CORONARY ARTERY DISEASES

It has been estimated that over one third of the population eventually will die of CAD, and 20% will develop symptoms when younger than age 60 years.

ANATOMY OF THE CORONARY ARTERIES

The first branches of the aorta are the right and left coronary arteries and arise from the sinuses of Valsalva.
The right coronary artery It gives

- **the sinus node artery**
- **right marginal artery**
- **posterior descending artery** and, by an extension to the crux, branches into an atrioventricular node artery and several terminal **posterolateral left ventricular branches(PLVs)**.

The left main coronary artery arises from the left posterior coronary sinus and averages 2 cm in length, although this may vary from 1 mm to 4 cm. it bifurcates into the **left anterior descending (LAD)** and **left circumflex coronary arteries**. In many instances, rather than bifurcating, a trifurcation occurs when the **ramus medianus** vessel originates between the anterior descending and the circumflex arteries.

NORMAL PHYSIOLOGY

Normal coronary blood flow approximates 0.7 to 0.9 ml. per gm (averages 225ml/min.). of myocardium per minute and delivers 0.1 ml. of oxygen per gm. per minute to the heart; The extraction of oxygen in the coronary bed is high, averaging 75% under normal conditions and increasing to nearly 100% during stress. Coronary artery blood flow occurs primarily during diastole because systolic myocardial contraction increases intramyocardial vascular resistance.

Factors influencing coronary vascular resistance

❶ metabolic

Factors that increase coronary vascular resistance

- α- sympathetic receptors
- Myogenic mechanism

Factors that decrease coronary vascular resistance

- β- sympathetic receptors
- Myogenic mechanism
- CO2
- Decreased O2 tension
- Hydrogen ions
- Lactate
- Potassium ions
- Adenosin

❷ physical

- Systolic compression
- Aortic pressure: the coronary vasculature can compensate and maintain normal coronary perfusion pressure between systolic pressure of 60 and 180 mm Hg via the process of autoregulation.

❸ Neural and hormonal

- α- sympathetic receptors are more prominent in the epicardial vessels, and the β- sympathetic receptors are more prominent in the intramuscular vessels.
- parasympathetic stimulation has only a slight vasodilatory effect.

PATHOLOGIC ANATOMY

A progressive disease, coronary atherosclerosis may begin early in life and has been seen microscopically in infants.

- The **earliest lesions consist of** rupture, degeneration, and regeneration of the internal elastic membrane, together with deposition of mucopolysaccharide and proliferation of endothelial cells and fibroblasts. At this early stage, such lesions are quite minimal and solely microscopic.
- **Gross lesions subsequently appear within a few years** in the form of small yellow deposits of lipoid material visible beneath the intima..
- **In the final stages of the disease**, rupture of an intimal atherosclerotic plaque appears to be a dominant mechanism of worsening symptoms, with deposition of platelets and thrombus progressing to thrombotic occlusion and acute myocardial infarction. Subtotal occlusions by dynamic thrombotic lesions appear to be of major importance in the pathogenesis of unstable angina.

ISCHEMIC PATHOPHYSIOLOGY

When an atherosclerotic plaque in a proximal coronary artery decreases the cross-sectional area by 75% or more, the resistance to flow caused by the plaque becomes significant. *Whereas flow may be adequate at rest, exercise or other factors that increase myocardial oxygen demand can produce relative ischemia*, a fall in the coronary pressure distal to the stenosis, and redistribution of blood flow away from the subendocardium. This appears to be the mechanism of **exercise-induced angina pectoris** and associated transient myocardial dysfunction. Superimposed on the phenomenon, coronary vasospasm and unstable thrombotic plaques can compound the obstructive physiologic process.

Atherosclerosis is the primary risk factors and other risk factors have been recognized as increasing the frequency of coronary artery disease. The major ones include

- **cigarette smoking**
- **hypertension**
- **Diabetes**
- **severe obesity**
- **family history**
- **lack of physical exercise**
- **aging**

ISCHEMIA AND MYOCARDIAL CELL INJURY

Myocardial ischemia can lead to reversible and/or irreversible injury.

