Chapter 1

Focus on Coronary Atherosclerosis

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Additional information is available at the end of the chapter

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Abstract

Atherosclerosis is a vascular disorder consisting of thickening of arteries and lack of elasticity. Result of atherosclerosis is that arteries become narrowed and hardened due to an excessive buildup of plaque around the artery wall. The disease disrupts the flow of blood around the body, posing serious cardiovascular complications. Arteries contain what is called an endothelium, a thin layer of cells that keeps the artery smooth and allows blood to flow easily. Endothelial damage starts the first step of atherosclerosis. After this, low-density lipoprotein (LDL) cholesterol accumulates in the artery wall. Inflammatory process starts after this accumulation, and macrophages reach the endothelium to clean up cholesterol. But some macrophages are stuck in the affected part of the artery wall in this process. Over time, this results in plaque being built up, consisting of cholesterol and macrophage white blood cells. The plaque clogs up the artery, disrupting the flow of blood. This potentially causes blood clots that can result in life-threatening conditions such as heart attack and other cardiovascular diseases. Atherosclerosis can be seen in all arteries in the body. Atherosclerosis is the most common cause of death in the western countries. Some risk factors are as follows: age, sex, familial predisposition, hyperlipidemia, hypertension, diabetes mellitus, smoking, obesity, insufficient physical activity, etc. Whatever the main reason or the risk factor is, once atherosclerosis is formed, several life-threatening cardiovascular disorders can be seen. So, it has to be revealed.

Keywords: atherosclerosis, coronary atherosclerosis, coronary artery disease, ischemic heart disease

1. Introduction

Atherosclerosis locution originates from the Greek-Latin word "atera" meaning "oat or milky mush." It describes a vascular disorder consisting of thickening of arteries and lack of elasticity. The general pathology refers to three situations:

The predominant type is often seen with intimal fatty plaque formation including central lipid-rich core. We want to mention about this type of atherosclerosis.

The second morphologic type of atherosclerosis, Mönckeberg's medial calcific sclerosis, is seen in the muscular arteries with a medial calcification. This form is not prominent as classic atherosclerosis and mostly seen after the age of 50. This can be seen radiologically and can be felt with palpation.

The third type is the disorders of the small arteries and arterioles, named arteriosclerosis. This is seen mostly with hypertensive and diabetic patients. This refers to stiffening or hardening of the artery walls.

The final result of atherosclerosis is that arteries become narrowed and hardened due to an excessive buildup of plaque around the artery wall. The disease disrupts the flow of blood around the body, posing serious cardiovascular complications.

Atherosclerosis can be seen in all arteries in the body. But we will instruct about the coronary atherosclerosis. Atherosclerosis is the most common cause of death in the western countries [1].

Arteries contain what is called an endothelium, a thin layer of cells that keeps the artery smooth and allows blood to flow easily. Endothelial damage starts the first step of atherosclerosis. After this LDL cholesterol accumulates in the artery wall. Inflammatory process starts after this accumulation, and macrophages reach the endothelium to clean up cholesterol. But some macrophages stuck in the affected part of the artery wall in this process. Over time this results in plaque being built up, consisting of cholesterol and macrophage white blood cells.

The plaque clogs up the artery, disrupting the flow of blood. This potentially causes blood clots that can result in life-threatening conditions such as *heart attack* and other cardiovascular diseases.

Some risk factors are follows: age, sex, familial predisposition, hyperlipidemia, hypertension, diabetes mellitus, smoking, obesity, insufficient physical activity, etc. Whatever the main reason or the risk factor is, once atherosclerosis is formed, several life-threatening cardiovascular disorders can be seen. So, it has to be revealed.

2. Background

The role of the circulation is to service the needs of the tissues. It includes transporting nutrients to thecells of the body and waste products away from the cells of the body. And finally transporting hormones from one part of the body to another. Naturally, circulation maintains an appropriate environment in all the tissue fluids of the body for optimal survival and function of the cells.

Systemic circulation, pulmonary circulation, peripheral circulation, etc. have some details to maintain the blood flow. The arteries are the large conductive vessels that transport blood

under high pressure to the tissues. The arterioles are the last small branches of the arterial system. And, the capillaries are where the exchange of fluid, electrolytes, nutrients, hormones, and other substances occurs. And, of course veins, venules, collect blood from the capillaries. Our main subject is atherosclerosis of arterial vessels.

We have to learn the normal state before discussing the pathological status. The normal artery wall is consisting of intima, media, and adventitia. Lumen is lined by a monolayer of endothelial cells that overlies smooth muscle cells. The inner layer of smooth muscle cells, known as the intima, is circumscribed by the internal elastic lamina. The media layer is between the internal elastic lamina and external elastic lamina. The media is another layer of smooth muscle cells. Outside the external elastic lamina is an adventitia part that is rarely populated by cells and microvessels of the vasa vasorum.

