

MODULE 16

MYOCARDIAL INFARCTION

OBJECTIVES

Upon completion of this module, you should be able to:

- Define Myocardial Infarction (MI).
- Describe predisposing factors of CAD (Coronary Artery Disease).
- Describe the pathophysiology and clinical manifestations of myocardial infarction.
- Identify diagnostic procedures to determine the presence of an MI.

COMMENTS

Myocardial Infarction (MI) is the leading cause of death among Americans. According to the American Heart Association, nearly one million Americans have acute MI's; one fourth of these people die of MI. One half of those that die never reach a hospital. The average person suffering a myocardial infarction waits three hours before seeking assistance. Many deaths occur because of the dysrhythmias that often accompany the MI within the first few hours. However, the advent of emergency medical systems (EMS) and hospital-based coronary care units providing cardiac monitoring have helped dramatically reduce the death rates of victims seeking medical attention.

DEFINITION AND ETIOLOGY

A myocardial infarction is the death (necrosis) of myocardial tissue. It is caused by an interruption of blood flow through the coronary arteries that oxygenate and nourish cardiac tissue. The most frequent cause of the interruption of blood flow is complete or nearly complete obstruction of a coronary vessel caused by atherosclerotic lesions (coronary artery disease). The formation of a thrombus or blood clot at the site of the lesion may serve to occlude an already narrow artery. Arterial spasm of vessel walls may also occur, further reducing blood supply to the affected tissue.

The size of the infarction and where it is located depends upon the portion of the coronary vessel that is occluded. The most common site is the anterior wall of the left ventricle near the apex. The second most common site is the posterior wall of the left ventricle. Ninety five percent of the time, the infarction occurs somewhere in the left ventricle. A transmural MI involves full thickness of myocardial tissue while a subendocardial MI involves only partial thickness destruction of cardiac tissue (see Figure 1).

Figure 1

When an occlusion of a coronary vessel occurs, the tissue distal to the occlusion is deprived of oxygen and nutrients. Ischemia begins subendocardially and gradually extends through to the epicardium. Necrosis occurs within six hours. The tissues involved appear grayish instead of the normal red/brown color. Around the necrotic tissue is an area of reversible ischemia. The final size of the evolving MI depends upon the extent and length of deprivation of the blood supply to the cardiac tissue. The development of collateral circulation will also play a role since this blood supply may be able to provide some oxygen and nutrients to tissues in the affected area, thereby limiting ischemia.

Functional contractility is lost to the necrotic areas leaving a heart muscle that may not pump effectively. Between the fourth and tenth day following the MI, phagocytes begin clearing away necrotic tissue and a collagen matrix begins to fill in the affected area. After the tenth day, beginning scar tissue is present but is very mushy and weak and is, therefore, especially vulnerable to stress.

PREDISPOSING FACTORS

Coronary artery disease (CAD) which may lead to myocardial infarction has many predisposing risk factors. Some of these are controllable and some are not.

Uncontrollable

age (> 45 years for men;
> 55 years for women)
genetics
sex (heart disease occurs
three times more often
in men than premenopausal
women)
race

Controllable

cigarette smoking
diabetes
hypertension
hyperlipidemia
stress
obesity
physical activity

DIAGNOSTIC CRITERIA

Clinically the individual suffering from an MI may display a variety of symptoms. Classic symptoms include:

- crushing chest pain
- shortness of breath
- nausea/vomiting
- diaphoresis
- weakness
- hypotension

The pain generally lasts longer than anginal pain and is not relieved by nitroglycerine or rest. At the opposite end of the spectrum some individuals suffer “silent MI’s” that appear to be without any noticeable symptoms. And in between these two extremes may

be persons suffering from vague complaints, such as indigestion, through the entire range of symptoms described above.

A series of diagnostic EKG's may trace the evolving MI. Changes on the EKG are usually seen in the leads facing the area of infarction. The presence of pathologic U wave and ST and T wave changes are diagnostic of an MI.

