Atherosclerosis

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

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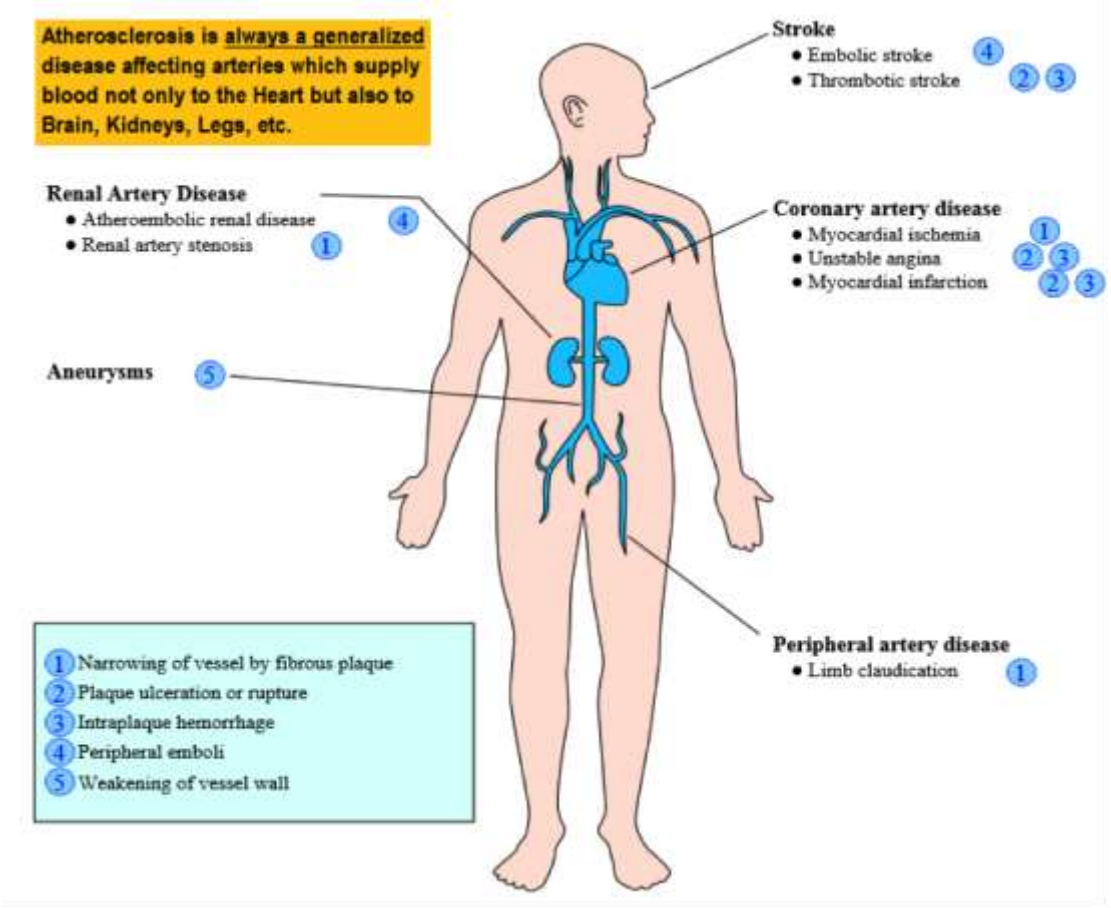
Plan of the Lecture

- Definition
- Epidemiology
- Risk factors
- Etiology
- Mechanisms
- Classification
- Clinical investigation
- Diagnosis
- Treatment
- Prognosis
- Prophylaxis
- Abbreviations
- Guidelines



juventasclinic.com/wp-content/uploads/2014/03/cardiac-regenerative-therapy-2.png

Definition

Atherosclerosis (arteriosclerotic vascular disease – ASVD, arteriosclerotic cardiovascular disease – ASCVD) is a disease affecting arterial blood vessels due to a chronic inflammatory response on cholesterol depositions within their walls by the formation of multiple atheromatous plaques and vascular lumens narrowing with manifestation in numerous clinical syndromes from sudden cardiovascular death, acute heart and brain attacks, cognitive decline and dementia, heart and renal failure up to peripheral artery disease

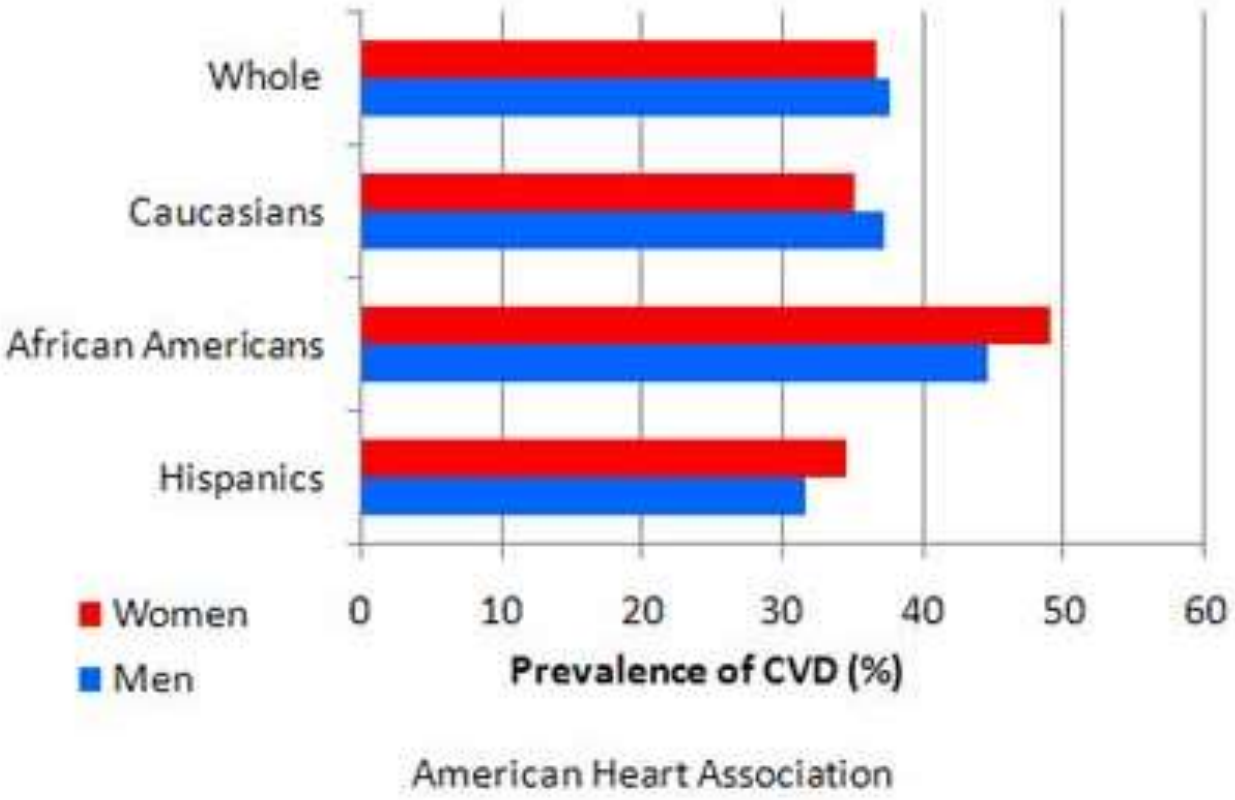
en.wikipedia.org/wiki/Atherosclerosis

Epidemiology
(Focus on Key Points)

- ASVD is a rising epidemic with severe health and economic consequences for all world regions, affecting men and women
- ASVD is the leading cause of death worldwide resulting in 17 million deaths per year
- ASVD begins early in life, and is a chronically progressive disorder that is initiated and progressively advanced by specific lifestyle conditions and genetic factors
- ASVD disease worsens exponentially with increasing age and the burden of atherosclerotic disease is expected to double by 2030 as the population ages

casinapioiv.va/content/dam/accademia/pdf/atherosclerosis.pdf

Epidemiology
(Prevalence of ASVD by Race)



indiana.edu/~k562/images/athero/atherorace.jpg

Risk Factors
(Modifiable)

- Diabetes or impaired glucose tolerance (IGT)
- Dyslipoproteinemia (unhealthy patterns of serum proteins carrying fats & cholesterol):
  - High serum concentration of low-density lipoprotein (LDL), and / or very low density lipoprotein (VLDL) particles
  - Low serum concentration of functioning high density lipoprotein (HDL)
  - An LDL:HDL ratio greater than 3:1
- Tobacco smoking
- Elevated serum C-reactive protein concentrations
- Vitamin B_6 deficiency
- Dietary iodine deficiency and hypothyroidism

en.wikipedia.org/wiki/Atherosclerosis

Risk Factors
(Nonmodifiable)

- Advanced age
- Male sex
- Having close relatives who have had some complication of atherosclerosis (e.g. coronary heart disease or stroke)
- Genetic abnormalities, e.g. familial hypercholesterolemia

en.wikipedia.org/wiki/Atherosclerosis

Risk Factors
(Lesser or Uncertain)

- Being of South Asian ethnicity
- Obesity
- Hypercoagulability
- Postmenopausal estrogen deficiency
- High intake of saturated fat
- Trans fat intake
- High carbohydrate intake
- Elevated serum levels of triglycerides
- Elevated serum levels of homocysteine
- Elevated serum levels of uric acid
- Elevated serum fibrinogen concentrations
- Elevated serum lipoprotein concentrations
- Chronic systemic inflammation
- Elevated serum insulin levels
- Short sleep duration
- *Chlamydia pneumoniae* infection
- Air pollution

en.wikipedia.org/wiki/Atherosclerosis

Risk Factors
(Total Cardiovascular Risk)

All guidelines recommend the assessment of Total Cardiovascular Risk because, in most people, ASVD is the product of a number of risk factors. Many risk assessment systems are available, including Framingham, SCORE (Systemic Coronary Risk Estimation), etc.

Very simple principles of risk SCORE ‘ assessment can be defined as follows:

1. Those with known cardiovascular disease (CVD), type 2 diabetes or type 1 diabetes with microalbuminuria, very high levels of individual risk factors, chronic kidney disease (CKD) are automatically at very high or high total cardiovascular risk and need active management of all risk factors
2. For all other people, the use of a risk estimation system such as Systemic Coronary Risk Estimation (SCORE) is recommended to estimate total cardiovascular risk (CV) risk because many people have several risk factors which, in combination, may result in unexpectedly high levels of total cardiovascular risk

escardio.org/static_file/Escardio/Guidelines/publications/DYSLIPguidelines-dyslipidemias-FT.pdf

Risk Factors
(Total Cardiovascular Risk)

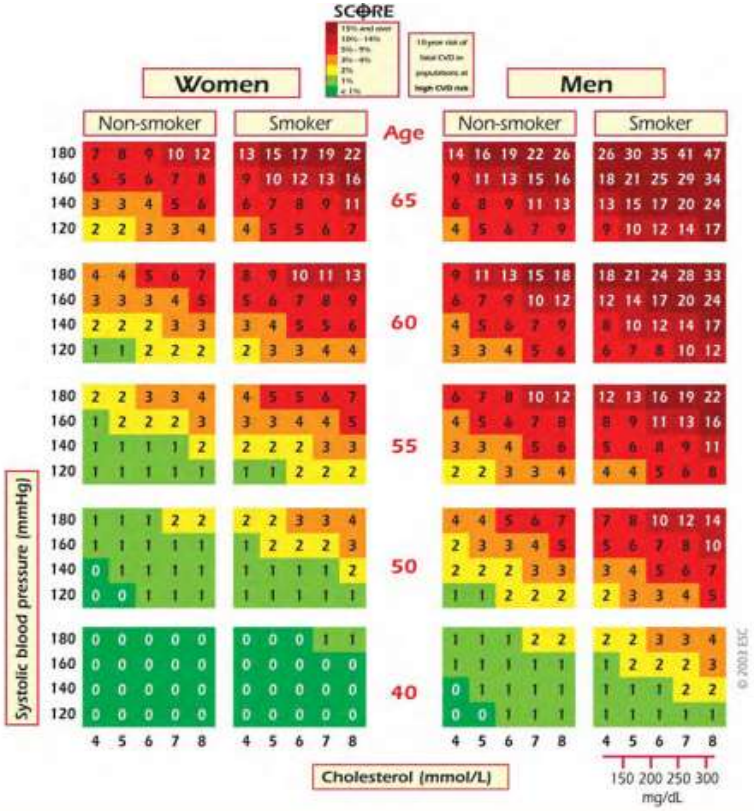


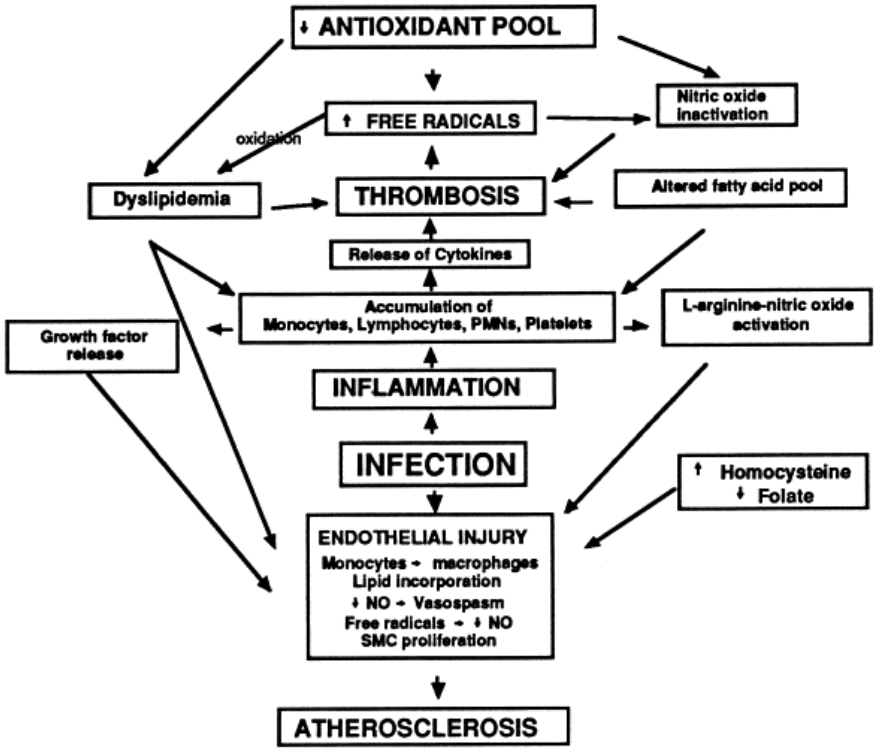
Figure 1 SCORE chart: 10 year risk of fatal cardiovascular disease (CVD) in populations at **high CVD risk** based on the following risk factors: age, gender, smoking, systolic blood pressure, and total cholesterol. To convert the risk of fatal CVD to risk of total (fatal + non-fatal) hard CVD, multiply by 3 in men and 4 in women, and slightly less in old people. Note: the SCORE chart is for use in people without overt CVD, diabetes, chronic kidney disease, or very high levels of individual risk factors because such people are already at high risk and need intensive risk factor advice.