Atherosclerosis is a progressive disease of medium- and large-sized arteries characterized by focal intimal lesions called atheromas or atherosclerotic plaques that protrude into vessel lumen and eventually leading to various complications [2]. There are several diseases led by atherosclerosis: coronary artery disease, peripheral artery disease, and carotid artery disease. These are real threats for mortality and morbidity in the developed countries.

3. Pathophysiology

Atherosclerosis is a chronic, inflammatory, fibroproliferative disease of medium- and large-sized arteries [3]. There are different stages to form the atherosclerotic plaque. The initiation phase is the beginning and the progression of the plaque and the final complication stage.

Chronic or recurrent endothelial damage is the cornerstone of the "response to damage" hypothesis. Hyperlipidemia, hypertension, smoking, immunoreactions, hemodynamic factors, toxins, and viruses can cause this chronic endothelial damage. Hemodynamic deformities such as endothelial shear stress, turbulent flow, or unfavorable effects of hypercholesterolemia have a role in the initiation phase. Due to endothelial damage and turbulent flow, endothelial permeability, cell regeneration, and receptor-mediated LDL endocytosis and leukocyte adhesion to endothelium increase.

Hyperlipidemia has an important role in the atherogenesis [4]. Chronic hyperlipidemia especially hypercholesterolemia can start the endothelial damage. After all, lipoproteins accumulate in these damaged endothelial sites. The cellular response after endothelial damage continues with increased permeability, leukocyte adhesion, monocyte migration, and increased adhesion. This is no longer the initiation phase after this stage; progression has started.

Some cellular events take an important part in this phase. Smooth muscles migrate from the media layer to intima and macrophages activated. Monocytes turn to macrophages. Activated macrophages, and smooth muscle cells absorb lipids. Modified lipid molecules due to the oxidative mechanisms of modified LDL arise. Oxide LDL makes some additive affects, in order; (a) with the help of altered receptors LDL absorbed easily by macrophages (b) they

are chemotactic to circulating monocytes (c) they enhance adhesion of monocytes (d) they prevent the mobility of macrophages because of this macrophages remain their position and hold on to there (e) they are cytotoxic to endothelium and smooth muscle cells (f) they are immunogenic.

The endothelial damage is like as peeling of the endothelium, because of this damage platelets hold on to the endothelium. Smooth muscle cells derived from media layer migrate to here and starts to duplicate and some of them absorb lipids inside and turns to the foam like cells. And this is shown as fatty streaks.

After this stage macrophages take a leading role in atherosclerosis. Macrophages secrete interleukin-1 (IL-1) and tumor necrosis factor (TNF), and they are increasing leukocyte adhesion. Again, monocyte chemoattractant protein-1 (MCP-1) produced by macrophages collects leukocytes in the plaque. They have a role to oxidate the LDL. And finally, they secrete stimulators to affecting the smooth muscle cell growth [5].

Fatty streaks are seen in the childhood phase. This lesion starts as a small 1-mm-diameter intimal color change. With the organization of atherosclerosis, this lesion varies 1–3 mm in diameter and 1–2 cm long. Some of them are raised and some of them not.

Atheromatous plaque is the definitive lesion, and it is rich in lipids, but more often it is a lipid and fibrotic lesion. Sometimes, this solid and fibrotic characterized plaque can be rich with cells. Plaques' diameter can reach to a few cm. Its color changes according to the amount of the lipid. It is changed to a round shape and has an irregular shape.

Atheromatous plaques can be seen in the different parts of the body. The prevelence of involvement is in order; Abdominal aorta, coronary arteries, popliteal arteries, descending thoracic aorta, internal carotid arteries and the circle of Willis.

Finally, microscopically atherosclerotic plaque has got the main components. These are lipids, vascular smooth cells, monocytes/macrophages, rarely lymphocytes, connective tissue matrix, and fibrils (**Figure 1**).

But more importantly, atherosclerotic plaque changes to four different types. These are complicated plaques:

a. Calcification of the arteries. They can be seen as a consecutive island, and some of them can be in the whole artery.

b. Ulceration of the surface of the atherosclerotic plaque. This can cause embolization.

c. Platelet aggregation can occur on the ulcerated plaque. This can lead to total occlusion of the artery. The most devastating effect of atherosclerosis such as heart attack and stroke is caused by the superimposed thrombosis.

d. Atherosclerotic aneurysm can occur due to atherosclerosis.

e. This ulceration can break endothelial integrity, and this can causes rupture of the plaque and can cause bleeding.

