CORONARY ARTERY DISEASE

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Ischemic Heart Disease

- Ischaemic (or ischemic) heart disease is a disease characterized by reduced blood supply to the heart.
- It is the most common cause of death in most western countries.
- Ischaemia means a "reduced blood supply".
- The coronary arteries supply blood to the heart muscle and no alternative blood supply exists, so a blockage in the coronary arteries reduces the supply of blood to heart muscle.

Most ischemic heart disease is caused by atherosclerosis, usually present even when the artery lumens appear normal by angiography.
Initially there is sudden severe narrowing or closure of either the large coronary arteries and/or of coronary artery end branches by debris showering downstream in the flowing blood.

It is usually felt as angina, especially if a large area is affected.
The narrowing or closure is predominantly caused by the covering of atheromatous plaques within the wall of the artery rupturing, in turn leading to a heart attack (Heart attacks caused by just artery narrowing are rare).
A heart attack causes damage to heart muscle by cutting off its blood supply.

Coronary artery disease (CAD), also known as
ischemic heart disease (**IHD**)
atherosclerotic heart disease,[1]
atherosclerotic cardiovascular disease,
coronary heart disease,
is a group of diseases that includes:
Stable angina,
Unstable angina,
Myocardial infarction,
sudden coronary death.
It is within the group of cardiovascular diseases of which it is the most common type. A common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw.

Occasionally it may feel like heartburn.
Usually symptoms occur with exercise or emotional stress, last less than a few minutes, and gets better with rest. Shortness of breath may also occur and sometimes no symptoms are present. The first sign is occasionally a heart attack. Other complications include heart failure or an irregular heartbeat.

Risk factors

- high blood pressure,
- smoking, diabetes,
- lack of exercise,
- obesity,
- high blood cholesterol,
- poor diet, and excessive alcohol,
- depression.

CLINICAL SYNDROMES ASSOCIATED WITH CAD

Angina Pectoris

Angina pectoris is defined as chest pain that is related to ischemia of the myocardium. However, the pain referred from ischemia may be in the left shoulder, jaw, or between the shoulder blades. Angina can be classified as stable, unstable, or variant.

Stable angina

Stable angina generally occurs during physical effort but may be related to stress. The individual is able to describe what type and intensity of activity causes the angina. Stable angina is characterized by sub sternal, usually non radiating pain that lasts between 5 to 15 minutes after the offending incident. Care would involve sublingual nitrates and cessation of the activity causing the angina. Usually the angina subsides completely with treatment. However, angina brought about by emotional stress is more difficult to treat, since the stress cannot be stopped as easily as cessation of exercise.

Unstable angina

- Unstable angina also occurs during physical exertion or psychological stress. The major difference between stable and unstable angina is the frequency, duration and intensity of the pain. In unstable angina, the episodes are more frequent and the duration of each event is usually greater than 15 minutes.
- In addition the intensity or the pain may be more severe.

Unstable angina

- Unstable angina is usually an indicator of CAD progression. Individuals with unstable angina are at greater risk to have a myocardial infarction (MI). Unstable angina is less responsive to treatment using rest and sublingual nitrates. Often times, the individual must be hospitalized and treated with IV nitrates.

Variant angina

- Variant angina occurs while the individual is at rest, usually during waking and often at the same time period. Exertion does not influence variant angina. However, the angina may benefit from rest and sublingual nitrates. Like unstable angina the pain is intense and of longer duration and likely to lead to an MI. In addition, arrhythmias are more likely to occur with an individual who has variant angina, as compared with exertion related angina (i.e., stable and unstable).

Variant angina

- Stable and unstable angina are believed to be caused primarily by progressive arterial occlusion and ischemia. It is believed that variant angina is caused by a combination of occlusion and coronary artery spasm. Therefore variant angina has been successfully treated with calcium channel blockers.

