

Disorders of peripheral arteries

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Arteries

- Arterial system of lower extremities
- Arterial system of upper extremities
- Vasospastic disorders

Arterial system of lower extremities

- Peripheral arterial disease
- Acute limb ischaemia
- Aneurysms
- Arterial dissection

Atherosclerosis

- Gradual development of AS plaques in arterial wall, progressive narrowing and arterial occlusion
- accumulation of smooth muscle cells in subendothelial space
 - Intra- and extracellular lipid accumulation
 - accumulation of connective fibers (collagen) and polysaccharides

Endothelium

- layer of highly specialized cells in internal part of arterial wall
- Permeability control – cells and non-cellular particles
- Optimal flow control (smooth muscle tension regulation)
- Non-adhesive surface – prevention of adhesion and aggregation of thrombocytes
- Reparation and angiogenesis control

Endothelial cell produces:

- **vasodilation factors** (NO, EDHF, prostacyclin, adrenomedullin, natriuretic peptide)
- **vasoconstrictors** (endothelin, angiotensin II, thromboxan A₂)
- **adhesive molecules** (VCAM-1, ICAM-1, e-selectin, thrombomodulin)
- **coagulation/fibrinolytic factors** (vWf, TPA, PAI-1)
- **growth factors** (VEGF, PDGF, TGF- β)
- **cytokines** (MCP-1, IL-8)