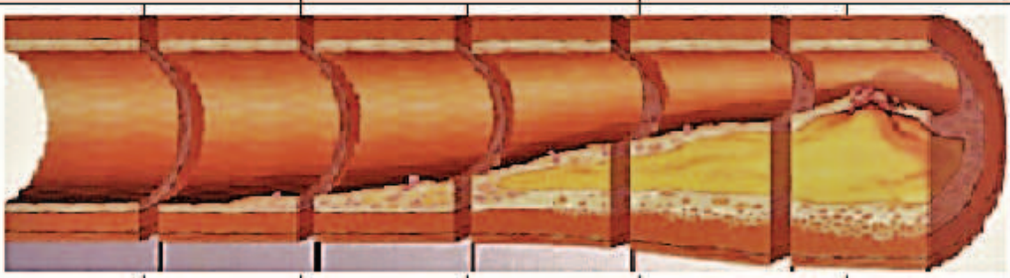
Clinical findings	Asymptomatic			Asymptomatic or Symptomatic		
Growth mechanism	Growth is mainly with lipid deposition				Proliferation of smooth muscle cells and increase of collagen	Thrombosis and /or hematoma
Onset of time	From first decade		From Third decade		From fourth decade	
Phases of progression of Atherosclerosis						
Main Histology of the progression	***First lesion***: -Normal Histology - Macrophage migration -Isolated foam cells	***Fatty Streak:*** -Mainly intracellular lipid deposition	***Intermediate phases:*** -New fatty streaks -Intracellular Lipid deposition and lipid pools	***Atheroma:*** -New fatty streaks -Intracellular and Extracellular lipid accumulation	***Fibroatheroma:*** -New fatty streaks -Single or multiple lipid cores -Fibrotic and calcific layers	***Complicated Lesions:*** -Disrupted surface (ulcerated plaque) -Thrombosis -Hematoma and hemorrhage

Figure 1. The stages of the progression of atherosclerosis.

4. Epidemiology and etiology

Due to the asymptomatic phase of atherosclerosis, it is impossible to say the frequency of atherosclerosis, because the process of atherosclerosis starts with fatty streak in the first decade of lifetime. More advanced lesions begin to develop when individuals are in their second and third decade. Complicated coronary atherosclerosis causes coronary artery disease (CAD) after all. CAD remains the most common pathology with which cardiologists and cardiac surgeons are facing. It is the most common cause of death in Turkey in 2013 [6]; 38.8% of the deaths were due to the ischemic cardiovascular disease. Ischemic heart disease is the most common cause of death in the world as reported by the World Health Organization (WHO) in 2012 [7].

Inactivation of genes coding for monocyte chemotactic protein-1 (MCP-1), its receptor on monocyte/macrophages (CCR2), and macrophage colony-stimulating factor has a profound impact on the development of atherosclerosis in otherwise identical mice that have been shown in the experimental studies [8]. The etiology of atherosclerosis is unknown, but in the development process of atherosclerosis, the pathophysiology is important to explain the nature. There are some important risk factors in this process. We have to classify risk factors in two. These are modifiable and non-modifiable risk factors.

5. Risk factors

5.1. Non-modifiable risk factors

a. Increased age.

b. Male gender: lack of atheroprotective properties of estrogen which raises HDL and lowers LDL.

c. Hereditary factors: history of coronary artery disease (CAD) among first-degree relatives at a young age (before 55 for males and before 65 for females). New markers of the cardiovascular risk factors:

d. Increased lipoprotein(a) level.

e. Increased homocysteine level: high levels may promote oxidative stress, vascular inflammation, and platelet adhesiveness. And, this process leads to atherosclerosis. A meta-analysis that collected a large number of prospective studies showed a significant association between the serum level of homocysteine and the incidence of cardiovascular disease [9]. Not just with it, increased blood homocysteine levels are shown in patients with acute myocardial infarction [10].

f. C-reactive protein (CRP), high-sensitivity CRP (hs-CRP), and other markers of inflammation: activate complement and contribute to a sustained inflammatory state. CRP is a biomarker of tissue damage and inflammation. It is an acute-phase reactant and increases in the inflammatory process. But nowadays, it has been used in the diagnosis of the cardiovascular diseases such as CAD. Sara et al. have showed that hs-CRP is associated with coronary endothelial dysfunction in the asymptomatic coronary artery disease [11].

5.2. Modifiable risk factors

a. Dyslipidemia: increased LDL and decreased high-density lipoprotein (HDL).

b. Tobacco smoking: enhances oxidative modification of LDL, contributes to endothelial dysfunction via oxidant stress, and increases expression of leukocyte adhesion molecules.

c. Hypertension: increases permeability of the vessel wall to lipoproteins and promotes retention of LDL in the vessel intima by accentuating production of LDL-binding proteoglycans by smooth muscle cells.

d. Diabetes mellitus: enhances glycation of LDL and is associated with endothelial dysfunction.

e. Obesity and lack of physical activity: can cause dyslipidemia, hypertension, and insulin resistance.

f. Stressful lifestyle: better known as Type A personality.