Prognosis of angina

- Individuals do not die from angina. However, even though there is no risk for mortality from angina, an individual's lifestyle can change drastically. People with angina may be fearful of being active and may deny that they are having exertion-related chest pain. Denial of CAD, depression and anger are common manifestation for individuals with angina,

Pathophysiology

Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the myocardial cells. Myocardial cells may die from lack of oxygen and this is called a myocardial infarction (commonly called a heart attack). It leads to heart muscle damage, heart muscle death and later myocardial scarring without heart muscle re growth. Chronic high-grade stenosis of the coronary arteries can induce transient ischemia which leads to the induction of a ventricular arrhythmia, which may terminate into ventricular fibrillation leading to death.

Pathophysiology

- Typically, coronary artery disease occurs when part of the smooth, elastic lining inside a coronary artery (the arteries that supply blood to the heart muscle) develops atherosclerosis.
- With atherosclerosis, the artery's lining becomes hardened, stiffened, and swollen with all sorts of "gunge" - including calcium deposits, fatty deposits, and abnormal inflammatory cells - to form a plaque.

Pathophysiology

Although the specific pathogenesis of atherosclerosis is not known it is hypothesized that the process is initiated by trauma to the intima of the arterial wall. The trauma may be related to various primary risk factors such as: high blood pressure and cigarette smoking.

High blood pressure has been indicated as a potential trauma inducer, since increased pressure and turbulence may damage the endothelial cells of the intima, thus exposing the media to the circulation. The media, which is composed primarily of smooth muscle is thought to be the origin of the atherosclerotic lesion.

Cigarette smoking has also been indicated as a potential trauma inducer. However, the hypothesized mode of injury is different than that observed with increased blood pressure. Cigarette smoke is high in carbon monoxide and hydrocarbons that are carried by the red blood cells and the plasma. It is thought that the hydrocarbons or carbon monoxide bind to the endothelial cells, causing damage and possibly death to these cells.

Once the media is sufficiently exposed to the circulation the process of atherosclerosis is initiated. Platelets aggregate at the injury site and release substances that induce endothelial and smooth muscle cell replication. It is at this site that fatty streaks and fibrous plaques are developed.

Myocardial Infarction

MI is defined as necrosis of a portion of the myocardium.

The death of the myocardium occurs as a result of ischemia and anoxia. The vessels affected are the right and left coronary arteries. The clinical symptoms are similar to that of angina, with emphasis on extreme pressure as well as tightness over the sternum region. In addition, pain can radiate to the jaw, upper back, and shoulders (with left more frequent than right).

MIs can be classified into categories by size, location, and degree of myocardial wall involvement. The terms small and large are often used to describe MIs.
However, degrees of complication are also used in conjunction with size. MIs can be described as uncomplicated and complicated based on size of the MI and recovery of the patient.

Uncomplicated myocardial infarction

An uncomplicated MI is described as a small infarction with no complications during recovery. Usually the result is full recovery without a significant decrease in cardiac performance at rest and during minimal to moderate activity. However the location and the extent of the MI is also critical. MIs located in the inferior portion of the heart are the least significant, and partial wall thickness is less significant than a trans mural MI.

Treatment

The treatment for an uncomplicated MI is initially like the complicated MI, where the patient is cared for in the coronary care unit. The medical treatment is designed to decrease myocardial work and oxygen demand. Therefore patients are on oxygen and administered vasodilators (nitroglycerin) to increase myocardial blood flow and analgesics to help further reduce ischemic pain. In addition, to reduce contractility of the myocardium, calcium channel blockers or beta-blockers are administered.

Anti arrhythmia medication may be prescribed if an aberrant cardiac rhythm is present or is highly probable to occur.

Since the course is uncomplicated, this means that a patient's stay in the coronary care unit may be only 2 to 3 days, with a total hospital stay of 7 to 10 days. Treatment following lCU discharge is oriented toward increasing physical activity and in educating the patient and family in risk factor reduction.

This process is described as cardiac rehabilitation Phase 1.

Complicated myocardial infarction

A complicated MI is different from an uncomplicated case since the patient may have one, a combination, or all four of the following conditions/complications: (I) arrhythmia, (2) heart failure, (3) thrombosis, and (4) damage to heart structures.