- SCORE is intended to facilitate risk estimation in apparently healthy persons with no signs of clinical or pre-clinical disease
- Patients who have had a clinical event such as an acute coronary syndrome (ACS) or stroke are at high risk of a further event and automatically qualify for intensive risk factor evaluation and management

escardio.org/static_file/Escardio/Guidelines/publications/DYSLIPguidelines-dyslipidemias-FT.pdf

Etiology
(Genetic and Acquired Factors)

- The etiology of ASVD is unknown, but there are multiple factors that contribute to atherosclerotic plaque progression
- These include genetic and acquired factors



Interactive Role of Infection, Inflammation and Traditional Risk Factors in Atherosclerosis

content.onlinejacc.org/article.aspx?articleid=1124505

Mechanisms
(Key Moments)

- Laying in the base of ASVD atherogenesis is the process of atheromatous plaques formation due to disturbed lipid metabolism (dyslipidemia) that is characterized by subendothelial accumulation of abnormal fatty substances with inflammatory response
- The buildup of an atheromatous plaques is a slow process, developed over a period of several years
- At first, as the plaques grow, only wall thickening occurs without any narrowing
- Stenosis is a late event, which may never occur and is often the result of repeated plaque rupture and healing responses, not just the atherosclerotic process by itself

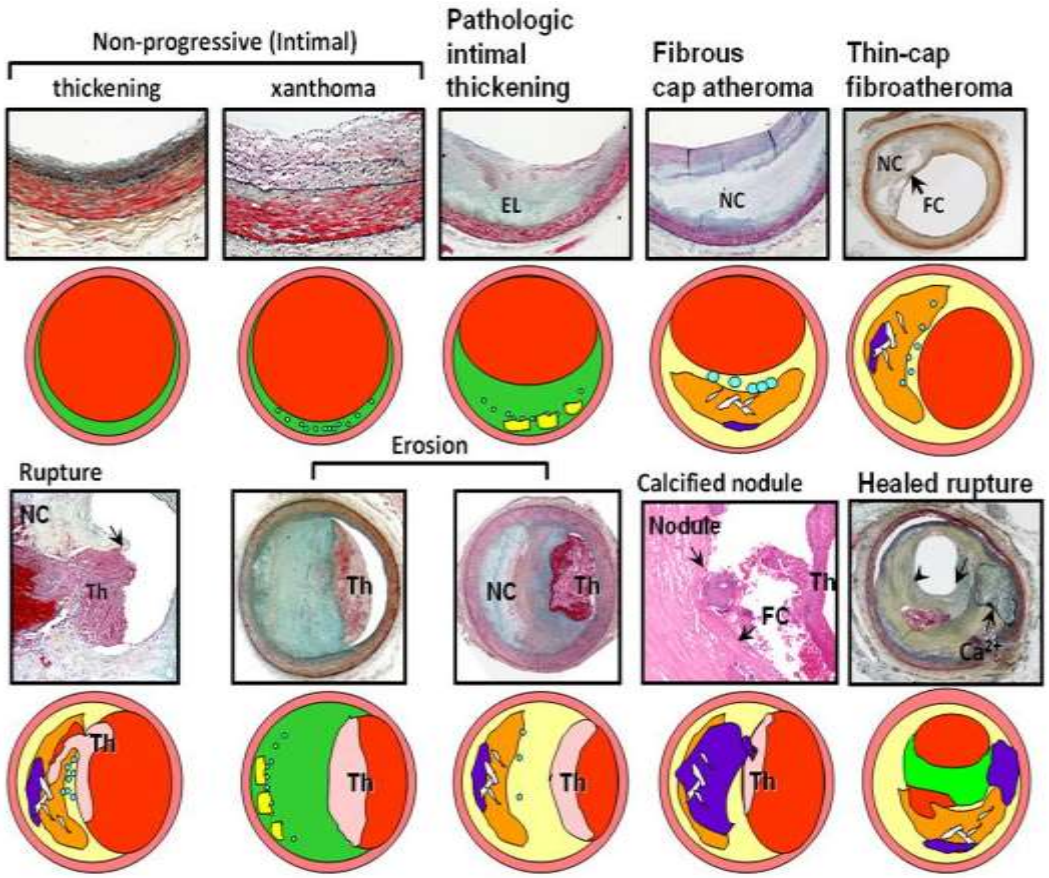
en.wikipedia.org/wiki/Atherosclerosis

Mechanisms
(Passions around Dyslipidemia)

- Dyslipidemia can be developed in different ways and by itself and through interaction with other cardiovascular risk factors may affect the development of ASVD
- Dyslipidemia may be related to other diseases (secondary) or to the interaction between genetic predisposition and environmental factors and cover a broad spectrum of lipid abnormalities, some of which are of great importance in ASVD prevention
- A particular pattern of dyslipidemia, termed the atherogenic lipid triad, is more common than others, and consists of the co-existence of increased very low density lipoprotein (VLDL) remnants manifested as mildly elevated triglycerides (TG), increased small dense low-density lipoprotein (LDL) particles, and reduced high density lipoprotein (HDL) levels

escardio.org/static_file/Escardio/Guidelines/publications/DYSLIPguidelines-dyslipidemias-FT.pdf

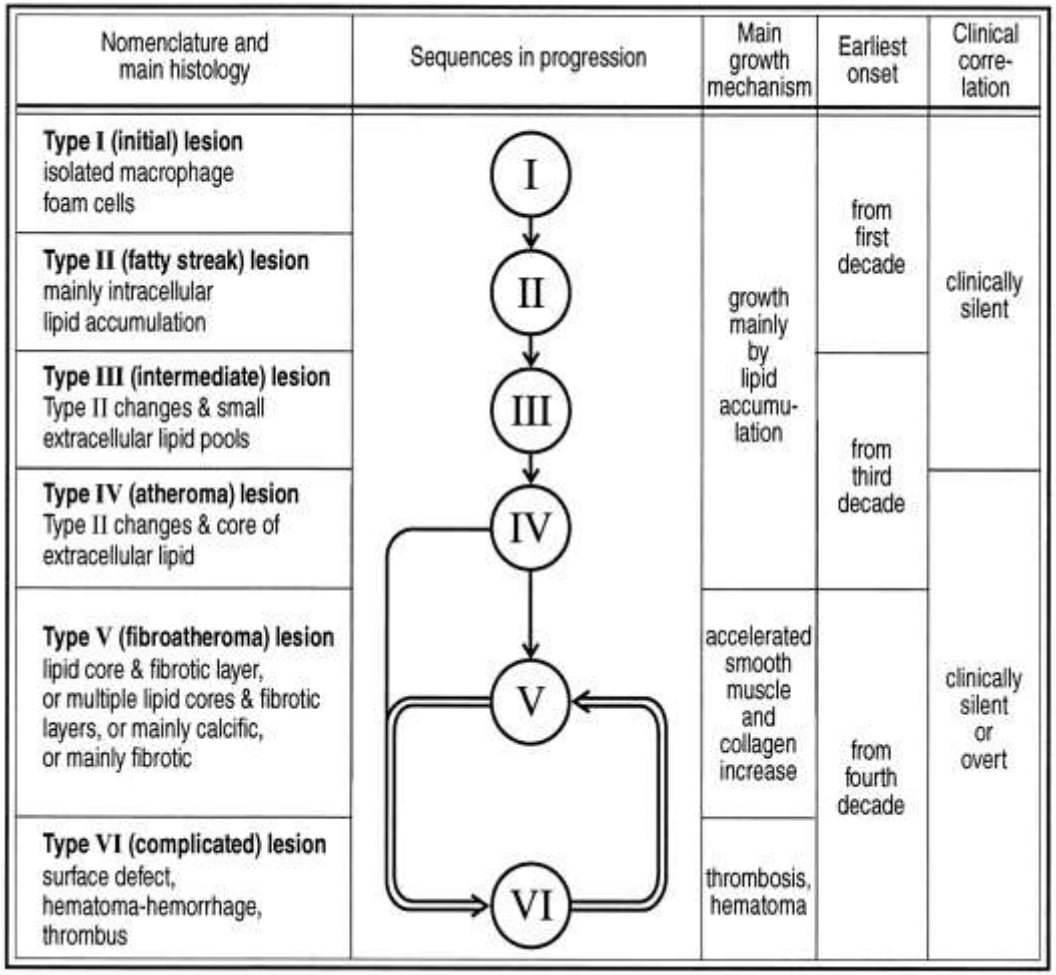
Mechanisms
(Progression of Atherosclerosis)



- Processes involved in atherosclerosis include coagulation, inflammation, lipid metabolism, intimal injury, and smooth muscle cell proliferation
- Early lesion development is marked by lipid retention with activation of endothelial adhesion molecules
- Inflammatory macrophages play a significant role throughout all phases of atherosclerotic progression; hyperlipidemia-induced macrophage infiltration of the arterial intima is one of the earliest pathologic changes

reference.medscape.com/article/1612610-overview#a3

Mechanisms
(Pathways in Evolution and Progression of Atherosclerotic Lesions)

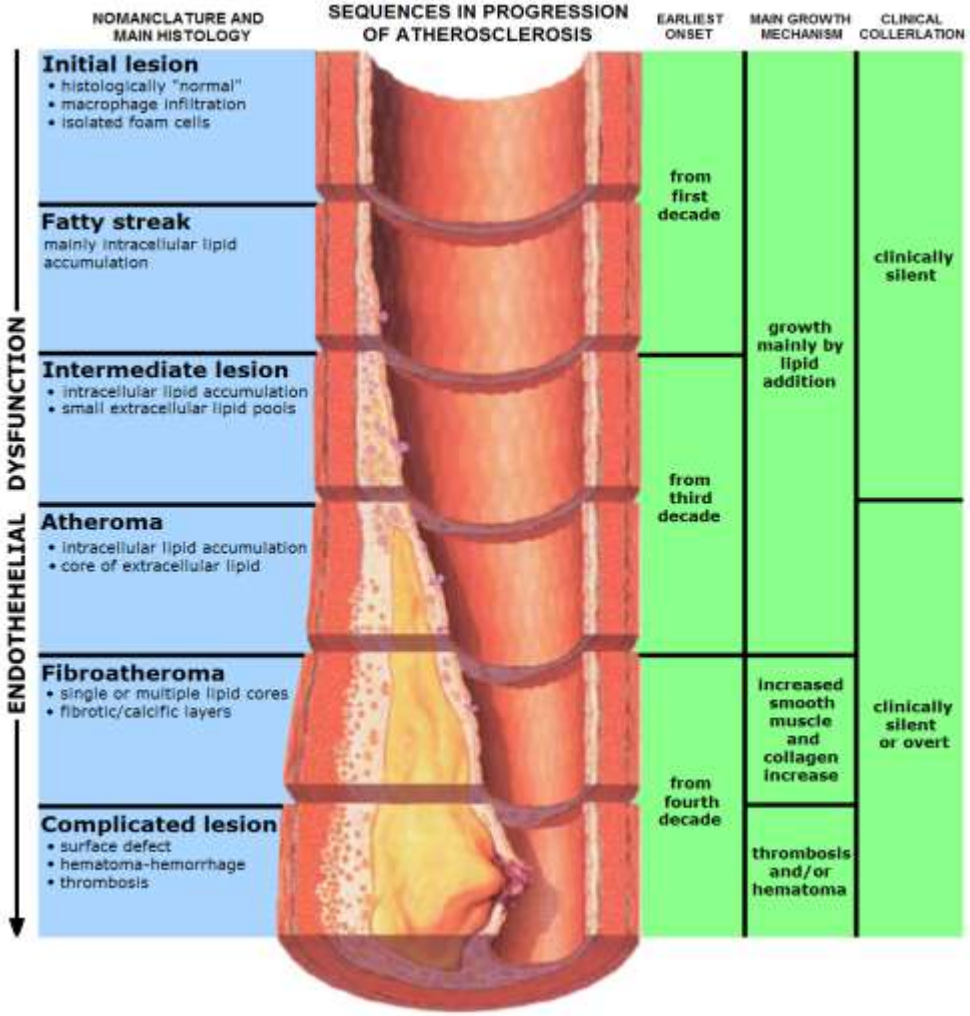


- Roman numerals indicate histologically characteristic types of lesions
- The direction of arrows indicates sequence in which characteristic morphologies may change
- From type I to type IV, changes in lesion morphology occur primarily because of increasing accumulation of lipid
- The loop between types V and VI illustrates how lesions increase in thickness when thrombotic deposits form on their surfaces
- Thrombotic deposits may form repeatedly over varied time spans in the same location and may be the principal mechanism for gradual occlusion of medium-sized arteries

circ.ahajournals.org/content/92/5/1355/F5.expansion.htr

Mechanisms

(Schematic Presentation of Evolution and Progression of Atherosclerotic Lesions)



upload.wikimedia.org/wikipedia/commons/9/9a/Endo_dysfunction_Athero.PNG

Classification
(Modified AHA Consensus Classification Based on Morphologic Descriptions:Nonatherosclerotic intimal lesions)