5.3. Atheroprotective factors

a. Exercise

b. High-density lipoprotein (HDL) and its major apolipoprotein (ApoA1)

Coronary atherosclerosis is an important site of atherosclerosis. There are various types of results due to coronary atherosclerosis. Especially, the size of the plaque and the type of the complicated plaque are important for this. Whatever the beginning of the atheromatous plaque, the result can be a fatal heart attack. In developed countries atherosclerosis causes more than half of total mortality. Coronary artery disease (CAD) is responsible for a major proportion of these deaths [12].

6. Signs and symptoms

Onset of the atherosclerotic plaque and speed of the growth and complications, there are several signs and symptoms. Atherosclerosis can be seen in every artery, but for the coronary atherosclerosis, the result of the disease is coronary artery disease, and the symptoms and the signs are due to this. Because of the impaired blood flow, there is a sort of symptoms. Some of them are in the side of the chest, and some of them are systemic because of the impaired circulation.

- Chest pain
- Shortness of breath
- Weakness, tiredness, reduced exertional capacity
- Dizziness, palpitations
- Leg swelling
- Weight gain
- Diaphoresis
- Tachycardia: common in persons with acute coronary syndrome (ACS) and acute myocardial infarction (AMI)
- High or low blood pressure
- S_4 gallop: a common early finding
- S_3 gallop: an indication of reduced left ventricular function
- Heart murmurs
- Tachypnea
- Xanthelasmas
- Livedo reticularis
- Syncope
- Leg edema
- Rales

Coronary atherosclerosis causes coronary artery disease. Complicated atherosclerotic plaque disrupts the blood flow in the coronary circulation. Impaired blood flow causes a corrupted supply and demand of the oxygen and the metabolites in the heart. This results in a decrease in coronary arterial blood flow and a decrease in oxygen supply. There are several symptoms such as chest pain (angina pectoris), dyspnea, syncope, and sometimes pulmonary edema. Increased demand of blood supply and oxygen starts the angina pectoris. Because of the decreased blood flow in coronary artery, sufficient blood cannot be supplied in the increased effort capacity. The spectrum of presentation includes symptoms and signs consistent with the following conditions:

- Asymptomatic state (subclinical phase)
- Stable angina pectoris
- Unstable angina (i.e., acute coronary syndrome)
- Acute myocardial infarction (AMI)
- Chronic ischemic cardiomyopathy
- Congestive heart failure
- Sudden cardiac arrest

7. Diagnosis and treatment

Atherosclerosis can be seen in all the arterial sites in the whole body as mentioned before. So, the physical examination can give us very important findings. A well-taken medical history and physical examination can be helpful for the diagnosis. Suspicious findings can lead us to make a decision for the advanced examination.

Medical history is the cornerstone of diagnosis. A positive history of typical chest pain, shortness of breath, impaired physical capacity, and the other signs and symptoms are very useful to diagnosis.

Atherosclerosis can cause both coronary artery and peripheral artery diseases. Concomitant coronary and peripheral artery disease prevalence is varied 28–94% in published reports [13]. So, on the calcified peripheral artery, palpation or lack of pulse in the peripheral arteries or signs of the peripheral artery disease are important parts of the physical examination.

What are the parts of the advanced examination?

Electrocardiography (ECG): impaired blood flow in acute events such as acute myocardial infarction and acute coronary syndromes are the changes we can see in the ECG.

Echocardiography (ECO): atherosclerotic calcification or plaque and thickness of aortic wall can be seen in ECO. Ventricular low ejection fraction and impaired contraction of ventricular segments can suspect us for coronary atherosclerosis.

Stress echocardiography: this echocardiography can be performed either by exercise method or pharmacological drugs that increase cardiac contractility and rate.

Exercise echocardiography: images are taken before and after the treadmill or stationary bike effort test. If exercise echocardiography cannot be performed due to peripheral artery disease, musculoskeletal disorders, etc., drug-stimulated (dobutamine, adenosine, dipyridamole) stress echocardiography can be performed. These drugs increase the cardiac contractility and rhythm. Doses of the drugs increases step by step, and images are taken gradually.

The purpose is to assess the exercise tolerance of the heart. If there is a myocardial perfusion defect due to coronary artery disease, stress echocardiography can give information about this. The severity of the coronary artery disease can be assessed with this test. Before and after revascularization either PCI or CABG cardiac risk can be evaluated. It can be performed for cardiac risk analyses for noncardiac surgeries. Exercise echocardiography can be used for risk stratification in asymptomatic patients with severe aortic stenosis too [14]. Yao et al. have showed in their clinical study; as the result of the exercise tests, monophasic/normal wall motion was associated with a benign prognosis, but abnormal wall motion responses were associated with a worse prognosis [15].

Myocardial perfusion scintigraphy: can show us the ischemic parts of the heart due to the occlusive effect of coronary atherosclerosis leading to coronary artery disease.

Computed tomography (CT): conventional thoracoabdominal CT scan can show atherosclerotic calcification and plaques in the aortic or arterial wall. But coronary CT scan can show us the presence of coronary atherosclerosis, the degree of the coronary artery disease, and the occlusive lesions.