- Ischemia of 15 to 20 minutes duration is associated with postischemic myocardial dysfunction that lasts from hours to days despite the restoration of normal coronary blood flow. This reversible injury is referred to as ***myocardial stunning***.
- Reversible contractile dysfunction that matches a reduction in resting coronary artery blood flow is termed ***hibernating myocardium.*** It is characterized by a balanced reduction in myocardial contractility and oxygen consumption and is typically found in patients with severe CAD who present with stable or unstable angina (UA), myocardial infarction, or congestive heart failure.
- ***Myocardial infarction*** represents cell death and necrosis: It is an irreversible injury that is associated with ischemia lasting more than 20 minutes. In the absence of adequate collateral flow, sustained ischemia often results in a transmural infarction within 6 to 12 hours.

CORONARY COLLATERAL CIRCULATION

The human heart has few natural collateral vessels of sufficient diameter for delivering a significant quantity of blood in the event of a major coronary occlusion. It is for this reason that sudden occlusion of an otherwise normal coronary artery is such a hazardous event.

Stenotic lesions of 90% or greater are required for production of significant collateral vessels in man.

Some patients experience myocardial infarction with minimal or even absent coronary narrowing. Coronary arterial spasm may be responsible for this phenomenon (***Prinzmetal's variant or atypical angina***)..

Myocardial ischemia is most often made manifest by retrosternal chest pain, or angina pectoris. Early in the symptomatic course, a number of factors, such as exercise, cold exposure, eating, and emotional stress, can initiate the symptoms.

The severity of chest pain can be graded by the New York Heart Association (NYHA) classification:

Class I indicating no symptoms;
Class II symptoms with severe exertion;
Class III chest pain with mild exertion; and
Class IV angina occurring at rest.

Significantly, a large proportion of patients do not follow the classic symptomatic progression and present initially with acute myocardial infarction or sudden death. Still others experience no symptoms during ischemia (***silent myocardial ischemia***), and coronary artery disease is discovered only in the late stage of congestive heart failure after severe ventricular damage has occurred.

Diagnostic evaluation

⌘ CXR

The **chest radiograph** is normal in the majority of patients. Left ventricular aneurysms can sometimes be detected radiographically as an isolated prominence of the left border of the heart. With advanced CAD there may be evidence of cardiomegaly, pulmonary edema, or pleural effusion, which are indicative of heart failure.

⌘ ECG

Myocardial ischemia may be manifested by the presence of inverted T waves on the resting electrocardiogram or, alternatively, by *transient ST-segment and T-wave changes* during the course of an anginal episode. ST-segment elevation or depression is an especially reliable sign and, if not present at rest, may be elicited by an exercise stress test.

⌘ Exercise testing

Importantly, exercise testing provides useful information regarding prognosis. Patients with early positive studies are likely to have severe CAD and are more likely to suffer acute myocardial infarction and cardiac death. Particularly, the left ventricular ejection fraction measured during exercise is a powerful predictor of survival.

⌘ Echocardiography

Common indications for a **resting echocar-diogram** include

♦heart murmers and suggested diagnosis such as *aortic stenosis or insufficiency, *hypertrophic cardiomyopathy, *mitral stenosis or regurgitation, and *congestive heart failure.

♦regional wall motion abnormalities

♦ventricular dilatation

♦wall thining,

Patients who are unable to exercise because of physical limitations can be evaluated by **dobutamine stress echocardiography**, which is an important noninvasive study. Dobutamine stress echocardiography assesses wall motion and response to inotropic stimulation wall motion abnormalities, and reduced inotropic response reflects underlying areas of ischemia.

⌘ Single Photon Emission Computed Tomographic Imag-ing (SPECT)

They are particularly useful in in patients with ●left ventricular hypertrophy and/or ●conduction abnormalities and ●for patients unable to achieve 85% of their maximum predicted exercise response.

⌘ Positron Emission Tomogr-aphy (PET)

This test is useful for **assessing myocardial viability and metabolism and evaluating myocardial blood flow.** It depends on the fact that myocardial extraction of glucose is increased in ischemic myocytes.