Morphology	Description	Thrombosis
Intimal thickening	Normal accumulation of smooth muscle cells (SMCs) in the intima in the absence of lipid or macrophage foam cells	Absent
Intimal xanthoma	Superficial accumulation of foam cells without a necrotic core or fibrous cap; based on animal and human data, such lesions usually regress	Absent

reference.medscape.com/medline/abstract/10807742

Classification
(Modified AHA Consensus Classification Based on Morphologic Descriptions: Progressive atherosclerotic lesions)

Morphology	Description	Thrombosis
Pathologic intimal thickening	SMC-rich plaque with proteoglycan matrix and focal accumulation of extracellular lipid	Absent
Fibrous cap atheroma	Early necrosis: focal macrophage infiltration into areas of lipid pools with an overlying fibrous cap Late necrosis: loss of matrix and extensive cellular debris with an overlying fibrous cap	Absent
Thin cap fibroatheroma	A thin, fibrous cap (< 65 µm) infiltrated by macrophages and lymphocytes with rare or absence of SMCs and a relatively large underlying necrotic core; intraplaque hemorrhage/fibrin may be present	Absent

reference.medscape.com/medline/abstract/10807742

Classification
(Modified AHA Consensus Classification Based on Morphologic Descriptions: Lesions with acute thrombi)

	Description	**Thrombosis**
Plaque rupture	Fibroatheroma with fibrous cap disruption; the luminal thrombus communicates with the underlying necrotic core	Occlusive or nonocclusive
Plaque erosion	Plaque composition, as above; no communication of the thrombus with the necrotic core; can occur on a plaque substrate of pathologic intimal thickening or fibroatheroma	Usually nonocclusive
Calcified nodule	Eruptive (shedding) of calcified nodules with an underlying fibrocalcific plaque with minimal or absence of necrosis	Usually nonocclusive

reference.medscape.com/medline/abstract/10807742

Classification
(Modified AHA Consensus Classification Based on Morphologic Descriptions: Lesions with healed thrombi)

Morphology	Description	Thrombosis
Fibrotic (without calcification) Fibrocalcific (+/- necrotic core)	Collagen-rich plaque with significant luminal stenosis; lesions may contain large areas of calcification with few inflammatory cells and minimal or absence of necrosis; these lesions may represent healed erosions or ruptures	Absent

reference.medscape.com/medline/abstract/10807742

Classification
(Frederic Classification of Lipid Disorders)

Type	Primary Lipid Elevation	Lipoprotein	Occurrence
I	TG	Chylomicrons	Rare
II_a	C	LDL	Common
II_b	C, TG	LDL, VLDL	Most Common
III	C, TG	IDL	Rare
IV	TG	VLDL	Common
V	TG	VLDL, Chylomicrons	Rare

LDL - low-density lipoproteins; C - total cholesterol; TG - triglycerides; VLDL - very low-density lipoproteins

Classification
(International Classification of Diseases (ICD))

Chapter V
(F00-F99) Mental and behavioral disorders

F00-F09 Organic, including symptomatic, mental disorders

Chapter IX
(I00-I99) Diseases of the circulatory system

I20-I25 Ischaemic heart diseases

(I60-I69) Cerebrovascular diseases

I70-I79 Diseases of arteries, arterioles and capillaries

170 Atherosclerosis

I70.0 Atherosclerosis of aorta

I70.1 Atherosclerosis of renal artery

I70.2 Atherosclerosis of arteries of extremities

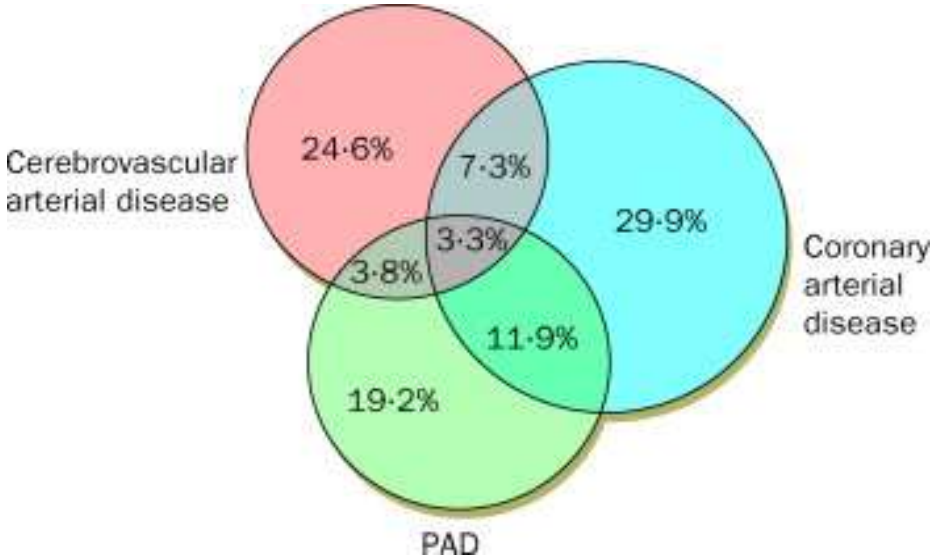
I70.8 Atherosclerosis of other arteries

I70.9 Generalized and unspecified atherosclerosis

google.com.ua/webhp?sourceid=chrome-instant&ion=1&espv=2&ie=UTF-8#q=international%20diseases%20classification%2010

Classification
(Overlap of Atherosclerotic Disease)

- Coronary artery disease
- Carotid artery disease
- Peripheral artery disease
- Kidney disease



Patients with one type of ASVD are also at risk of having other types

healthline.com/health/atherosclerosis#Types2 thelancet.com/cms/attachment/2001932257/2006685340/gr1.jpg

Clinical Investigation
(Passions around Symptoms)

- ASVD is asymptomatic for decades, even most plaque ruptures do not produce symptoms until enough narrowing or closure of an artery, due to clots, occurs
- Symptomatic ASVD is typically associated with men in their 40s and women in their 50s to 60s
- Signs and symptoms only occur after severe narrowing or closure impedes blood flow to different organs enough to induce symptoms and most of the time, patients realize that they have the disease
- Clinical manifestation and picture of ASVD strictly depends from a level and severity lesions of systemic circulation arteries, more often aorta, coronary, carotid, iliac, femoral, renal, etc.

en.wikipedia.org/wiki/Atherosclerosis

Clinical Investigation
(Modalities of Symptoms)

- ASVD causes symptoms by arterial obstruction, embolization of plaque material, and weakening with rupture of the arterial wall
- Obstruction with or without embolization causes ischemia of the circulation supplied by the vessel:
  - Ischemic strokes result from ASVD of the carotid arteries and aortic arch, which embolize thrombi and atherosclerotic material, as well as local atherosclerosis of the cerebral vessels
  - Obstruction of coronary arteries causes myocardial ischemia that may present as acute coronary syndromes (acute ST elevation infarct, non-ST elevation infarct, and unstable angina), sudden death, or chronic congestive heart failure
  - Obstruction of iliac vessels results in ischemia of the lower extremities (claudication)
  - Atherosclerotic aneurysms show a predilection for the aorta, especially the abdominal aorta, that may rupture and cause death by hemorrhage into the retroperitoneal space or pleural cavities, depending on the location

reference.medscape.com/article/1612610-overview#a5

Clinical Investigation
(Involved Organs' Symptoms)

- Heart - chest pain of angina and shortness of breath, sweating, nausea, dizziness or light-headedness, breathlessness or palpitations
- Brain - dizziness or confusion; weakness or paralysis on one side of the body; sudden, severe numbness in any part of the body; visual disturbance, including sudden loss of vision; difficulty walking, including staggering or veering; coordination problems in the arms and hands; and slurred speech or inability to speak
- Abdomen - dull or cramping pain in the middle of the abdomen, usually beginning 15 to 30 minutes after a meal
- Legs - cramping pain in the leg muscles, especially during exercise; if narrowing is severe, there may be pain at rest, cold toes and feet, pale or bluish skin and hair loss on the legs

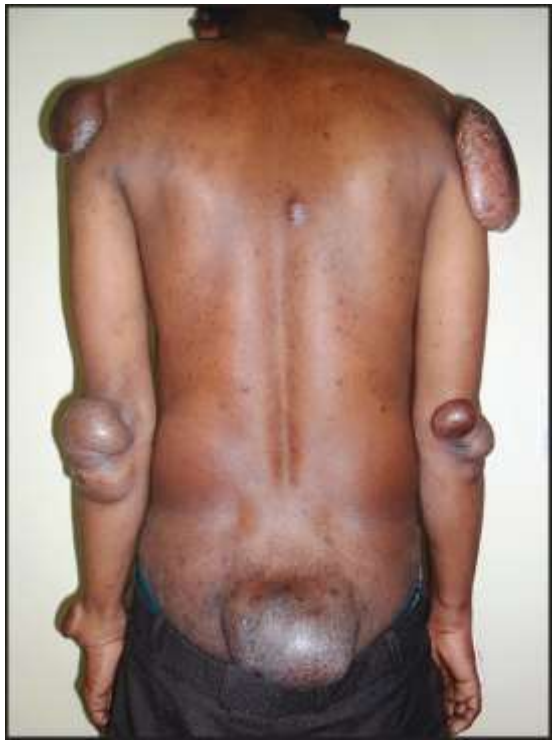
drugs.com/health-guide/atherosclerosis.html

Clinical Investigation
(Physical Examination)

- A weak or absent pulse below the narrowed area of affected artery
- Decreased blood pressure in an affected limb
- Whooshing sounds (bruits) over affected arteries
- Weak pulses
- Cool skin that is pale or blue in an affected limb
- Hair loss on an affected limb
- Male erectile dysfunction
- Numbness in an affected limb
- The toenails get thicker
- Weakness in an affected limb
- Early gray hair
- Reduced skin turgor
- Xanthomas and xanthelasma
- Senile corneal arc
- Abundant growth of hair on the ears (Gabrielli symptom)
- Vertical or diagonal crease in the earlobe (Frank's sign)

eurheartj.oxfordjournals.org/content/ehj/34/28/2159.full.pdf

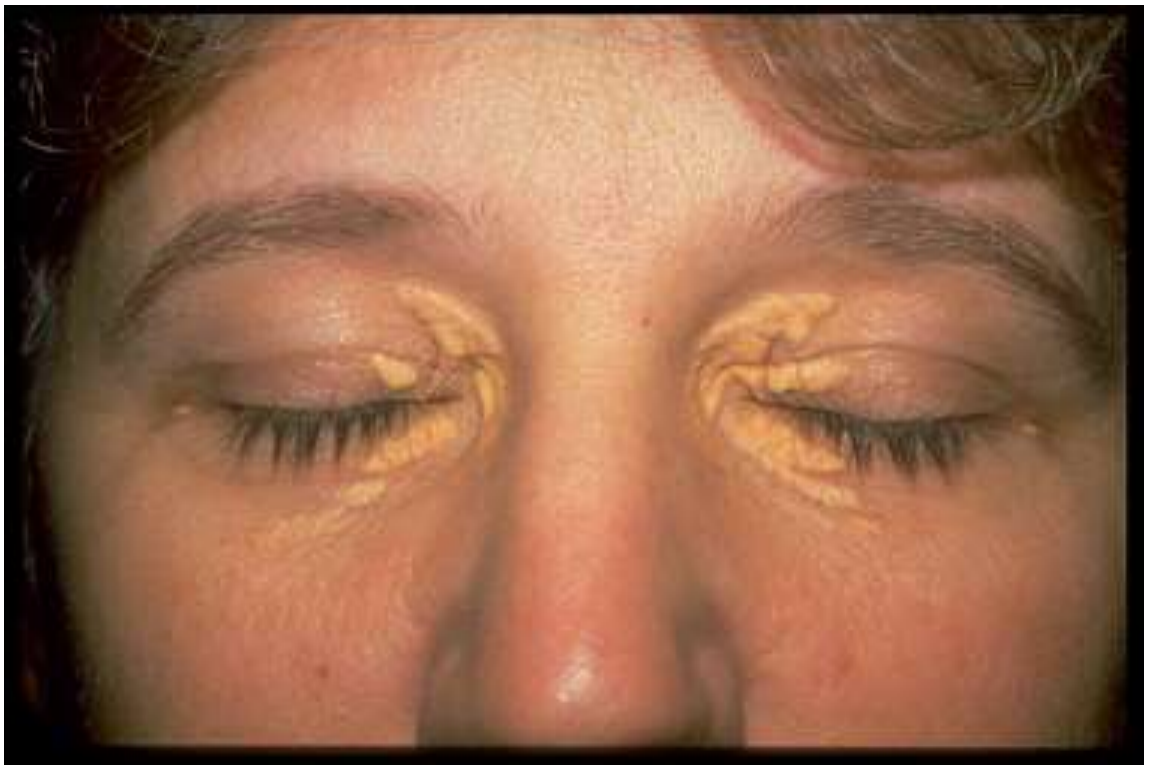
Clinical Investigation
(Xanthoma)



A 19-year-old male presented with effort angina of 6-month duration, which has worsened since 1 month associated with rest angina, and postprandial angina. Extensive xanthoma tuberosum around shoulders, elbows, wrists, and sacrum.

annalspc.com/article.asp?issn=0974-2069;year=2014;volume=7;issue=2;spage=118;epage=119;aulast=Moorthy

Clinical Investigation
(Xanthelasma)



Xanthelasma of four eyelids in patient with hyperlipidemia. Xanthelasma can be soft, semisolid, or calcareous. Frequently, they are symmetrical. The upper lids are more frequently involved than the lower lids. Xanthelasma have a tendency to progress, coalesce, and become permanent.