Intravenous ultrasound (IVUS): can be useful for the controversial lesions. This is an invasive technique that localizes plaques and quantifies plaque seriousness. Virtual histology-intravascular ultrasound (VH-IVUS) can identify plaque components. Optical coherence tomography (OCT), also known as optical frequency domain imaging (OFDI), identifies intimal hyperplasia and also detects and quantifies the key features of vulnerable plaque [16].

Coronary angiography: is the gold standard for diagnosis of coronary atherosclerosis and coronary artery disease. Moving image of each coronary artery and the atherosclerotic lesions can be seen. It is the most specific and sensitive test for the diagnosis of coronary artery disease.

Coronary angiography can come out to such results. This can be a follow-up with medical therapy, a percutaneous coronary intervention can be necessary, or a coronary artery bypass grafting (CABG) is essential to be performed to the patient. All of the interventions are selected due to the percentage of the affected coronary artery lesion, the lesion type, lesion location, the number of the affected coronary artery, and of course the general condition of the patient.

Treatment: There are several treatment modalities. These include lifestyle changes, risk factor modification, and medical therapies. But we want to mention about the clinically important occlusive coronary artery diseases' invasive treatment.

Percutaneous coronary intervention (PCI): it is also known as coronary angioplasty, and this is a nonsurgical technique to treat obstructive coronary artery disease. It can be a choice in stable angina pectoris, in acute myocardial infarction, or in multivessel coronary artery disease. The procedure is performed in angiography catheter laboratory. An x-ray fluoroscopy and opaque fluid are necessary for the procedure. Entry ways for the procedure are femoral arteries and radial arteries for the individual cases.

Some urgent cases such as acute myocardial infarction PCI can be performed emergent. Primary PCI is called in this situation. But also PCI is used for elective coronary artery disease usually. The procedure starts with a local anesthesia from the arterial puncture side; this can be even femoral or radial artery. Hydrophilic and micro-catheters and guidewires are used to reach coronary arteries. These radiopaque wires are seen easily on fluoroscopy. A balloon angioplasty can be performed to the occlusive lesion. Coronary stents can be implanted to the occluded lesion. Coronary stents vary from bare metal stents to drug eluding stent. These drug eluding stents vary to the first, second, and third generation. Nowadays, fourth-generation bioresorbable stents are mentioned in some clinical trials [17]. Whatever the kind of the stents, the main purpose is to improve blood flow of the myocardium tissue.

Coronary artery bypass grafting (CABG): so, is this the only technique that we can improve blood supply of the myocardium? Is there any other way of myocardial revascularization? The answer is yes. It is coronary artery bypass grafting (CABG). This is an open cardiac surgical procedure. This means that it is more invasive than PCI. But in some cases, PCI cannot be the concluding treatment for the coronary artery disease. Lesion type, region of the lesion, collateral and main side branches extending from the lesion, severity of the lesion, and the number of the lesions is important for the physician to make the choice.

Before explaining CABG, we have to mention the indications and guidelines (**Table 1** and **2**).

7.1. Guidelines for coronary artery bypass graft surgery

7.1.1. Asymptomatic CAD

7.1.1.1. Class I

1. LMC stenosis.[18, 20]

2. LMCE disease.

3 Three-vessel disease.

Revascularization	CABG			DES		
	No risk	DM	LVD	No risk	DM	LVD
One-vessel disease	N	N	N	Y	Y	Y
Proximal LAD	Y	Y	Y	N	N	N
Two-vessel disease without LAD	N	N	N	Y	Y	Y
Two-vessel disease with LAD	Y	Y	Y	Y	Y	Y
Two-vessel disease + proximal LAD	Y	Y	Y	N	N	N
Three-vessel disease	Y	Y	Y	C	C	C
Three-vessel + proximal LAD	Y	Y	Y	N	N	N
LMC ± other lesions	Y	Y	Y	N	N	N

CABG, coronary artery bypass grafting; DES, drug-eluting stent; DM, diabetes mellitus; LAD, left anterior descending artery; LMC, left main coronary artery disease; LVD, left ventricular dysfunction. *Y, yes; N, no; C, controversial.

Table 1. The reality of myocardial revascularization strategies in patients with isolated coronary artery disease [18].

Recommendation	CABG		PCI	
	Class	Level	Class	Level
One or two-vessel disease without LAD	IIb	C	I	C
One-vessel disease with proximal LAD	I	A	I	A
Two-vessel disease with proximal LAD	I	B	I	C
LMC with SYNTAX score < 22	I	B	I	B
LMC with SYNTAX score 23–32	I	B	IIb	B
LMC with SYNTAX score > 32	I	B	III	B
Three-vessel disease SYNTAX score > 22	I	A	I	B
Three-vessel disease SYNTAX score 23–32	I	A	III	B
Three-vessel disease SYNTAX score > 32	I	A	III	B

Recommendation for the type of revascularization (CABG or PCI) in patients with stable CAD with suitable coronary anatomy for both procedures [19].