Endothelial dysfunction

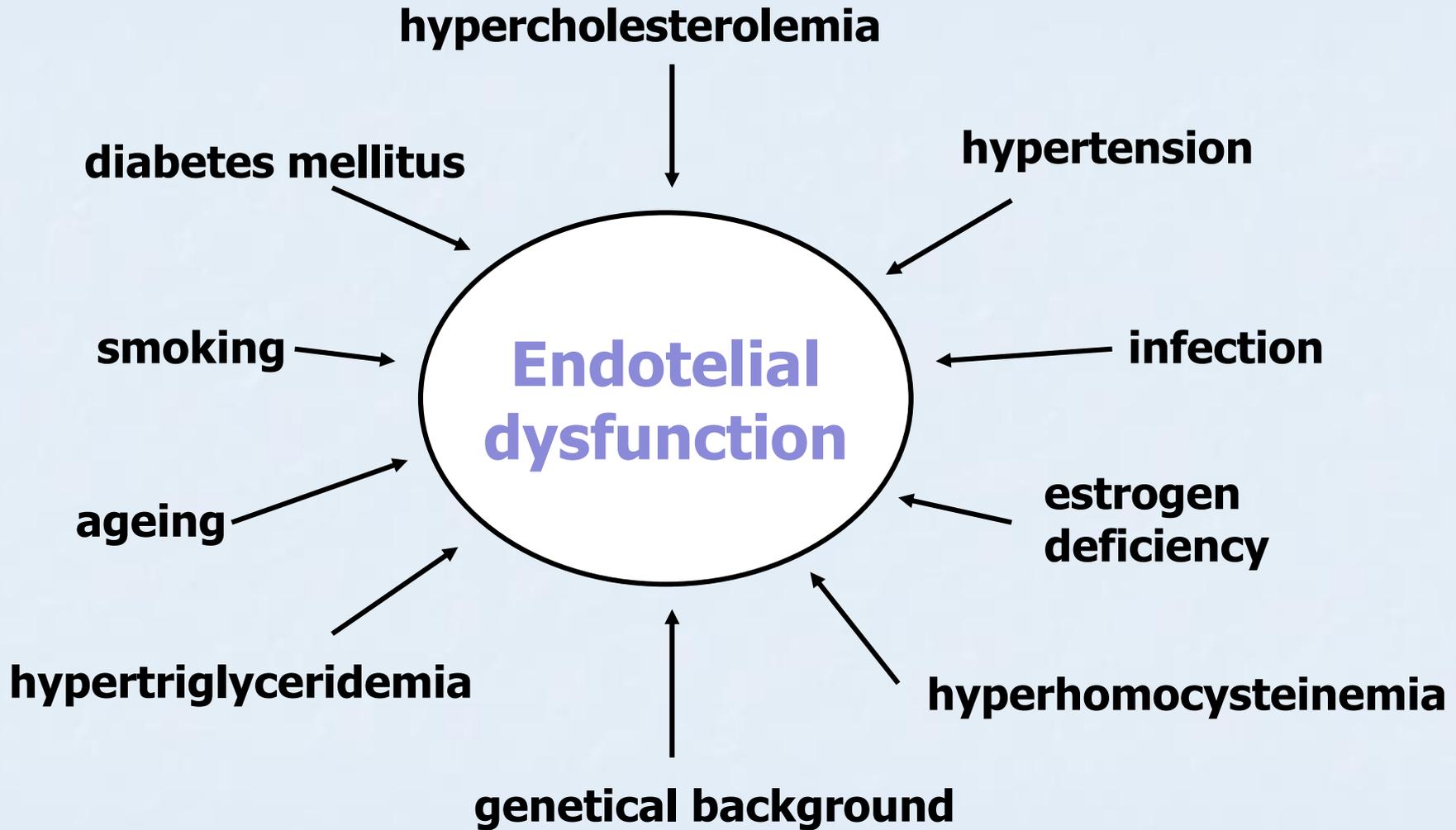
- Endothelial function impairment

Dysbalance of:

- vasoactive x hemocoagulation factors

Result:

- Pro-atherogenicity
- Vasoconstriction
- Pro-trombogenicity



Peripheral arterial disease (PAD)

- **Atherosclerosis (progressive stenoses and obliterations)**
- Trombangiitis obliterans (von Winiwarter – Buerger)
- Compressive syndroms (entrapment syndrom of a.poplitea)
- Cystical adventitial disease
- Vasculitis
- Traumatically or iatrogenically caused arterial obliterations
- Drug abusos
- Trombosis and embolization of peripheral arteries

PAD – epidemiology

Symptomatic PAD

- 1,8 % under 60 years
- 3,7 % 60 – 70 years
- 5,2 % above 70 years
- men : women ratio = 3 : 1
- Serious , progressive disorder, 60% requires revascularization, in 20 – 30% of critical limb ischemia amputation

Prognosis of the patients suffering from PAD

- Coronary heart disease
- Carotic arteries affection
- Diabetes mellitus
- Clinical stadium of PAD
- Non – stopping of smoking
- Arterial hypertension – especially if it isn't appropriately treated

Clinical presentation of PAD:

- **Family history:** AS (PAD, CHD, stroke), hypertension, DM
- **Personal history:** risk factors of AS (smoking, dyslipidemia, hypertension, diabetes mellitus), CHD, stroke
- **Clinical presentation:** claudications, rest pain

Clinical presentation of PAD:

- I.st. – asymptomatic
- II.st. – ***claudications***
 - IIa > 200m
 - IIb < 200m
- III.st. – ***rest pain***
- IV.st. – ***ulcerations/dystrophy***

Peripheral pressures measurements

- CW Doppler or photoplethysmography
- Ankle cuff (= ankle pressure)
- Toe cuff (= toe pressure)
- Absolute values x indexes against the brachial pressure

Peripheral pressures

ABI value	ischaemia
0.91 – 1.30	Normal
0.51 – 0.90	Mild to medium ischaemia
below 0.50	Severe ischemia

TBI under 0.70
= pathological

- ABI above 1.3 = mediocalcinosis
- Ankle pressure < 50mmHg or
- Toe pressure < 30mmHg = critical limb ischemia

Treadmill test

- Measurement of claudication distance
- Treadmill
- speed 3,2 – 4 km/hour, angle 7 – 12gr.
- Worsen vascular compensation = shorter claudication distance

Non-invasive examination methods

- **Duplex ultrasound** – morphological and haemodynamic information, combination with doppler's signal – flow speed measurement. Information about stenosis grade, arterial wall quality
- **CT angiography**
- **MR angiography**

Semi + Invasive examination methods

- CT angiography
- MR angiography
- **angiography** – direct entrance to the artery, imaging of arterial system using contrast agent

Indications of angiography:

- PAD stage IIb, III, IV
- Critical limb ischaemia
- Before revascularization procedure
- Before amputation
- In acute arterial occlusion

Contraindications of angiography:

- **Heart and renal failure** (x only with small amount of contrast agent)
- **Intolerability of contrast agent**

After AG: 24hours rest with inguinal compression and extended leg is necessary

Complications: allergy, renal failure, pseudoaneurysma

Treatment of PAD: general principles (from Ist st.)

- Stop smoking
- Alcohol intake lower than 30g/day
- Diet with restriction of fat
- Dyslipidemia treatment, if LDL-cholesterol $> 3,0$ mmol/l
- Hypertension and diabetes treatment
- Antiaggregation treatment – ASA, ticlopidin, clopidogrel

Treatment of PAD: (from IIth st.)