emedicine.medscape.com/article/1213423-overview

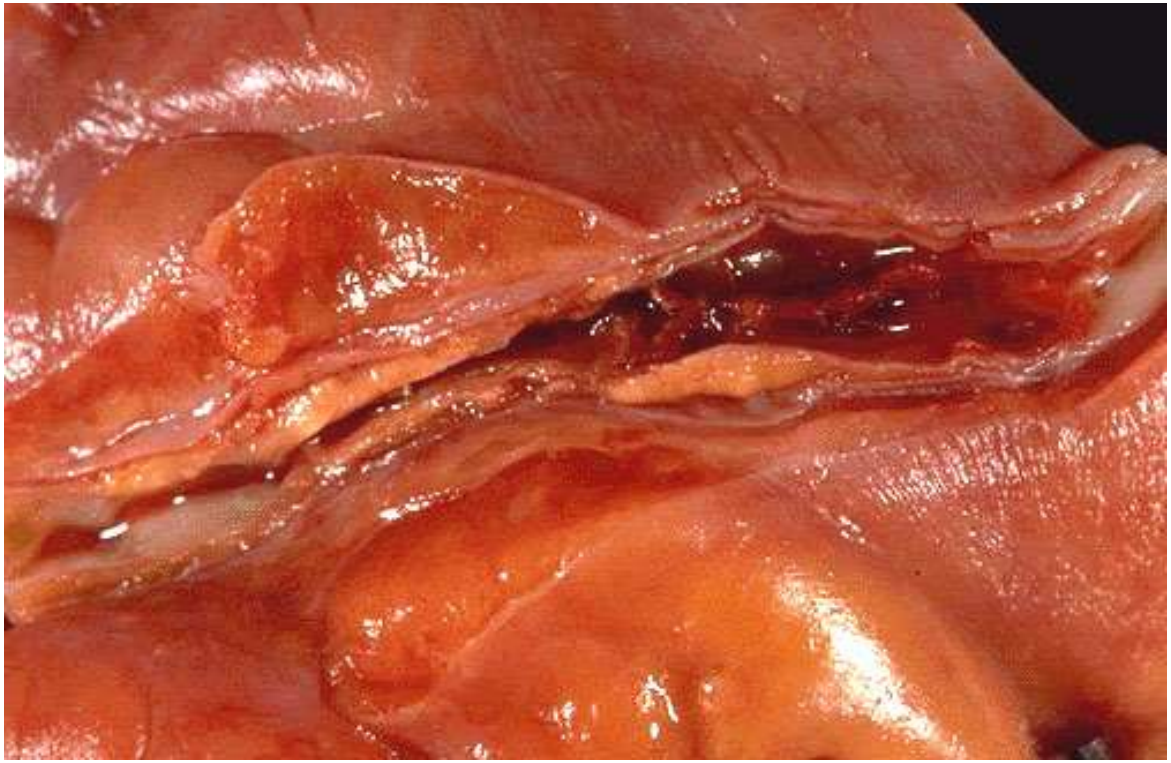
Clinical Investigation
(Vertical or diagonal crease in the earlobe)

Hairs protruding out of the ear-tube and crease on the ear lobe have significant correlation with higher incidence of coronary risk

Clinical Investigation
(Outcomes)

- Premature death
- Thrombosis
- Vascular aneurisms
- Severe vascular narrowing
- Vascular ruptures and hemorrhages
- Acute ischemic vascular syndromes
- Chronic multiple organ dysfunction and failure (cognitive impairment and dementia, chronic heart failure, chronic kidney disease, limb gangrene)
- Premature aging
- Etc.

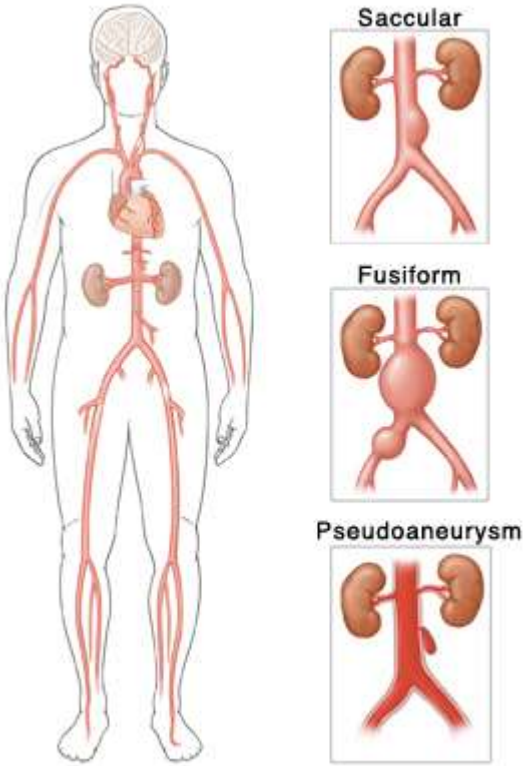
Clinical Investigation
(Thrombosis)



This cross section reveals a large myocardial infarction involving the anterior left ventricular wall and septum. Here is the coronary thrombosis at higher magnification. The thrombus occludes the lumen and produces ischemia

jpkc.gdmc.edu.cn/blx/10jxwz/tupu/bltp/8xxg10.htm

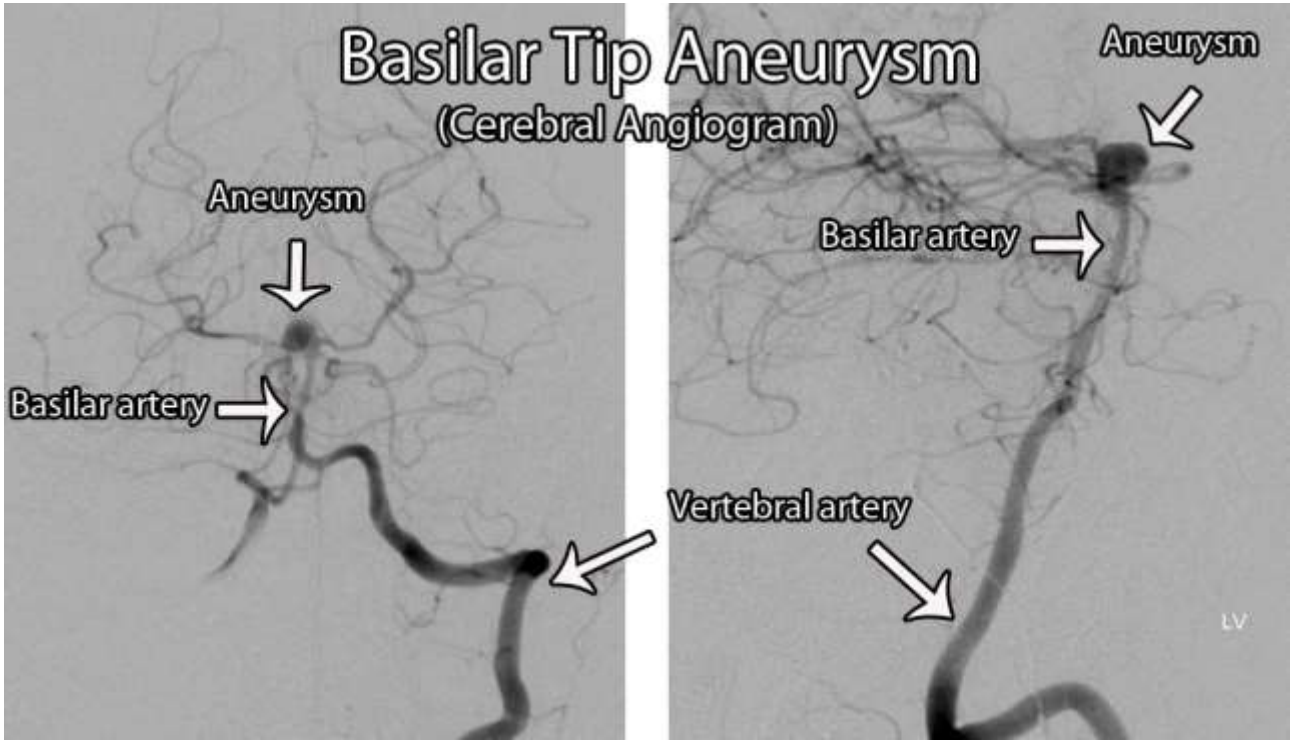
Clinical Investigation
(Vascular aneurisms)



- Different types of (aortic) aneurysm
- An aneurysm is a bulging, weakened area in the wall of a blood vessel resulting in an abnormal widening or ballooning greater than 50% of the vessel's normal diameter (width)
- An aneurysm may occur in any blood vessel, but is most often seen in an artery rather than a vein

hopkinsmedicine.org/healthlibrary/conditions/cardiovascular_diseases/aneurysm_85,p00193/

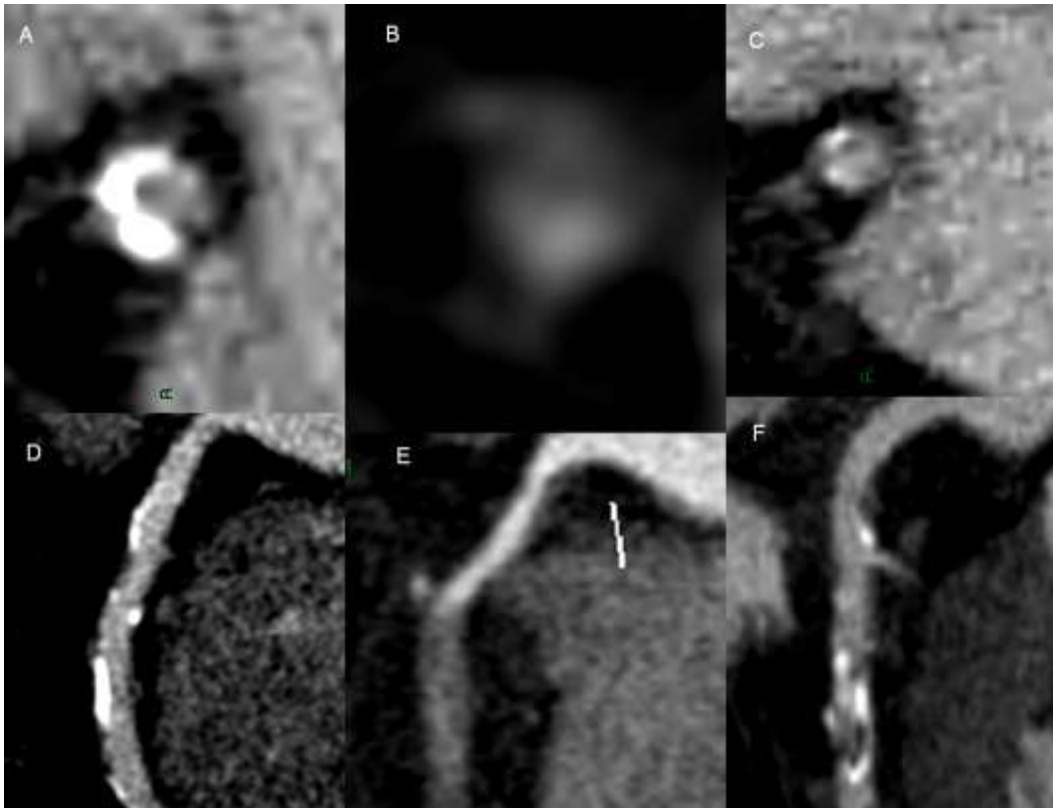
Clinical Investigation
(Vascular aneurisms)



Basilar tip aneurysms are usually diagnosed after a subarachnoid hemorrhage or workup for cranial nerve dysfunction. The best methods for diagnosing basilar tip aneurysms are with CT angiograms and formal cerebral angiograms. Non-contrasted head CT scans can illustrate blood in the subarachnoid space if the aneurysm has ruptured.

virtualmedstudent.com/links/neurological/aneurysm_basilar_artery.html

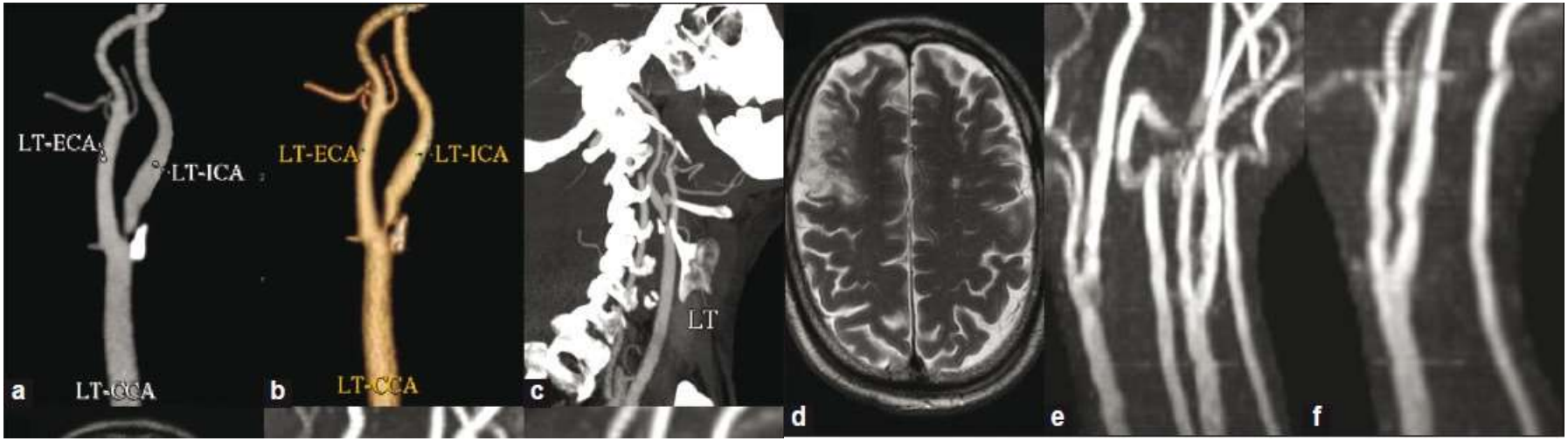
Clinical Investigation
(Severe vascular narrowing)



Different Types of Coronary Plaques by Coronary Computed Tomography Angiography (CCTA). The 3 main types of coronary plaques are shown: calcified plaques (A, D), non-calcified plaques (B, E) and partially calcified plaques (C, F), illustrated in curved planar reformatted and cross-sectional views.

ntechopen.com/books/coronary-artery-disease-assessment-surgery-prevention/coronary-ct-angiography-and-the-napkin-ring-sign-indicates-high-risk-atherosclerotic-lesions

Clinical Investigation
(Severe vascular narrowing)



Imaging findings in a patient with bilateral internal carotid artery (ICA) atherosclerotic lesions. CT angiography images (a and b) showing critical stenosis at the left ICA origin. Localization of level of stenotic ICA in relation to the cervical spine bony anatomical landmarks on imaging to plan optimum level of exposure (c). A preoperative MRI T2-axial image (d) showing evidence of subcortical and deep white matter ischemic lesions. A postoperative MR angiography (e and f) showing complete restoration of normal ICA caliber.