Arrhythmias

Arrhythmias occur in 95% of all patients with MIs. The type and severity of the arrhythmia is dependent on the extent of myocardial damage and the location of the damage. Arrhythmias that are life-threatening include (I) complete A V heart block, (2) ventricular paced arrhythmia, and (3) ventricular tachycardia, including ventricular flutter and fibrillation. In these conditions, either the heart rate is too slow and thus cardiac output is impaired,

Arrhythmias

Or the heart rate is too rapid with poor stroke volume and ejection fraction, and again impaired cardiac output. Treatment of the above conditions is immediate and requires drugs and potentiallyand electrical shock (for flutter/fibrillation). Usually,an artificial pacemaker is implanted once the patient is stabilized.

Heart failure

Heart failure is a condition where the heart is weakened and is unable to produce a significant

cardiac output to meet the body's need for oxygen,

nutrition, and removal of waste products. the heart experiences ischemia the myocardium contracts

with less force, and conduction abnormalities may alter the mechanics of the contraction. the affected myocardium does not contract, thus affecting overall cardiac output.

Heart failure

Post-MI, cardiac output is reduced significantly. However, the compensatory response is to increase sympathetic innervations, resulting in increased heart rate and myocardial contractility. result of this compensation is a cardiac output that may approach normal resting values. However, if the damage has been great, the kidneys compensate by retaining sodium and water in an attempt to improve circulatory volume and venous return.

Heart failure

Depending on the amount of myocardial tissue death, the individual may survive with resulting chronic congestive heart failure through persistent fluid retention and hypotension. If greater than 40% of the left ventricle is infracted, the result is usually cardinogenic shock followed by death of the individual.

Thrombosis

Another complication is increased incidence of thrombosis from deep leg veins and from the damaged heart itself. Thrombosis from deep leg veins occurs from lower limb inactivity and circulatory stasis. This is a complication that can be observed for all surgical patients. Emboli from deep leg vein thrombus usually result in pulmonary complications. If the emboli are large or numerous the result can be pulmonary tissue infarction and potentially death.

Thrombosis

The incidence of pulmonary emboli has grown less since early ambulation is now the rule rather than the exception. However, a pulmonary emboli must be considered a distinct possibility in all surgical, MI, and gunshot patients. Heart wall or mural wall thrombosis can lead to an emboli lodging in the brain, intestine, kidney, artery to the extremities, or any location in the systemic arterial circulation. Usually mural thrombosis do not affect the pulmonary system, since even the smallest fragments are caught in capillary beds and do not enter into the venous system.

Structural damage

The last complication is structural damage to critical myocardial tissue that affects heart function. If conductant pathway (bundle branch) tissue located primarily at the septum is damaged, arrhythmias result. In addition, papillary muscles that assist in closing valves can be infracted. The result of improper valve function is decreased cardiac out put. Besides these two critical tissues, if significant full thickness damage occurs to the myocardial wall cardiac function is compromised.

Structural damage

- Heart wall damage can result in ventricular aneurysms or ventricular wall rupture. Ventricular aneurysm or bulging of the weakened ventricular wall occurs in trans mural (full wall thickness) infarcts. Ventricular wall I rupture, which can occur acutely following trans mural infarction, but more often occurs in the first to second week post-MI following an aneurysm is usually fatal. Therefore following a n MI, it is critical to determine if an aneurysm has occurred in the myocardium so that appropriate surgical intervention can be performed.

Treatment

The medical treatment is designed to decrease myocardial work and oxygen demand. Patients are on oxygen and administered vasodilators (nitroglycerin) to increase myocardial blood flow and analgesics to help further reduce ischemic pain. Myocardium calcium channel blockers or beta-blockers are administered to reduce contractility. Finally, anti arrhythmia medication are prescribed if an aberrant cardiac rhythm is present.