- **Exercising**
- **Vasoactive treatment:**
 - vasodilation – not used
 - haemorheological drugs – naftidrofuryl (Enelbin), prostanoids (Prostavasin), sulodexide (VesselDue F)
- **Revascularization** (PTA, stenting, rotablaters, laser systems, endarterectomy, bypasses)

Treatment of critical limb ischaemia:

- **Revascularization:**
PTA, trombolysis,
bypass, angiogenesis
stimulation (in
research)
- **Conservative
approach:**
vazodilation -
infusions, HBOT

Other causes of PAD: popliteal artery entrapment syndrome

- rare vascular disease
- Young athletes are affected
- The muscle and tendons near the knee compress the popliteal artery
- anatomic abnormalities may be seen in up to 3% of the population and are often bilateral
- Arterial compression can result in chronic vascular microtrauma, local premature arteriosclerosis, and thrombus formation.
- Acute limb-threatening thrombosis requires urgent bypass surgery. Intermittent occlusion can usually be cured with release of the popliteal artery alone or with saphenous vein bypass

Other causes of PAD: Trombangiitis obliterans (Buerger's disease)

- a progressive inflammation and thrombosis (clotting) of small and medium arteries and veins of the hands and feet.
- It is strongly associated with use of tobacco products
- the tobacco may trigger an immune response in susceptible persons
- In Europe 0.5 – 1% PAD patients, 60 – 80% Algeria, Israel, Indonesia
- men : women = 10 : 1
- Claudications in distal parts of calf, ulceration and gangrenes of fingers, toes, superficial venous flebitis
- Treatment: anti-platelets, prostanoids

Other causes of PAD: Cystic adventitial disease of popliteal artery

- young to middle-aged individuals without evidence of atherosclerosis or other systemic vascular disease
- predilection in the popliteal region in ~85% of cases
- collection of mucinous material (mucous cysts) within adventitial wall of the affected vessel
- rapidly progressive calf claudication
- Treatment: surgical

Acute arterial obliteration

- Embolie (70 – 80%), arterial trombosis
- Clots from left heart: 80 – 90% (FiS, valvular failures, aneurysma), 10% from aorta or big arteries
- Acute trombosis: AS obliteration, endothelial impairment etc.
- Trombosis occurred on chronic stenosis – less dramatical presentation (collateral circulation) than in case of embolisation

Acute arterial occlusion:

„6P“ :

- **Pain** – starts suddenly
- **Paleness** – below the arterial obliteration
- **Pulselessness** – non-palpable pulses below obliteration
- **Parestezia** – hypesthesia
- **Paralysis** – muscle rigidity – interruption of energy production
- **Prostration** – vasovagal reflexes could lead to general prostration, fatigue, collaps

Aneurysms of peripheral arteries

risk of acute thrombosis/peripheral embolism

- Any branch of aorta could be affected
- Common causes include atherosclerosis, popliteal artery entrapment, and septic emboli (which cause mycotic aneurysms).
- About 70% of peripheral arterial aneurysms are popliteal aneurysms
- 20% are iliofemoral aneurysms
- Aneurysms at these locations frequently accompany abdominal aortic aneurysms, and > 50% are bilateral
- Another location: a.subclavia, mesenteric arteries
- may cause limb ischemia, distal embolism

Arterial affections of upper extremities

- Atherosclerotic arterial disease – stenosis of a.subclavia, a.brachialis – typical claudication pain in upper extremities
- Arterial compression syndromes – compression of a vascular structure
 - Thoracic outlet syndrome - refers to a group of clinical syndromes caused by congenital or acquired compression of brachial plexus or artery or vein as they pass through the thoracic inlet.
Arterial compression causes ischaemia with coolness, pallor, claudication, paraesthesia and decreased upper limb pulses (vein – thrombosis, plexus – paresthesia, numbness...)

Vasospastic disorders

- Caused by vasospasm = a reversible localized vasoconstriction of smaller arteries
- primary (no trophic changes develop) – unknown etiology, disturbance of microcirculatory regulation
- secondary – accompany another disease, systemic sclerosis, SLE, malignancies, TOS, damage by vibration..

Raynaud's disease

- Three colors – pale (spasmus), blue (cyanosis), red (hyperemia)
- One to four fingers affected (except thumb)
- dg: cold exposition test, capillaromicroscopy
- th: avoiding of smoking, avoid cold exposure, Ca channel blockers, prostaglandins, sildenafil in systemic disease

Acrocyanosis

- Permanent painless blue color of periferal tissues
- Primary
- Blue and wet skin
- Worsen in cold, better in warm weather
- Specific test don't exist

Erythromelalgia

- Very rare
- Attacks of skin microcirculatory vasodilation in feet or hands, sharp burning pain, redness and hot skin
- Primary – unknown etiology
- Secondary – myeloproliferative diseases, systemic diseases
- Th: bath in cold/icy water, aspirin, antidepressants