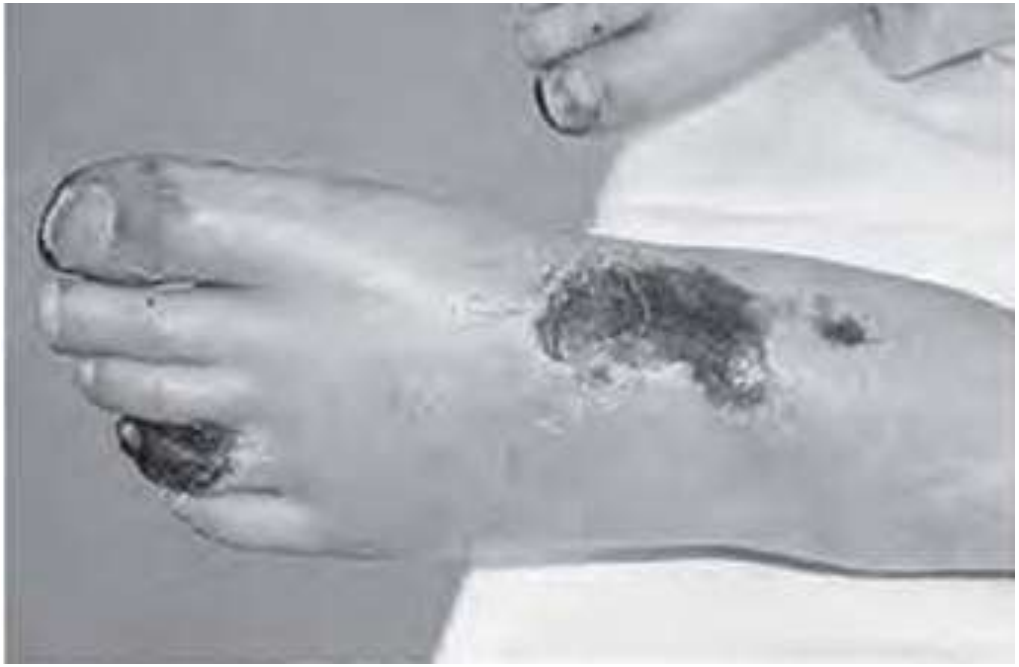
neurologyindia.com/article.asp?issn=0028-3886;year=2011;volume=59;issue=3;spage=390;epage=396;aulast=Misra;type=3

Clinical Investigation
(Severe vascular narrowing)

- Severe atherosclerosis of cerebral arteries
- Atheromatous plaques may cause narrowing or occlusion of the vascular lumen by themselves or after rupture and thrombosis
- Cholesterol crystals from ruptured plaques may embolize

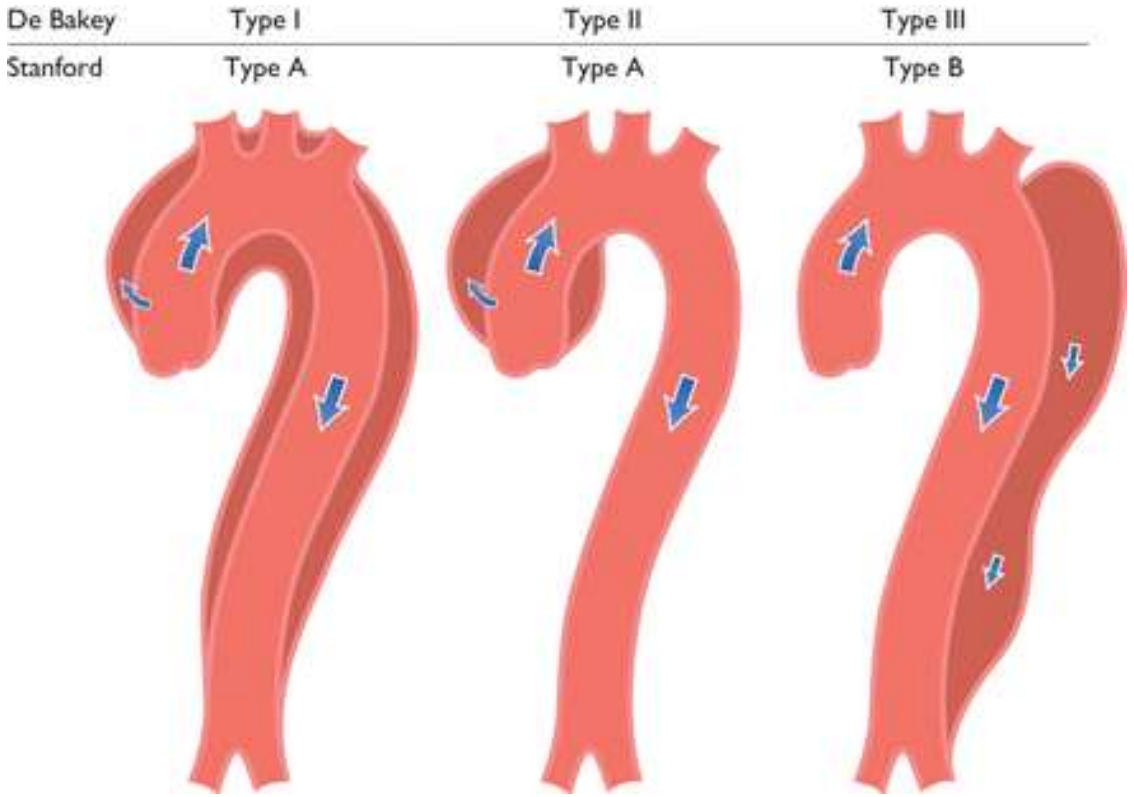
neuropathology-web.org/chapter2/chapter2bCerebralinfarcts.html

Clinical Investigation
(Severe vascular narrowing)



An ischemic ulcer is characterized by a gangrenous skin change in the foot or toes. The foot is usually cold to touch with absent pedal pulses. The foot is painful to touch with decreased distal capillary refills.

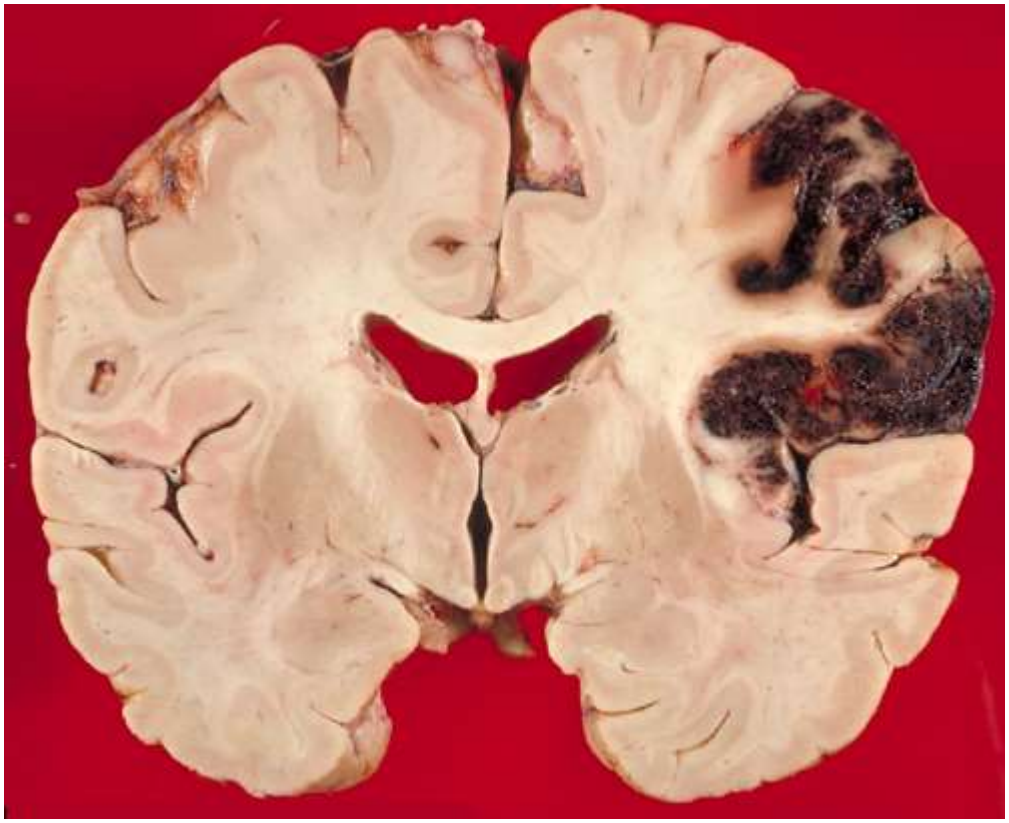
Clinical Investigation
(Vascular ruptures and hemorrhages)



Classification of aortic dissection localization

eurheartj.oxfordjournals.org/content/35/41/2873

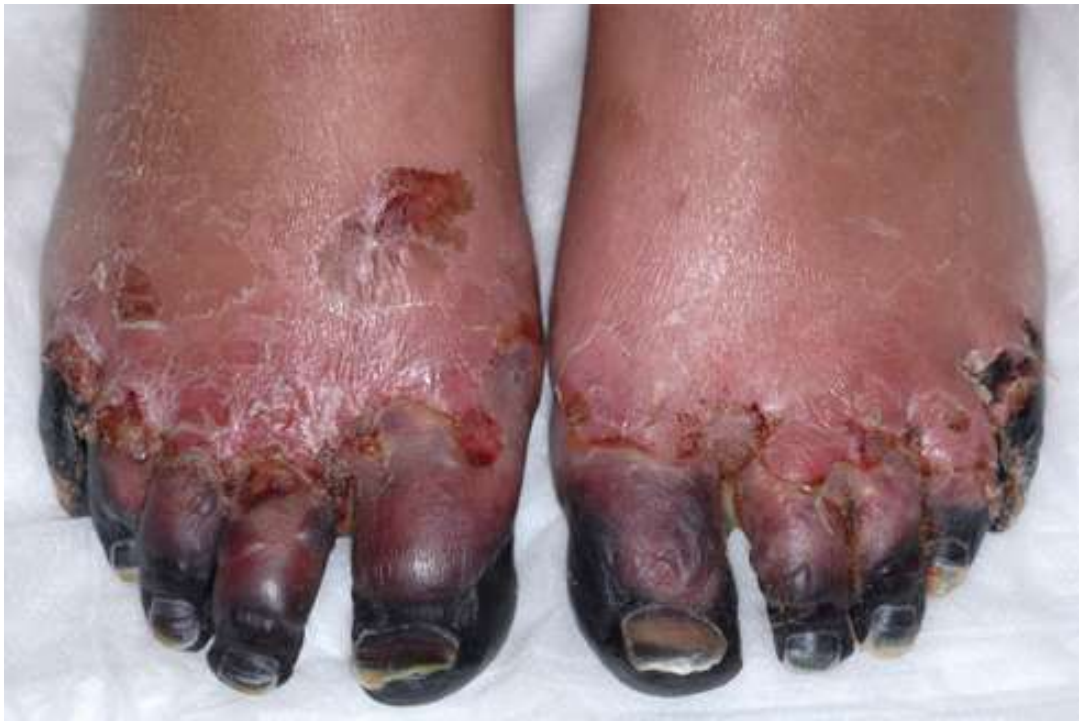
Clinical Investigation
(Vascular ruptures and hemorrhages)



Hemorrhagic infarcts are most common in embolism. Use of thrombolytics or anticoagulants may convert a bland infarct into a hemorrhagic one.

neuropathology-web.org/chapter2/chapter2bCerebralinfarcts.html

Clinical Investigation
(Limb gangrene)



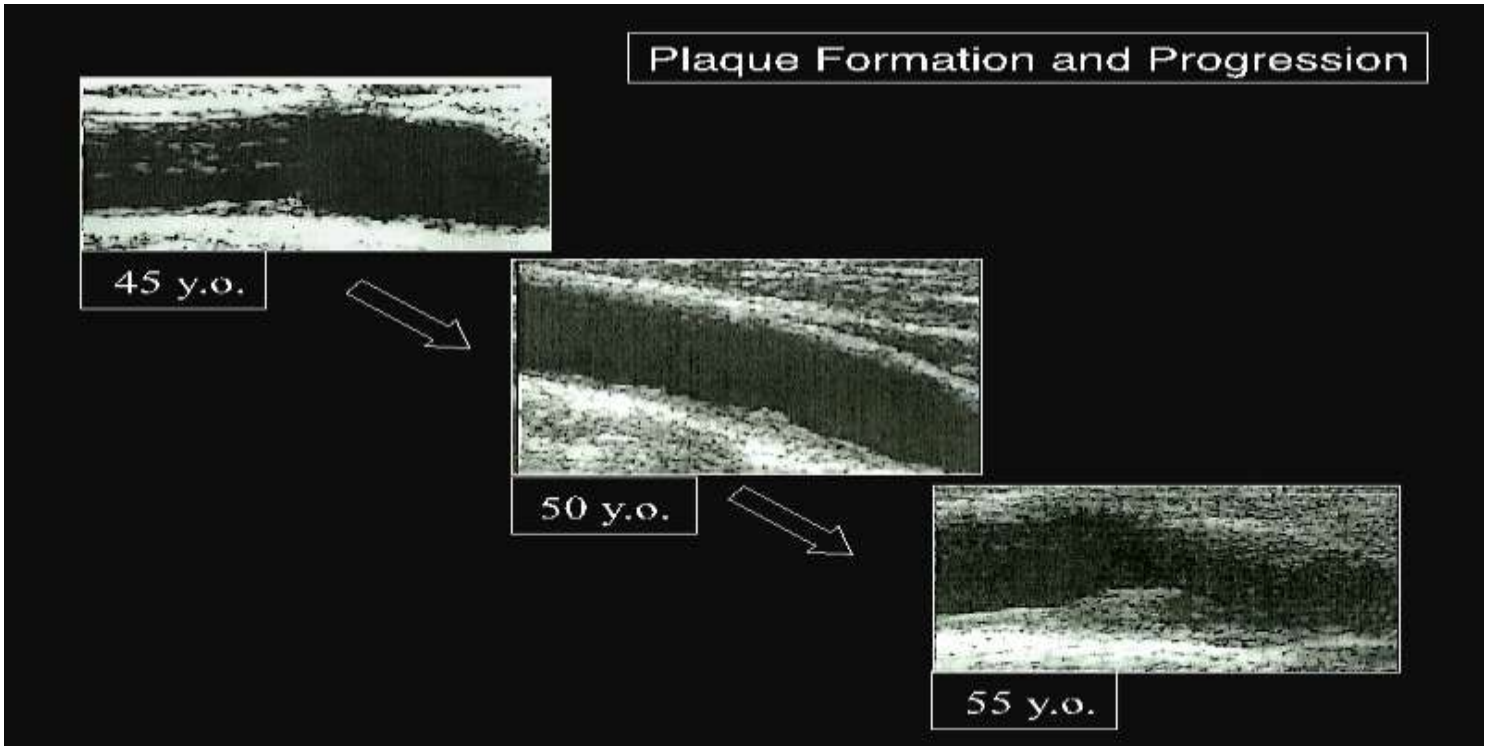
Dry gangrene of the toes showing the areas of total tissue death, appearing as black and lighter shades of discoloration of the skin demarcating areas of impending gangrene. (Photo contributor: Lawrence B. Stack, MD.)