Table 2. 2014 ESC/EACTS Guidelines on myocardial revascularization guidelines.

7.1.1.2. Class IIa

(1) Proximal LAD (one- or two-vessel disease)

7.1.1.3. Class IIb

(1) One- or two-vessel disease not involving proximal LAD (if a large territory at risk on non-invasive studies or LVEF <50%, class IIa and IIb become class I indications)

7.1.2. Stable angina

7.1.2.1. Class I

1. LMC stenosis.

2. LMCE disease.

3. Three-vessel disease.

4. Two-vessel disease with proximal LAD stenosis and LVEF <50% or demonstrable ischemia.

5. One- or two-vessel disease without proximal LAD stenosis but with a large territory at risk and high-risk criteria on noninvasive testing.

6. Disabling angina refractory to medical therapy.

7.1.2.2. Class IIa

1. Proximal LAD stenosis with one-vessel disease.

2. One- or two-vessel disease without proximal LAD stenosis, but with a moderate territory at risk and demonstrable ischemia.

7.1.3. Unstable angina/non-ST-segment elevation MI (non-STEMI)

7.1.3.1. Class I

1. LMC stenosis.

2. LMCE disease.

3. Ongoing ischemia not responsive to maximal nonsurgical therapy.

7.1.3.2. Class IIa

Proximal LAD stenosis with one- or two-vessel disease.

7.1.3.3. Class IIb

One- or two-vessel disease without proximal LAD stenosis when PCI not possible (becomes class I if high-risk criteria on noninvasive testing).

7.1.4. ST-segment elevation (Q wave) MI

7.1.4.1. Class I

1. Failed PCI with persistent pain or shock and anatomically feasible.

2. Persistent or recurrent ischemia refractory to medical treatment with acceptable anatomy, which has a significant territory at risk and not a candidate for PCI.

3. Requires surgical repair of post-infarct VSD or MR.

4. Cardiogenic shock in patients <75 years of age who have ST elevation, LBBB, or a posterior MI within 18 hours onset.

5. Life-threatening ventricular arrhythmias in the presence of ≥50% LMC stenosis or three-vessel disease.

7.1.4.2. Class IIa

1. Primary reperfusion in patients who have failed fibrinolytics or PCI and are in the early stages (6–12 h) of an evolving STEMI.

2. Mortality with CABG is elevated in the first 3–7 days after STEMI/NSTEMI. After 7 days, criteria for CABG in previous section are applied.

7.1.5. Poor LV function

7.1.5.1. Class I

1. LMC.

2. LMCE.

3. Proximal LAD stenosis and two- to three-vessel disease.

7.1.5.2. Class IIa

Significant viable territory and noncontractile myocardium.

7.1.6. Life-threatening ventricular arrhythmias

7.1.6.1. Class I

1. LMC.

2. Three-vessel disease.

7.1.6.2. Class IIa

1. Bypassable one- or two-vessel disease.

2. Proximal LAD disease and one- or two-vessel disease. These become class I indications if arrhythmia is resuscitated cardiac death or sustained ventricular tachycardia.

7.1.7. Failed PCI

7.1.7.1. Class I

1. Ongoing ischemia with significant territory at risk.

2. Shock.

7.1.7.2. Class IIa

1. Foreign body in critical position.

2. Shock with coagulopathy and no previous sternotomy.

7.1.7.3. Class IIb

Shock with coagulopathy and previous sternotomy.

7.1.8. Previous CABG

7.1.8.1. Class I

1. Disabling angina refractory to medical therapy.

2. Nonpatent previous bypass grafts, but with class I indications for native CAD.

7.1.8.2. Class IIa

1. Large territory at risk.

2. Vein grafts supplying LAD or large territory are "/> 50% stenosed.

Class I: Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective.

Class II: Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness or efficacy of a procedure.

Class IIa: Weight of evidence/opinion is in favor of usefulness/efficacy.

Class IIb: Usefulness/efficacy is less well established by evidence/opinion.

Class III: Conditions for which there is evidence and/or general agreement that the procedure/treatment is not useful/effective and in some cases may be harmful.

ACC, American College of Cardiology; AHA, American Heart Association; CABG, coronary artery bypass grafting; CAD, coronary artery disease; LAD, left anterior descending artery; LBBB, left bundle branch block, LMC, left main coronary artery; LMCE, left main coronary equivalent; LVEF, left ventricular ejection fraction; MI, myocardial infarction; MR, mitral regurgitation; NSTEMI, non-ST elevation myocardial infarction; PCI, percutaneous transluminal coronary angioplasty; STEMI, ST elevation myocardial infarction; VSD, ventricular septal defect [20].

There are several grafts that are used in CABG. Arterial grafts such as left internal and right internal mammary artery (LIMA and RIMA), especially LIMA has got the longest patency rate (10-year patency is 95%). Radial artery can be used, but it is a muscular artery and has got a predisposition to vasospasm. Vena saphena magna is the most used venous graft.

