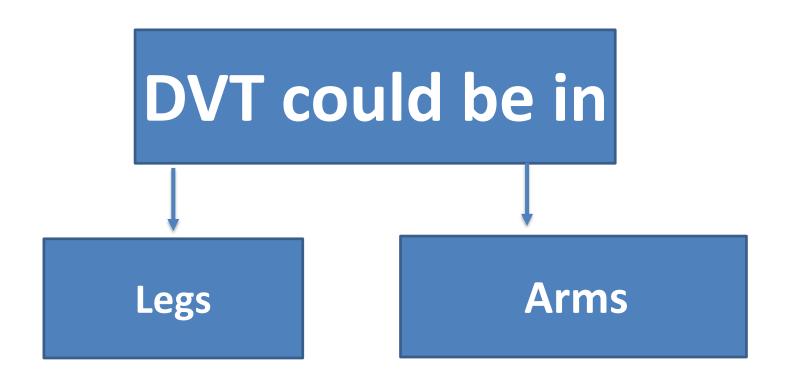
6.41 Clinical features of venous and arterial ulceration

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Clinical feature	Venous ulceration	Arterial ulceration
Age	Develops at age 40-45 but may not present for years; multiple recurrences common	First presents in over-60s
Sex	More common in women	More common in men
Past medical history	Deep vein thrombosis (DVT) or suggestive of occult DVT, i.e. leg swelling after childbirth, hip/knee replacement or long bone fracture	Peripheral arterial disease, cardio- and cerebrovascular disease
Risk factors	Thrombophilla, family history, previous DVT	Smoking, diabetes, hypercholesterolaemia and hypertension
Paln	One-third have pain (not usually severe) that Improves with elevating the leg	Severe pain, except in diabetics with neuropathy; Improves on dependency
Site	Gaiter areas; usually medial to long saphenous vein; 20% are lateral to short saphenous vein	Pressure areas (malleoli, heel, fifth metatarsal base, metatarsal heads and toes)
Margin	irregular, often with neoepithellum (appears whiter than mature skin)	Regular, Indolent, 'punched out'
Base	Often pink and granulating under green slough	Sloughy (green) or necrotic (black), with no granulation
Surrounding skin	Lipodermatoscierosis always present	No venous skin changes
Veins	Full and usually varicose	Empty with 'guttering' on elevation
Swelling (oedema)	Usually present	Absent
Temperature	Warm	Cold
Pulses	Present, but may be difficult to feel	Absent



Venous Ulceration

affects the medial aspect, is shallow; is pink (granulation tissue) or yellow/green (slough); has an irregular margin; and is always associated with other skin changes of chronic venous insufficiency (varicose eczema, lipodermatosclerosis)



Deep Venous Thrombosis (DVT) in Legs

- The clinical features of DVT depend upon its site, extent and whether it is occlusive or not
- The so-called 'classical' features of DVT relate to well-established occlusive thrombus.
- Most patients who die from pulmonary embolism have non-occlusive thrombosis and the leg is normal on clinical examination.

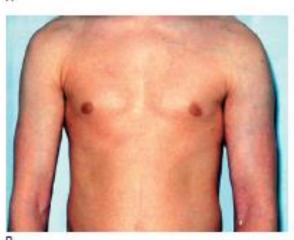
DVT

- Non-occlusive DVT poses the greatest threat of pulmonary embolism as the clot lies within a flowing stream of venous blood, is more likely to propagate and has not yet induced an inflammatory response in the vein wall to anchor it in place.
- Perform an urgent duplex Doppler ultrasound scan of the leg in any patient with a suspected DVT

DVT in Arms

- Axillary subclavian vein thrombosis can occur as a result of repetitive trauma at the thoracic outlet due to vigorous, repetitive exercise.
 Upper limb DVT may also complicate indwelling subclavian/jugular venous catheters.
- Symptoms include arm swelling and discomfort, often exacerbated by activity, especially when holding the arm overhead.





Axillary vein thrombosis due to Trauma

• The arm is swollen and the skin is cyanosed and mottled, especially when dependent. Look for superficial distended veins (acting as collaterals) in the upper arm, over the shoulder region and on the anterior chest wall

6.42 Features of deep vein thrombosis of the lower limb Clinical feature Non-occlusive Occlusive thrombus thrombus Often absent Usually present Paln Calf tendemess Often absent Usually present Swelling Absent Present Temperature Normal or slightly Increased Increased Superficial veins Distended Normal Pulmonary embolism High risk Low risk

Risk factors for deep vein thrombosis



6.43 Risk factors for deep vein thrombosis

- Recent bed rest or operations (especially to the leg, pelvis or abdomen)
- Recent travel, especially long flights
- Previous trauma to the leg, especially long-bone fractures, plaster of Paris splintage and immobilisation
- Pregnancy or features to suggest petric disease
- Malignant disease
- Previous deep vein thrombosis
- Family history of thrombosis
- Recent central venous catheterisation, injection of drugs, etc.

Case Scienario

A 40 Years old - Patient had Hip replacement 5 days ago.

- Whats your diagnosis?
- Mention other differntials
- What are the complications?



- What'``s your diagnosis? DVT
- Mention other differentials? Cellulites/ chronic venous insufficiency.
- What are the complications?
- Pulmonary embolism
- Ulcers
- Ischemia

THANK YOU

Resources

 Macleod's Clinical Examination – Graham Douglas, Fiona Nicol, Colin Robertson.