Laboratory studies are also significant, especially the levels of iso-enzymes CK-MB and LDH muscle cells. With an MI the damaged cells rupture, spilling their enzymes into the circulation causing abnormal increases. Blood samples taken serially will demonstrate an increase in these enzymes. Elevations in CK-MB, which is an iso-enzyme specific to cardiac tissue, can be detected in blood sampling as early as four hours following an MI. The presence of leukocytosis and an elevated sedimentation rate are also diagnostic indicators.

After the acute episode, radionuclide imaging may be used to identify the extent of cardiac damage suffered. The MUGA scan, for example, allows radionuclide tracers attached to the blood to pass through the cardiac circulation providing visualization on a screen of actual ventricular wall movement. The technesium scan outlines "hot spots" where radionuclides are taken up in concentration by damaged myocardial tissue. The Thallium scan, on the other hand, outlines "cold spots". Areas of decreased uptake are identified as damaged.

MEDICAL MANAGEMENT

Medical treatment centers on efforts to increase the blood flow to the affected area of the heart quickly before extensive damage occurs. It can therefore be anticipated that the patient will receive oxygen, usually by nasal cannula, at a rate of 2-4 L/min. Achieving patient comfort is extremely important since the stress of pain increases cardiac workload and therefore the myocardial need for increased blood supply.

Because of this, patients are often given analgesia such as IV morphine. Intravenous access is essential to ensure a route of administration for emergency medications. An IV solution of 5% D/W at a "keep vein open" (KVO) rate is commonly ordered. Continuous cardiac monitoring is also essential to ensure the prompt detection and treatment of dysrhythmias. An electrocardiogram is done, and if a baseline cardiogram is available, a comparison is made to determine the presence of any changes. Often serial cardiograms are performed for this purpose.

Some persons may receive Streptokinase in an effort to dissolve the clots in the blocked artery. To be a candidate for this therapy, the onset of symptoms is less than six hours. Among other criteria there must be no previous history of any bleeding disorders. Sometimes Streptokinase is given intravenously but other times it is given in conjunction with cardiac catheterization where it may be bloused into the involved coronary artery. A continuous infusion of Streptokinase usually follows. The nurse should assess the patient carefully for any signs that may indicated bleeding. A percutaneous transluminal

angioplasty may also be performed. This involves insertion of a balloon tipped catheter into the coronary vessel where inflation of the balloon crushes plaque against the vessel wall enlarging the lumen and increasing blood flow.

Upon transfer to the cardiac care unit, the person is usually placed on bed rest, initially given a liquid diet, and continuous cardiac monitoring. The nurse assists the client with hygiene, toileting, and all activities. Nitroglycerine may be given sublingual (SL) or via IV drip. Beta blockers such as Inderal or Corgard, and calcium channel blockers such as Nifedipine may be given. A stool softener, such as Colace, and a pain-controlling medication may be ordered. Monitoring of vital signs, especially blood pressure, as often as every hour with continuous observation and assessment for complications such as CHF, dysrhythmias, and signs of cardiogenic shock are routine procedures. Within 24-48 hours the patient, if stable, is usually allowed to sit in a chair and is placed on a soft diet. The patient's level of fatigue and changes in vital signs are carefully monitored with each increase in activity.

Some suggested nursing diagnosis for MI patients are:

- Activity intolerance related to cardiac O₂ supply vs. demand.
- Anxiety related to fear of death and change in health.
- Decreased cardiac output related to cardiac malfunction.
- Altered tissue perfusion (cardiopulmonary) related to cardiac O₂ supply vs. demand.

Depending on the individual patient and family responses, there may be many other appropriate diagnostic categories, such as:

- Body image disturbance
- Ineffective breathing pattern
- Ineffective denial
- Altered family process
- Fatigue
- Fear
- Excess fluid volume
- Impaired gas exchange
- Altered health maintenance
- Knowledge deficit related to all aspects of health care
- Pain
- Powerlessness
- Self-care deficits
- Self-esteem disturbance

As the patient progresses beyond the acute stage, the nurse may identify other concerns such as those related to role performance, health seeking behaviors, sexuality, continued low self-esteem, denial, and non-compliance.

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