This procedure can be performed with cardiopulmonary bypass (CPB) machine (on-pump), without CPB (off-pump CABG-OPCAB), or beating heart procedures. In the last decade, minimally invasive techniques are rising to individual cases. MIDCAB (minimally invasive direct coronary artery bypass) can be performed without full median sternotomy. This can serve minimal surgical trauma and avoid wound complication.

TECAB: this is a robotically assisted total endoscopic coronary artery bypass procedure. This is a complex procedure; surgeon has to steep a learning curve. This procedure can perform both on-pump and off-pump CABG.

Awake coronary artery bypass (ACAB) procedure: This avoids side effects of general anesthesia. This includes a minimal invasive procedure without intubation and mechanical ventilatory support. A somatosensory and motor block is made via the T1–T8 level of vertebra. This preserves diaphragmatic ventilation.

Early outcomes after CABG continue to improve, and the early cumulative mortality rate is below 2% and lower than 1% in lower-risk patients. The most common reasons for death are heart failure (65%), neurologic events (7.5%), hemorrhage (7%), respiratory failure (5.5%), and dysrhythmia (5.5%).

The survival rate after isolated CABG is higher than 98% for the first month and 97% for first year, 92% for 5 years, 80% for 10 years, 65% for 15 years, and 51% for 20 years. Usage of LIMA is a predictive parameter for late survival.

8. Prevention

Prevention of the coronary atherosclerosis has to be lifelong. Individuals need to be careful for risk factors. Adopting a healthy lifestyle. What is inside of this healthy lifestyle?

Healthy eating habit for the heart: eating habits are very important in the process of developing atherosclerosis. Healthy diet consists of low amounts of white bread, unsaturated fat products, fast foods, salt, and sugar. It also includes eating dairy products, fruits, vegetables, whole grain, seafood, poultry without skin, lean meats, low-fat milk, or fat-free milk.

After the start of healthy diet for the heart, weight control can be achieved, because overweight and obese people have high risk for coronary atherosclerosis.

Physical activity: stressful and sedentary lifestyles are the risk factors for coronary atherosclerosis. So, a programmed physical activity can improve the fitness level and the health of the individuals.

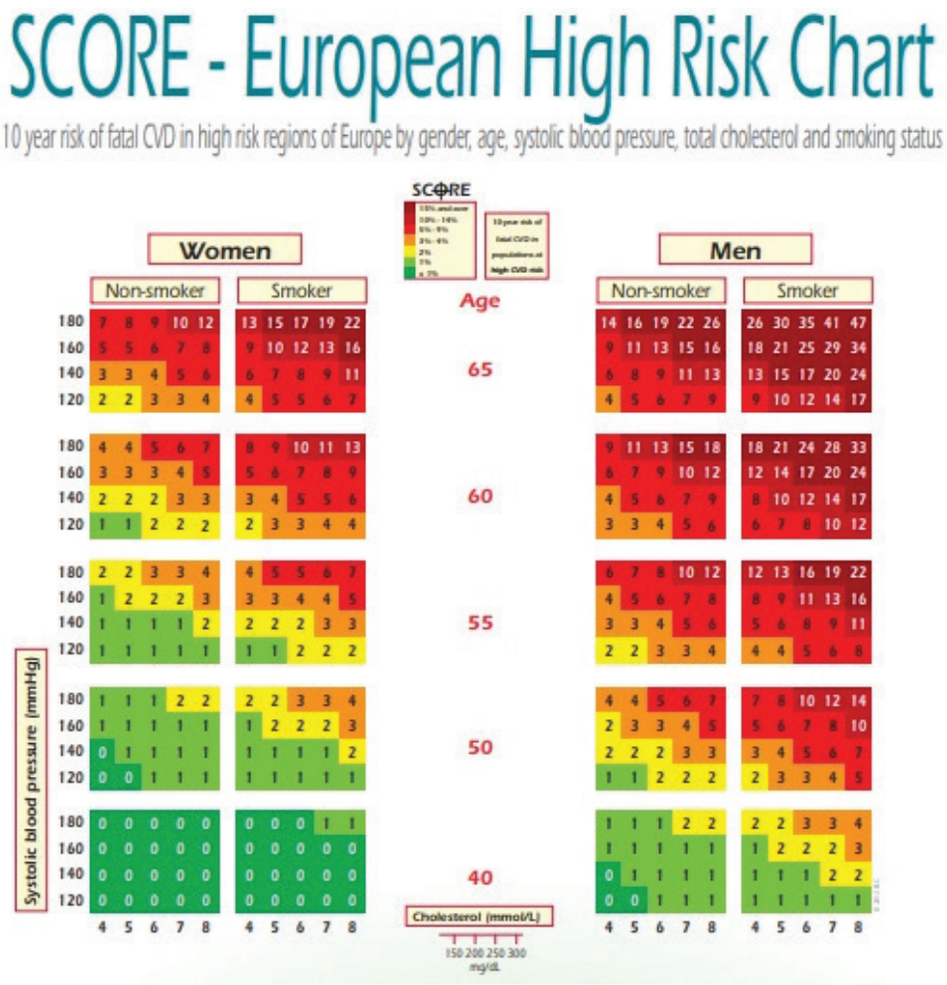


Figure 2. Ten year risk of fatal CVD in high risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status.