Diagnosis

- Blood tests: cholesterol and C-reactive protein levels, lipoproteinogram
- B-mode, Doppler and intravascular ultrasound: degree of any blockages in arteries
- Ankle-brachial index: recognition of peripheral vascular disease, which is usually caused by atherosclerosis
- Electrocardiogram (ECG): reveal evidence of a previous heart attacks
- Stress test (including echocardiogram): reveal evidence of a chronic coronary artery disease
- Vascular catheterization and angiogram: reveal evidence of narrowed, blocked or aneurismatic arteries
- Other imaging tests (CT, MRA, etc.): reveal evidence of narrowed or blocked arteries, as well as aneurysms and calcium deposits in the artery walls

mayoclinic.org/diseases-conditions/arteriosclerosis-atherosclerosis/diagnosis-treatment/diagnosis/dxc-20167050

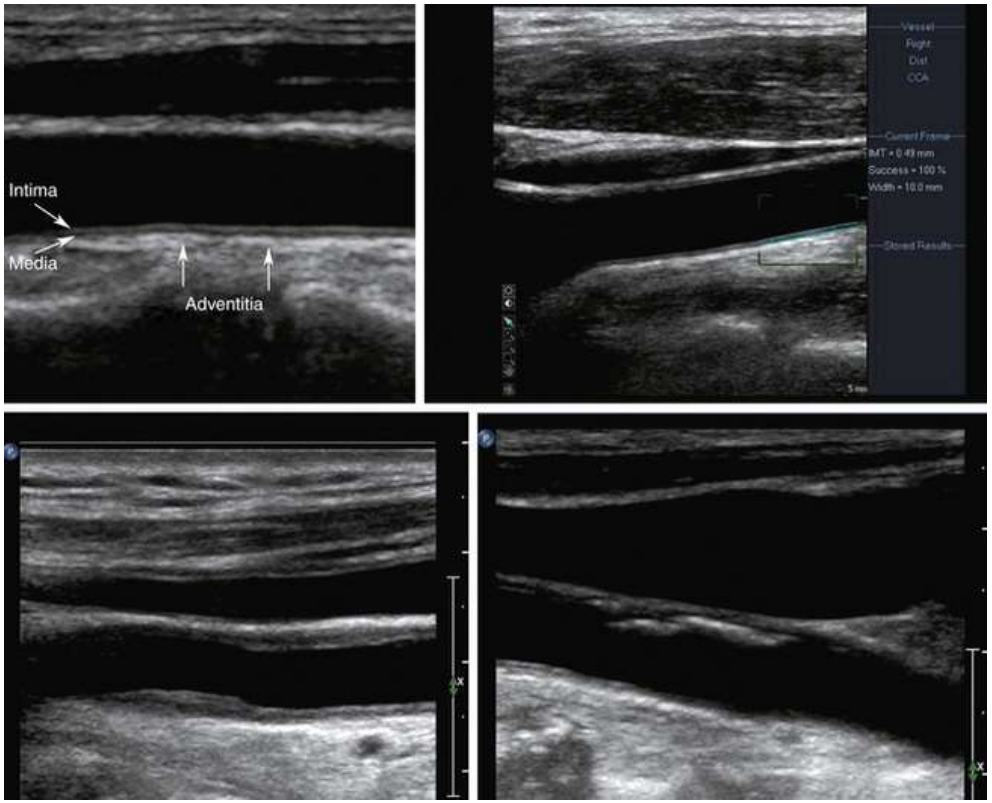
Diagnosis
(B-mode ultrasound)



B-mode ultrasound imaging of the left carotid artery in a long-axis during a 10 years period follow-up (1998-2008) in a healthy man from 45 to 55 years of age.

ntechopen.com/books/ultrasound-imaging-medical-applications/atherosclerotic-plaque-regression-and-arterial-reverse-remodelling-in-carotid-and-femoral-arteries-b

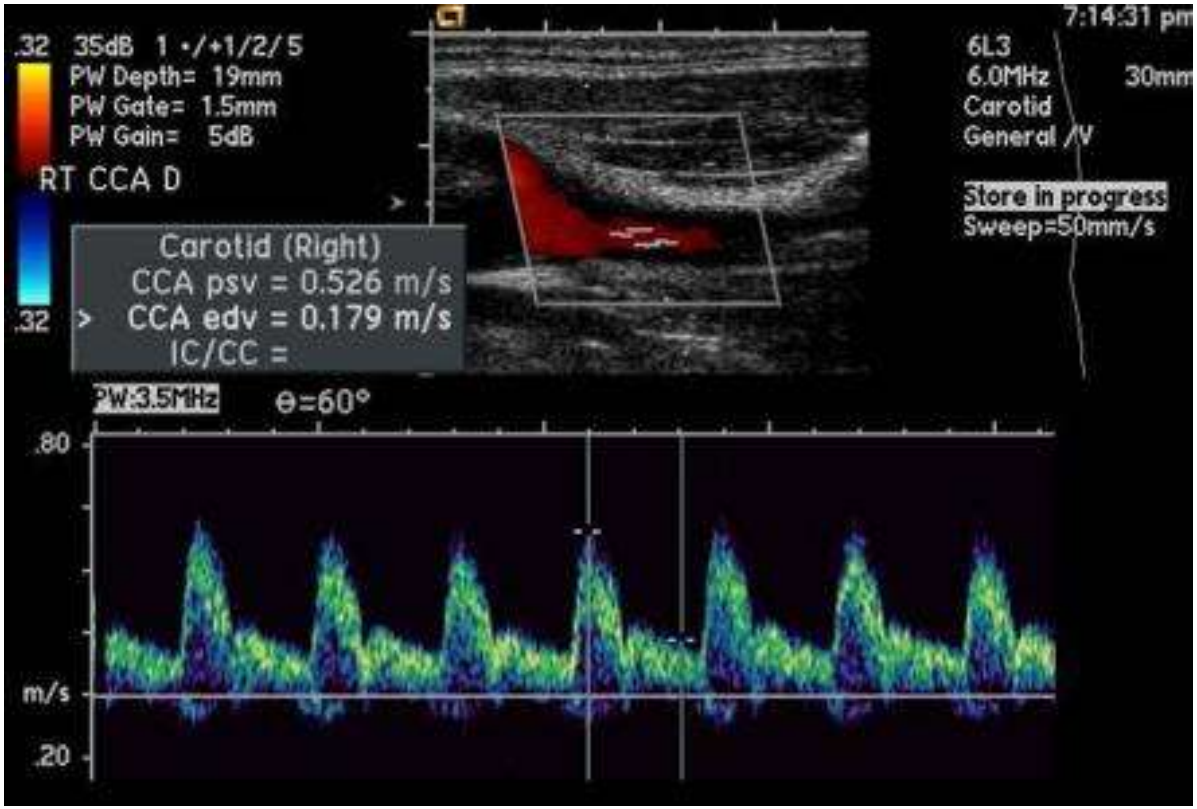
Diagnosis
(B-mode ultrasound)



Normal intima-media thickness (IMT) appearance *(top left),* IMT measurement example *(top right),* fatty streak *(bottom left),* and a homogenous hyperechoic nonstenosing plaque *(bottom right).*

radiologykey.com/the-role-of-ultrasound-in-the-management-of-cerebrovascular-disease/

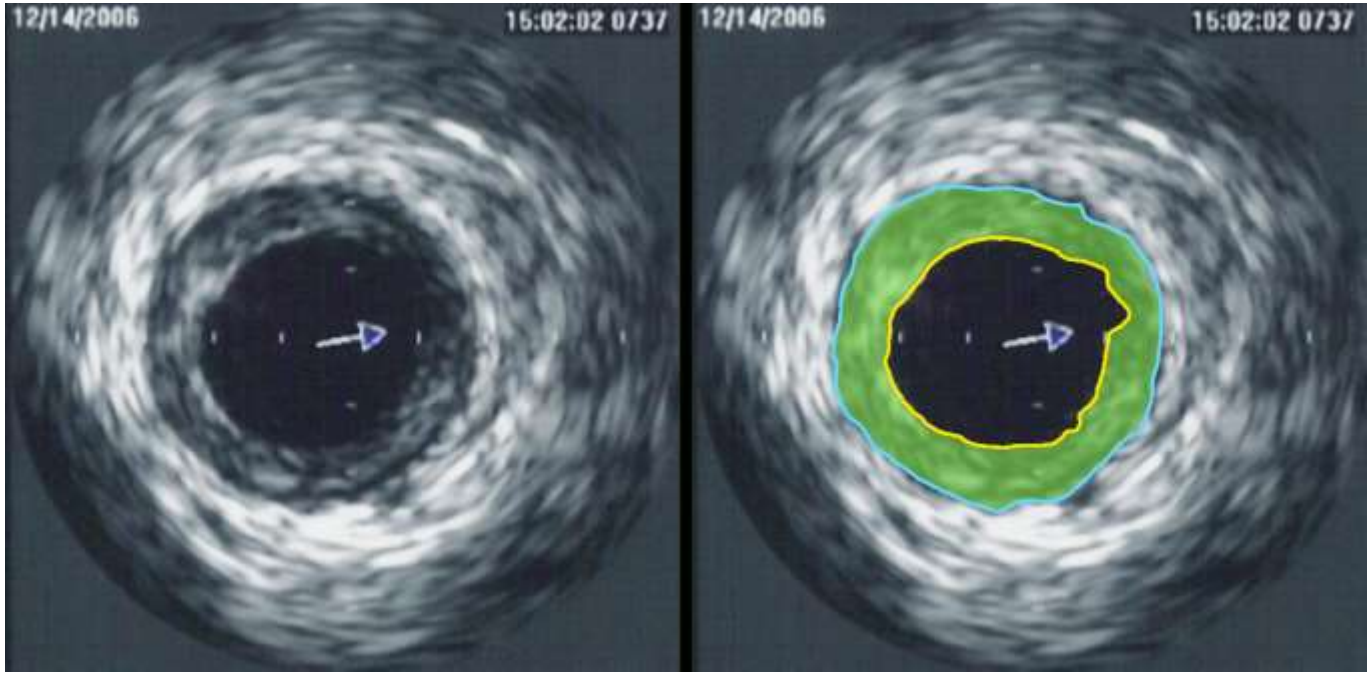
Diagnosis
(Doppler ultrasound)



The right internal carotid artery stenosis estimated between 50 - 69%

radiopaedia.org/articles/ultrasound-assessment-of-carotid-arterial-atherosclerotic-disease

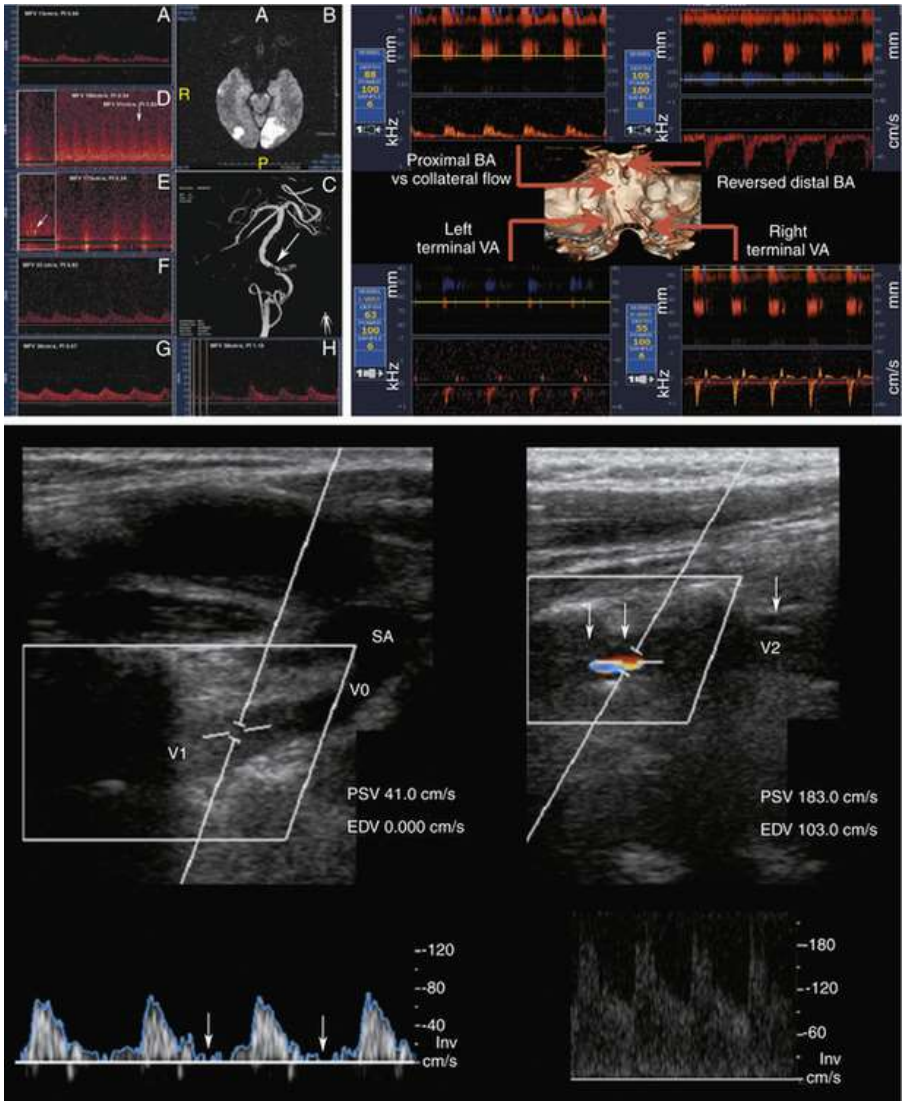
Diagnosis
(Intravascular ultrasound)