⌘ MRI-Gadolinium MRI

For evaluation of myocardial viability.

⌘ ECG-Gated Multidetector Spiral Computed Tomography / Electron Beam Computed Tom-ography

ECG-Gated Multidetector Spiral CT can identify regional wall thinning and the presence of mural thrombus.

EBCT quantification of coronary artery calcium may correlate with existing asymptomatic myocardial ischemiain clinically high risk patients. This suggest that EBCT may be a good screening test for CAD.

⌘ Coronary Arteriography

Is essential in defining the presence and extent of coronary atherosclerotic lesions. At angiography, the major anatomic predictors of coronary death, such as

●the number of coronary vessels diseased

●the resting left ventricular ejection fraction, are documented.

MEDICAL MANAGEMENT

- **Hypertension** should be controlled
- **smoking** should be avoided
- **Hyperlipidemias** have a major role in the pathogenesis of coronary atherosclerosis, and clinical efforts should be directed toward aggressive management of lipid abnormalities by use of either dietary or pharmacologic means.
- **Beta-adrenergic blocking agents**, such as propranolol, atenolol, and timolol, are highly effective and safe in the chronic management of angina pectoris. These drugs act by lowering myocardial oxygen requirement through a reduction in heart rate with secondary effects on arterial blood pressure and myocardial contractility.
- **Calcium channel blocking agents** such as nifedipine, diltiazem, and verapamil are well established in clinical practice and are effective in anginal management.
- **Angiotension converting enzyme inhibitors** have been shown to improve survival in patients with impaired ventricular function and those after myocardial infarction.
- **Antiplatelet** agents, such as aspirin, have a definite therapeutic role and have been shown to decrease coronary events significantly.
- **short term heparinization** has been effective in preventing coronary thrombosis and infarction in patients with unstable angina.

In current practice, **adverse prognostic characteristics that might suggest abandoning medical treatment and referral for coronary revascularization** include

★severe or progressive angina on medical therapy(NYHA Class **III-IV**)

★significant left main coronary disease(> 75% stenosis)

★multivessel coronary obstruction (especially with proximal LAD involvement)

★ventricular impairment with a reduced ejection fraction(LVEF **<50%**).

★evidence of exercise induced ischemia.

Complications of Acute MI

1) Rupture of the ventricular free wall causes hemopericardium and resulting tamponade. This accounts for **10%** of hospital deaths due to acute myocardial infarction.Urgent surgical therapy with ventricular reconstruction can lead to survival.

2) Postinfarction VSD new onset of a harsh holosystolic murmur, with a thrill. Hemodynamic compromise with ventricular failure occurs early. Except in moribund patients, correction by patch closure of the VSD and CABG should be undertaken.

3) Rupture of the papillary muscle leads to severe mitral regurgitation and rapid development of pulmonary edema and hypotension. A holosystolic murmur is usually present. Echo is diagnostic. Early operative therapy with mitral valve repair when possible or mitral valve replacement in conjunction with bypass grafting is indicated.

SURGICAL MANAGEMENT

The natural history of patients with CAD indicates that ***survival is decreased with the***
✠*increasing amount of myocardium at risk for ischemia*. Arterial obstruction occurring
★proximally, as compared with distally
★involving the LAD as compared with the right coronary artery
★involving the left main artery or three-vessel disease as compared with two-vessel or single-vessel disease is associated with impaired survival.
✠*increase in the severity of symptoms*
✠*impaired left ventricular function*
✠*ischemia inducible by low levels of exercise*.

An ***advantage in survival*** in patients treated with ***coronary artery bypass grafting (CABG)*** has been demonstrated in a number of randomized clinical trials ***in the following patient groups***:
①patients with more than 75% stenosis of the left main artery
②patients with three-vessel or two-vessel disease with proximal LAD involvement
③in association with one of the following:
★moderate to severe anginal symptoms (NYHA Class III-IV)
★impaired left ventricular ejection fraction (LVEF <50%)
★easily inducible ischemia with exercise.

Therefore, CABG is recommended as therapy in these patients, although some may be amenable to treatment with percutaneous transluminal coronary angioplasty (PTCA).