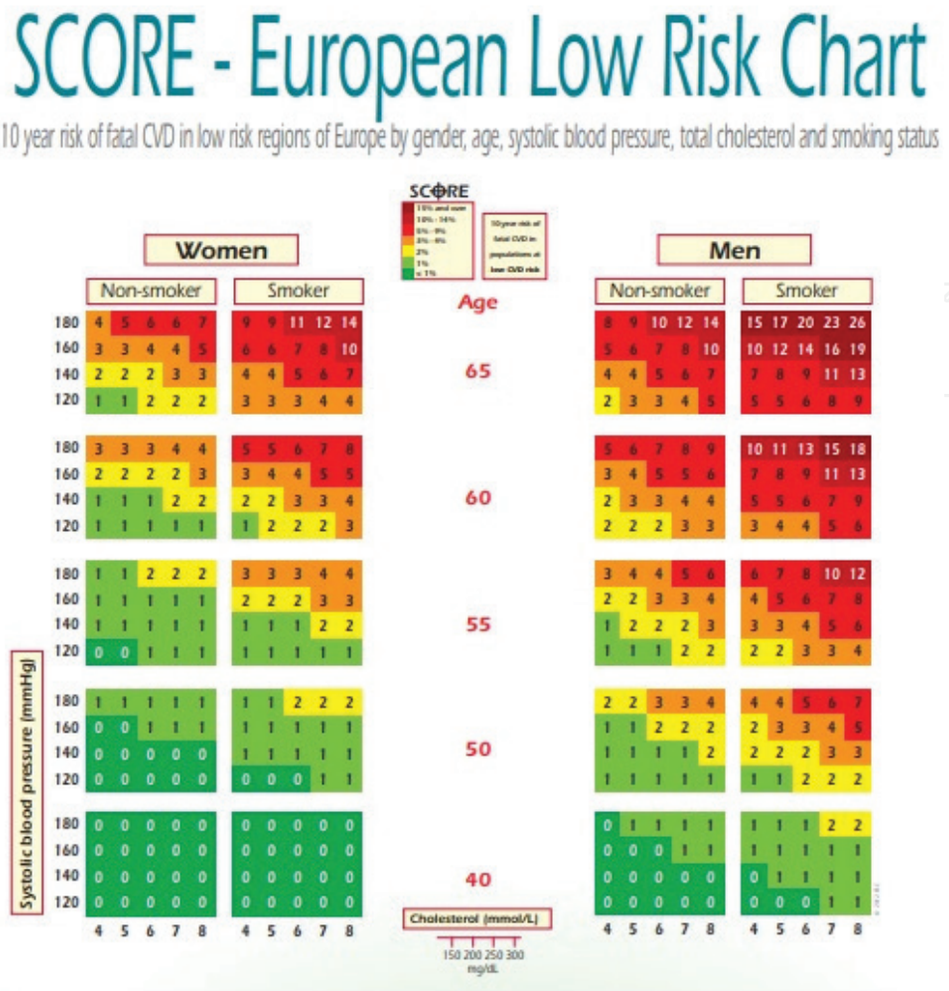


Figure 3. Ten year risk of fatal CVD in low risk regions of Europe by gender, age, systolic blood pressure, total cholesterol and smoking status.

8.1. Stopping tobacco smoking

We have to mention about risk scores and charts. Risk scores can give us several information about the cardiac risk of our body. This can lead the person to change avoidable habits.

Framingham Risk Score: age, sex, cigarette smoking, cholesterol level, high-density lipoprotein (HDL) cholesterol level, systolic blood pressure, and usage of antihypertensive drugs. Some clinics include diabetes mellitus (DM), low-density lipoprotein (LDL) cholesterol, and diastolic blood pressure to modify this risk score.

Another risk score system is SCORE risk charts. This includes SCORE—European High Risk Chart and SCORE—European Low Risk Chart. This score system is based on gender, age, total cholesterol, systolic blood pressure, and cigarette smoking (**Figures 2** and **3**).

9. Conclusion

Coronary atherosclerosis and coronary artery disease (CAD) are the most frequent causes of hospitalization in western countries. It is an important mortality and morbidity cause. The

onset of the first lesions begins in the first decade of the life period and proceeds with the lifetime. Risk factors are important and decisive for the progression of the atheromatous plague. A healthy and modified life is the key to prevent from the disease.

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