Intravascular ultrasound image of a coronary artery (left), with color-coding on the right, delineating the lumen (yellow), external elastic membrane (blue) and the atherosclerotic plaque burden (green). The percentage stenosis is defined as the area of the lumen (yellow) divided by the area of the external elastic membrane (blue) times 100. As the plaque burden increases, the lumen size will decrease and the degree of stenosis will increase.

en.wikipedia.org/wiki/Intravascular_ultrasound

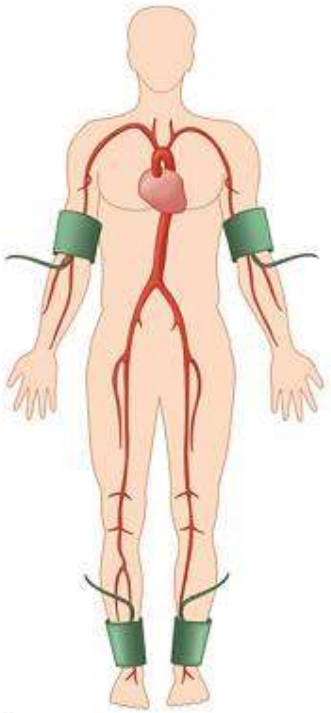
Diagnosis
(Combine visualization)



Example of the terminal vertebral stenosis as shown in the upper left insert of images (*large arrow,* stenosis on arteriogram). On the upper right insert, bilateral terminal vertebral artery (VA) occlusions with reversal of the basilar artery (BA) flow. The lower inserts show a high-grade proximal VA stenosis with a high-resistance *(arrow)* preobstructive flow *(left image)* and a velocity jet *(double arrows)* across the lesion *(right image).* EDV, end-diastolic velocity; PSV, peak systolic velocity.

radiologykey.com/the-role-of-ultrasound-in-the-management-of-cerebrovascular-disease/

Diagnosis
(Ankle-brachial index)



Right ABI = ratio of

Higher of the right ankle systolic pressures (posterior tibial or dorsalis pedis)

Higher arm systolic pressure (left or right arm)

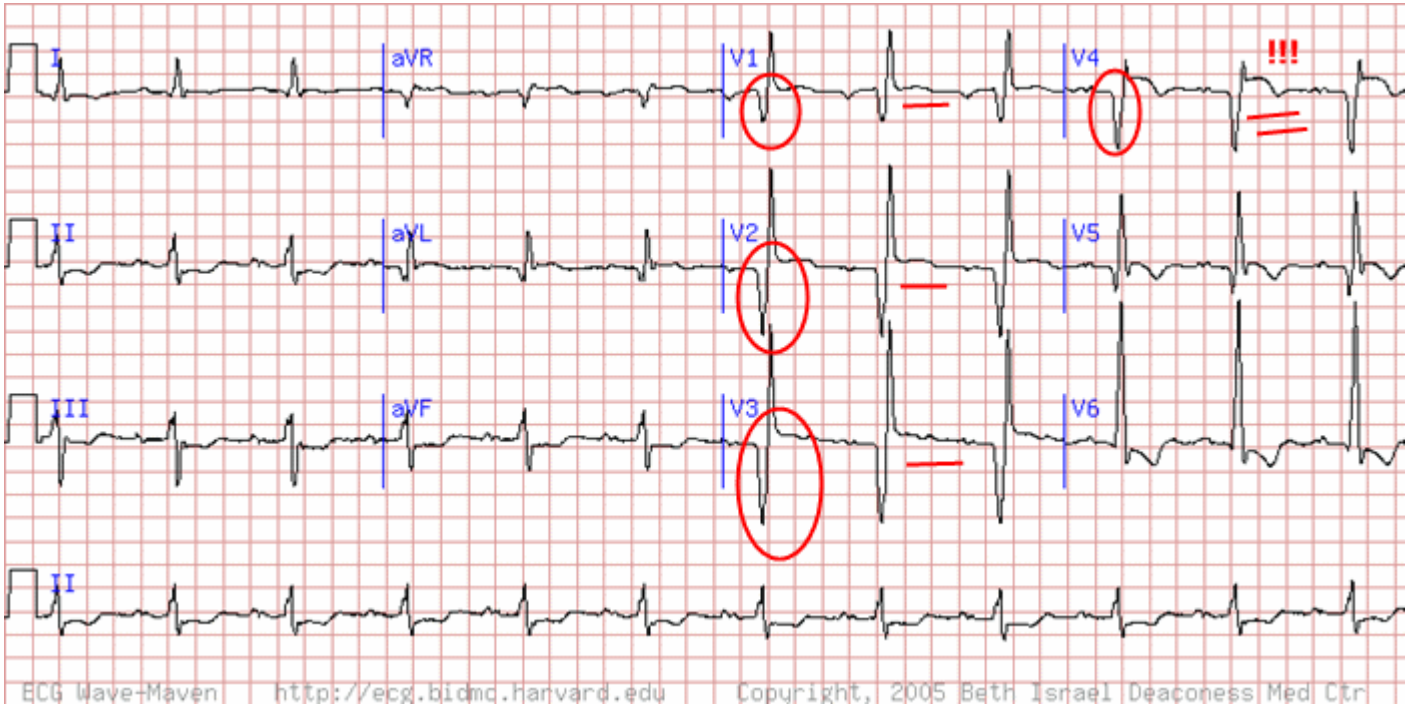
Left ABI = ratio of

Higher of the left ankle systolic pressures (posterior tibial or dorsalis pedis)

Higher arm systolic pressure (left or right arm)

intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/surgery/5/Topic%2008.%20Arteries%20occlusive%20diseases.Mesenteric%20artery%20diseases.htm

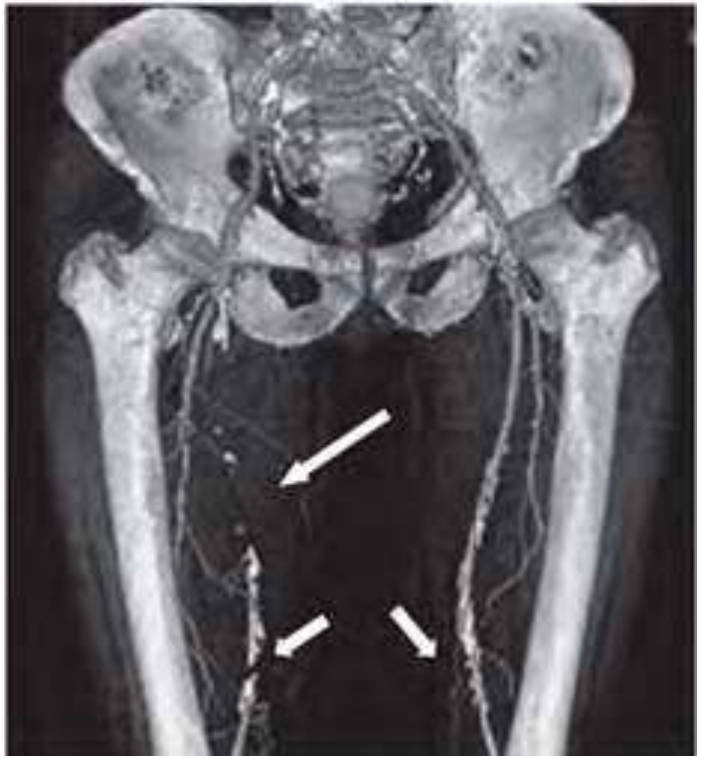
Diagnosis
(Electrocardiogram)



An old post-infarction scar of the front wall (deep Q in V1- V4). The situation is most probably complicated by a suspected ventricular aneurysm. ST sections for QRS in V1 -V4 are elevated, best seen in V4 where the elevation reminds the Pardee's wave (underlined in red).

health-tutor.com/myocardial-scar-ecg.html

Diagnosis
(Vascular catheterization and angiogram)



A multidetector computed tomographic angiogram with three-dimensional reconstruction of the iliofemoral arterial circulation. A 50-year-old man with an occluded right superficial femoral artery (*single long arrow*) with reconstituted superficial femoral artery at the level of midthigh. Arterial calcifications (*short arrows*) in the bilateral distal superficial femoral arteries.

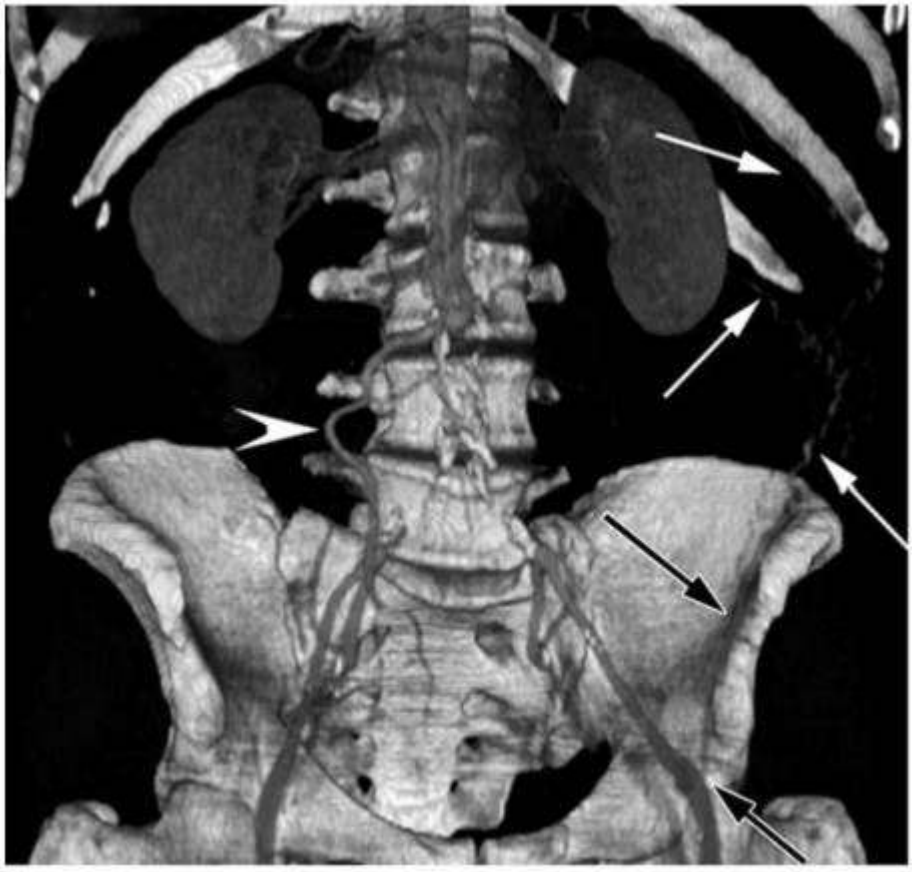
intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/surgery/5/Topic%2008.%20Arteries%20occlusive%20diseases.Mesenteric%20artery%20diseases.htm

Diagnosis
(Vascular catheterization and angiogram)

Three-dimensional computed tomographic angiogram of an abdominal aortic aneurysm that displays various aneurysm components, including thrombus, aortic calcification, blood circulation, and aneurysm wall.

intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/surgery/5/Topic%2008.%20Arteries%20occlusive%20diseases.Mesenteric%20artery%20diseases.htm

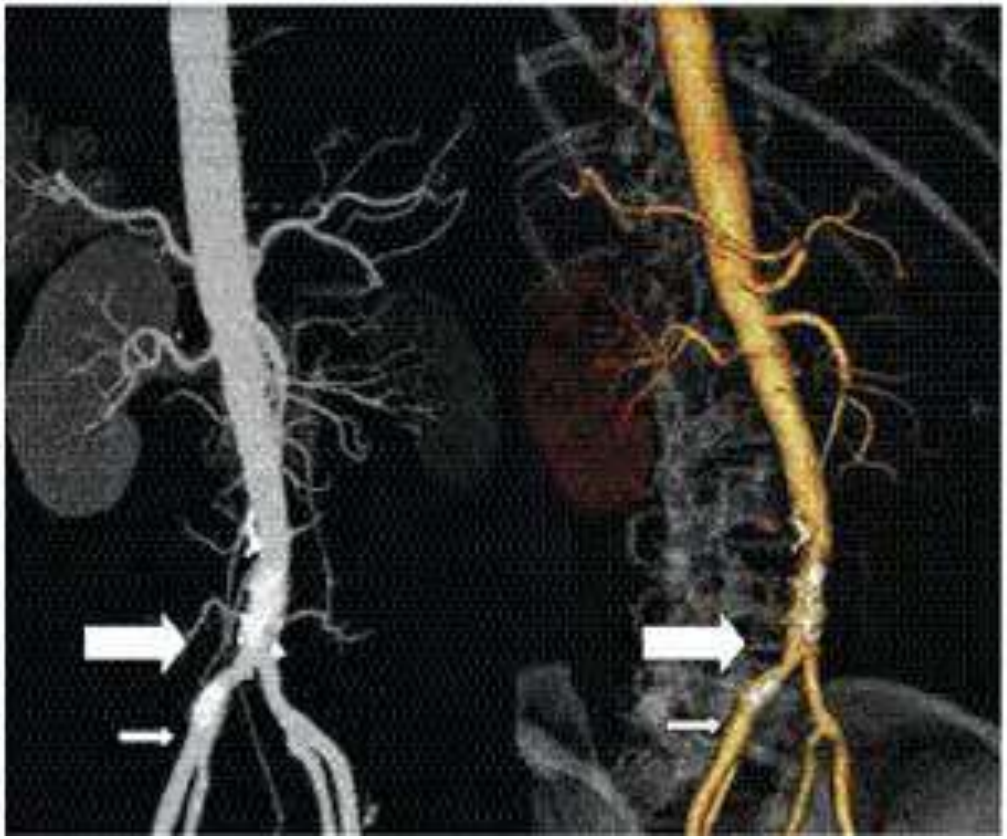
Diagnosis
(Vascular catheterization and angiogram)



Multidetector computed tomography (CT) angiography. Pertinent collateral pathways are developed in the event of chronic severe aortoiliac occlusive disease. The collaterals include epigastric arteries (*large white arrows*), an enlarged inferior mesenteric artery (*white arrowhead*), and enlarged lumbar arteries (*black arrows*).

intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/surgery/5/Topic%2008.%20Arteries%20occlusive%20diseases.Mesenteric%20artery%20diseases.htm

Diagnosis
(Vascular catheterization and angiogram)



Multidetector computed tomography angiography of the aortoiliac artery circulation in a 63-year-old man with buttock claudication. Three-dimensional image reconstruction showing intra-arterial calcification of the aorta (*large arrows*) and right common iliac artery (*small arrows*).