Treatment

Individuals with a complicated MI have a much longer stay in the coronary care unit, and their total hospital stay time is greatly increased when compared with the un complicated MI patient. The time in coronary care and total hospital stay are dependent on the complications that occur following the MI. Individuals with heart failure, thrombolytic events, or structural damage requiring surgery may be in the coronary care unit for more than 2 weeks. Total hospital stay may exceed 3 weeks

Treatment

Treatment following ICU discharge is similar to that of the uncomplicated MI patient with the goal of increasing physical activity and in educating the patient and family in risk factor reduction. The major difference in Phase I cardiac rehabilitation for the complicated versus the uncomplicated MI patient is the initial work load intensity, duration, and frequency. recurrent MI event is much greater in the complicated MI patient

Prognosis post-myocardial infarction

Prognosis following MI is dependent on many factors.

Usually cardiovascular performance is reduced, unless the structural damage to the ventricle is minor. The most important factor is extent of ventricular damage. However, with early detection of trans mural infarction and improvement in surgical intervention and coronary care, acute post-MI deaths have been reduced Other critical factors include remaining cardiac capacity and status of CAD. Even though CAD mortality has declined ,still the disease remains the top cause of sudden death in adults.

Congestive Heart Failure

CHF is characterized by the inability of the heart to maintain adequate cardiac output. The etiologic factors of heart failure is usually from ischemia and MI secondary to CAD. To maintain an appropriate amount of blood flow to the pulmonary and systemic circulation, heart rate and stroke volume must be adequate. Usually, stroke volume is the critical factor to maintain adequate cardiac output. Stroke volume is a function of the amount of blood in the left ventricle at the end of diastole (preload);

Congestive Heart Failure

The amount of pressure and resistance the heart must overcome to eject blood into the systemic circulation (after load); and myocardial contractility, which is the amount of force the left ventricle can apply to the blood within the chamber. If any of these three variables are negatively affected then cardiac output is reduced. The cause of heart failure is often decreased contractility.

Acute heart failure

If an individual has a significant myocardial infarction, the contractility and pumping ability of the heart is immediately reduced. The initial result is decreased cardiac output and damming of blood in the veins. The result is increased systemic venous pressure. This acute phase, which may reduce cardiac output to 40% of normal resting values is short-lived lasting only a few seconds before the sympathetic nervous system is stimulated,

Acute heart failure

Sympathetic innervation causes an increase in contractility of viable myocardial tissue, and the increase in cardiac output may be 100%. In addition, sympathetic innervation also increases venous return, since the tone of blood vessels is increased. The result is increased systemic filling pressure, and thus increased preload. The sympathetic reflex following MI becomes maximally operational within 30 seconds;

Chronic heart failure

Following a MI→ The kidney begins to retain fluid almost immediately→ there is an increase in renin output and therefore an increase in angiotensin production.→ Angiotensin promotes re absorption of water and salt from the renal tubules. → increase in blood volume and an increase in venous return. → increases preload and thus cardiac output.

However, if the MI was severe, the result can be excess fluid retention.

Second process that is activated immediately following an MI is recovery of the myocardium. New collateral arteries are formed to supply the peripheral portions of the infracted region. This revascularization can assist cells that were marginally active to become fully functional again. In addition, the unaffected myocardial cells hypertrophy. The result in a mild to moderate MI is a great improvement in cardiac function that takes 6 weeks to several months, depending on extent of injury.

Compensated heart failure

- The final state following acute and chronic physiological changes is called compensated heart failure. In this state, the heart is able to pump blood effectively, but at a reduced cardiac output compared with the pre-MI condition. The individuals cardiac reserve has been greatly reduced. When an individual exercises or is active at a heavy load, they experience the same symptoms of acute heart failure, because the heart is unable to supply the cardiac output required of the activity. The symptoms include rapid heart rate, pallor, and diaphoresis.

Decompensated heart failure

Decompensated failure occurs when the heart is so severely damaged or weakened that normal cardiac output can not be attained. The result is that cardiac output is not high enough to allow for normal renal function.


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