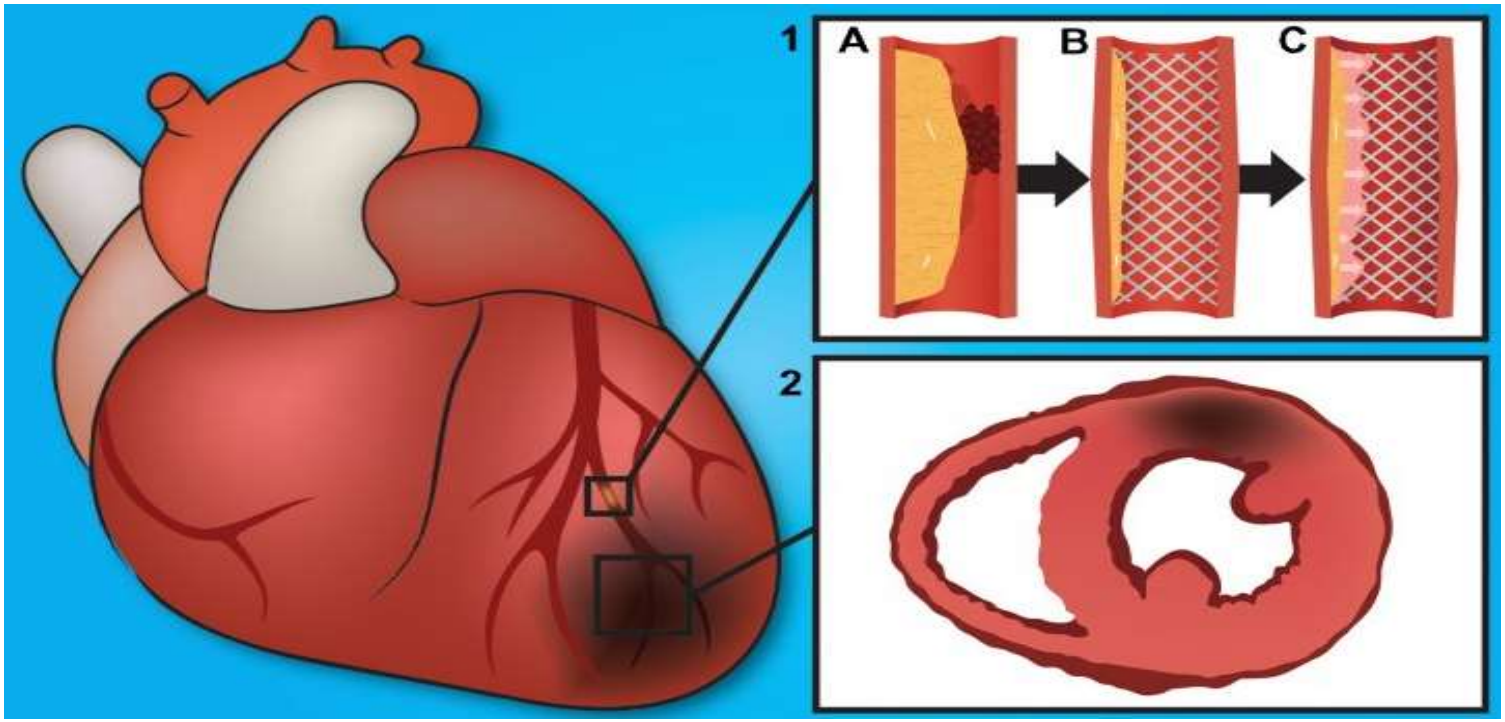
intranet.tdmu.edu.ua/data/kafedra/internal/surgery2/classes_stud/en/med/lik/ptn/surgery/5/Topic%2008.%20Arteries%20occlusive%20diseases.Mesenteric%20artery%20diseases.htm

Treatment
(Key Points)

1. Acute and chronic treatment strategies for acute vascular events are proven to reduce disability/death but access to care is often lacking
2. Interventions to consider: the polypill approach for primary and secondary prevention is a cost-effective and evidence-based
3. Percutaneous interventions with stenting and surgeries (by-pass and endarterectomy) are effective measures for certain scenarios of coronary, cerebral and other artery stenosis and occlusions
4. The most cost-effective management of ASVD is one of an integrated treatment based on a Total Vascular Risk approach

Treatment

(Percutaneous interventions with stenting and surgeries)



(1) Progression of coronary artery disease. (A) Atherosclerosis plaque buildup in the artery and blood clot occluding blood fow; (B) stent compressing the plaque, with widening of the artery; (C) in-stent restenosis decreasing and occluding the blood flow. (2) Myocardial infarction highlighting a localized area of ischemia and dead heart muscle.

researchgate.net/figure/235436182_fig2_1-Pathophysiological-presentations-and-progression-of-coronary-artery-disease-A

Treatment
(Lifestyle Changes)

- Salt restriction
- Moderation of alcohol consumption
- Other dietary changes (vegetables, low-fat dairy products, dietary and soluble fibers, whole grains and protein from plant sources, reduced in saturated fat and cholesterol)
- Weight reduction
- Regular physical exercise
- Smoking cessation

emedicine.medscape.com/article/198475-overview#a3

Treatment
(Education)

- There should be a change in focus in the education of all physicians to more strongly emphasize evidence based primary prevention of ASVD
- Education focused on school children, especially targeted to create sustainable healthy behavior and sustainable control of risk factors
- Telemedicine has been proven effective in selected world regions
- Engage the media to promote education for ASVD prevention, and the need for rapid access to treatment
- Produce easily accessible (through medical journals and internet) guidelines for management of ASVD

Treatment
(Pharmacotherapy)

- Cholesterol medications (statins, fibrates, nicotinic acid (niacin), omega-3 supplementation, bile acid sequestrants)
- Anti-inflammatory medications (selective inhibition of Lp-PLA2 with darapladib)
- Anti-platelet medications (aspirin, clopidogrel, etc.)
- Beta blocker medications (for coronary artery disease)
- Angiotensin-converting enzyme (ACE) inhibitors
- Calcium channel blockers
- Diuretics (for ASVD arterial hypertension)
- Other medications (to control specific risk factors for atherosclerosis, such as diabetes)
- Efferent therapy (enterosorption, hemadsorption, LDL hemadsorption)

mayoclinic.org/diseases-conditions/arteriosclerosis-atherosclerosis/diagnosis-treatment/treatment/txc-20167054

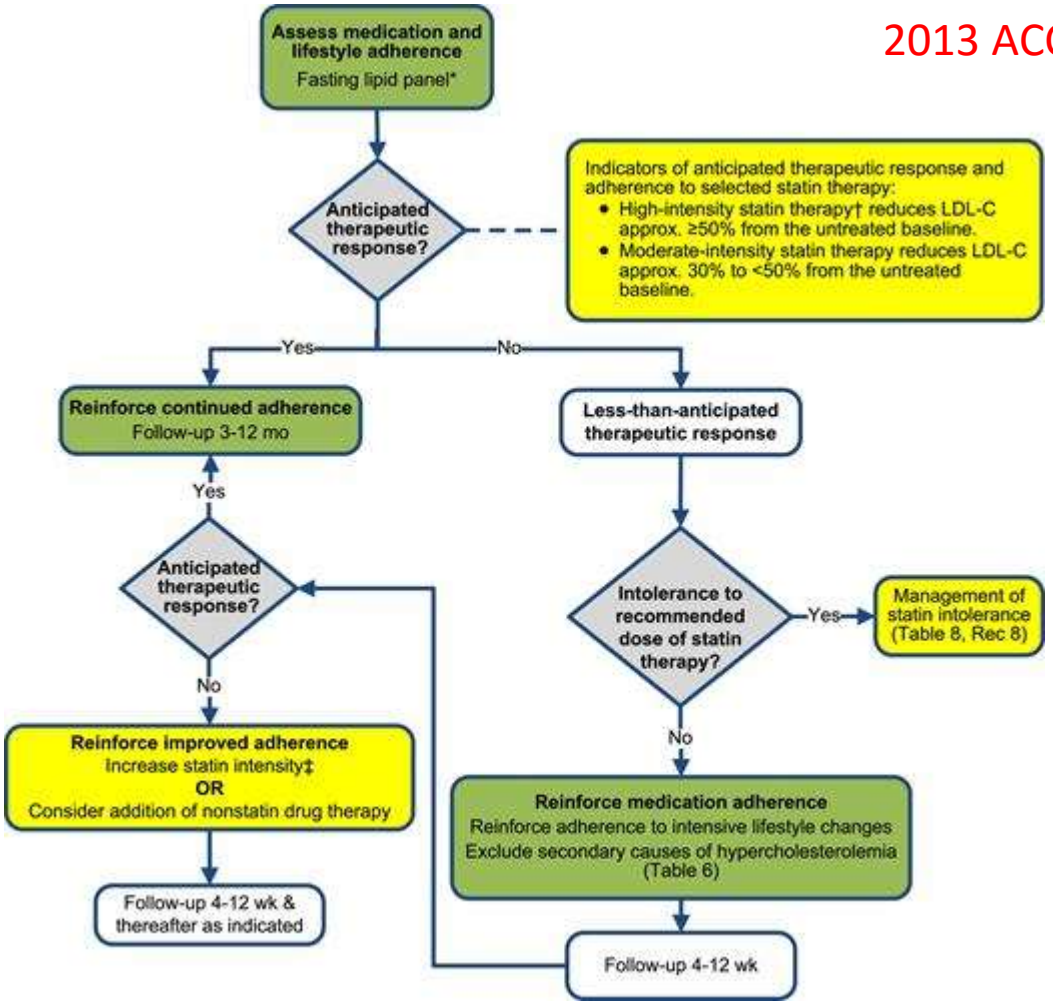
Treatment
(Statins)

- Statins or HMG-CoA reductase inhibitors, are a class of lipid-lowering medications that inhibit the enzyme HMG-CoA reductase which plays a central role in the production of cholesterol
- Statins have been found to reduce ASVD disease and mortality in those who are at high risk
- Side effects of statins include muscle pain, increased risk of diabetes mellitus, and abnormalities in liver enzyme tests
- A number of statins are on the market: atorvastatin, fluvastatin, lovastatin, pitavastatin, pravastatin, rosuvastatin and simvastatin

en.wikipedia.org/wiki/Statin

Treatment
(Statins)

2013 ACC/AHA



Major recommendations for statin therapy for ASCVD prevention

circ.ahajournals.org/content/early/2013/11/11/01.cir.0000437738.63853.7a

Prognosis

- ASVD leads to the number one cause of death around the World for both men and women
- However, people with ASVD are living longer with better quality of life than ever before
- For many, this is disease can be prevented
- Even those people genetically programmed for atherosclerosis can delay the beginning and worsening of the disease with a healthy lifestyle, the right foods, and medication to lower LDL cholesterol

drugs.com/health-guide/atherosclerosis.html

Prophylaxis

- Risk factor assessment and modification should begin early in life (childhood)
- Most risk factors can be modified by lifestyle changes as opposed to drug or device treatment
- Solidarity in primary prevention efforts will benefit both the individual and the society as a whole
- Effective treatment reduces the first clinical manifestations of ASVD
- The role of Governments is crucial
- Aggregate risk factor reduction can decrease cardiovascular events by as much as 70%
- Adequate Secondary prevention measures can reduce vascular event recurrence by 75%

Abbreviations

- ACE - angiotensin-converting enzyme
- ACS - acute coronary syndrome
- ASCVD -arteriosclerotic cardiovascular disease
- ASVD – atherosclerotic vascular disease
- CKD - chronic kidney disease
- CCTA - coronary computed tomography angiography
- CT – computer tomography
- CV - cardiovascular risk
- CVD - cardiovascular disease
- ICD - International Classification of Diseases
- IGT - impaired glucose tolerance
- HDL – high-density lipoprotein
- LDL - low-density lipoprotein
- MRA – magnetic resonance angiogram
- PAD –peripheral artery disease
- SCORE - Systemic Coronary Risk Estimation
- TG -triglycerides
- VLDL - very low-density lipoprotein

Diagnostic and treatment guidelines

Europe

- ESC/EAS Guidelines for the management of dyslipidemias
- Joint 2016 European Guidelines on Cardiovascular Disease Prevention in Clinical Practice

North America

- 2013 ACC/AHA Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults
- Global Recommendations for the Management of Dyslipidemia