QUICK LESSON

Acute Myocardial Infarction

Description/Etiology

Acute myocardial infarction (MI; also called heart attack) is the rapid development of myocardial necrosis (i.e., the death of an area of heart muscle) due to prolonged myocardial ischemia, which results from a disruption in the balance between oxygen supply and demand. (The term "acute" refers to infarction that is less than 3–5 days old.)MI is most commonly caused by a thrombus that blocks a coronary artery previously narrowed by the buildup of fatty plaque. Additional causes include coronary artery spasm, embolic infarction, mechanical obstruction (e.g., chest trauma, aortic dissection), hematologic disorders (e.g., disseminated intravascular coagulation [DIC],), aortic stenosis, drug abuse (e.g., cocaine), severe burns, prolonged hypotension, and arteritis.

Current classification of MI recognizes five categories: spontaneous MI (MI type 1), MI secondary to ischemic imbalance (MI type 2), cardiac death secondary to MI (MI type 3), MI secondary to percutaneous coronary intervention (PCI) (MI type 4), and MI secondary to coronary artery bypass graft (CABG) surgery. Based on electrocardiographic findings, MI can be further classified as ST elevation MI (STEMI), in which there is ST elevation in two contiguous leads, and non-STelevation MI (NSTEMI). It is important that healthcare professionals differentiate whether the patient is experiencing a STEMI or NSTEMI, as treatment differs between these two conditions. Diagnosis of MI is based on patient history, clinical presentation, and indirect evidence of myocardial necrosis using blood sampling for serum biomarkers, electrocardiographic findings, and diagnostic imaging. MI should be differentiated from acute coronary syndrome with or without stable or unstable angina pectoris; anxiety; aortic stenosis or dissection; gastroesophageal reflux disease (GERD); esophageal spasm or biliary colic; musculoskeletal or neurologic chest wall pain; chronic obstructive pulmonary disease (COPD); pulmonary embolism; spontaneous pneumothorax; and pericarditis.

Treatment involves emergency department evaluation, admission to and stabilization in the coronary care unit (CCU) or intensive care unit (ICU), oxygen supplementation, medications (e.g., intravenous morphine, beta blockers), restriction of physical activities, and prompt coronary reperfusion with fibrinolytic therapy or primary PCI. Coronary angiography is usually performed before reperfusion procedures. PCI involves advancing a catheter usually through the femoral artery into the coronary arteries, balloon inflation at the occlusion site, and stent placement. PCI is an effective procedure in the treatment of patients with confirmed STEMI if performed within 12 hours of symptoms onset and less than 90 minutes of door to balloon time. CABG surgery, which is indicated when PCI fails, can be performed via conventional, off-pump, or minimally invasive direct, robotic, or endoscopic techniques. Constant electrocardiographic and hemodynamic monitoring is essential. Prognosis varies and depends largely on the size, type, severity, and location of infarct and the amount of remaining functional cardiac muscle. In general, prognosis worsens with advanced age or the presence of arrhythmias, post-MI angina, pericarditis, or concomitant illnesses (e.g., diabetes mellitus), and improves with appropriate use of betablockers or lipid-lowering medications. Potential complications include recurrent or persistent chest pain, heart failure, stroke, deep vein thrombosis, pulmonary embolism, pulmonary edema, ventricular and/or papillary muscle rupture, mitral insufficiency, cardiogenic shock, cardiac arrest, and death.

ICD-9
410.9

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December 18, 2020

Facts and Figures

MI is a leading cause of morbidity and mortality in the United States and other developed nations. Worldwide,31% of all deaths are associated with cardiovascular disease. An estimated 500,000–700,000 deaths associated with coronary artery disease (CAD) occur each year, with 600,000–1.5 million new cases of MI in addition to 320,000 of recurrent attacks. MI predominantly occurs in people > 45 years of age, yet more common in men than in women between the ages of 40–70 years, and occurs more frequently in the black and Hispanic populations in comparison to whites.Approximately26% of women and 19% of men will die within a year of the first AMI, and 47% of women and 36% of men will die within 5 years of the first AMI.

Risk Factors

Risk factors for MI include tobacco smoking, obesity, age > 40 years, sedentary lifestyle, being male although women are at a 1.43-fold higher risk for bleeding complications (e.g., intracranial bleeding secondary to thrombolytic therapy), being a postmenopausal female, high-fat diet, family history of premature onset (age < 55 for women or age < 45 for men) of MI, genetic predisposition, diabetes mellitus, dyslipidemia, and hypertension. Other risk factors include HIV infection; elevated levels of homocysteine, C-reactive protein (CRP), low-density lipoprotein (LDL) cholesterol, or fibrinogen; use of birth control pills, cocaine, or amphetamines; and psychological factors (e.g., depression, anger, hostility, chronic stress); metabolic acidosis, when PH < 7.3 is associated with 100% mortality; and widened pulse pressure (i.e., the difference between the systolic and diastolic pressure readings) of > 60mmHg.

Signs and Symptoms/Clinical Presentation

Signs and symptoms of MI can vary but most often include crushing substernal chest pressure/pain that lasts > 30 minutes and is unrelieved by rest or sublingual nitroglycerin or is rapidly recurring. The pain can radiate to the arms, jaw, neck, shoulders, back, or abdomen and can be associated with shortness of breath or hyperventilation, diaphoresis, cough, syncope, anxiety, a feeling of impending death, and epigastric discomfort with or without nausea and vomiting. Nonetheless, approximately 20% of AMI cases are silent, where the patients are either asymptomatic or associating their discomfort to other medical issues (e.g., indigestion, heartburn).

Assessment

› **Patient History**
- Assessment for personal or family history of angina or other heart problems including cardiac interventions (e.g., PCI) is critical because physical examination can be unremarkable in MI
- Obtain information about the onset and the duration of symptoms

› **Physical Findings of Particular Interest**
- Physical findings can include tachycardia or bradycardia, diaphoresis and skin pallor, blood pressure changes, widened pulse pressure, jugular venous distension, and abnormal breath or heart sounds (e.g., crackles, S3 and S4), diaphoresis, pale and cool skin
- Elderly patients may present silent/unrecognized MI or atypical symptoms including syncope, unexplained nausea, weakness, shortness of breath, and altered mental status

› **Laboratory Tests That May Be Ordered**
- Levels of serum cardiac enzymes and specific cardiac biomarkers—including cardiac specific troponin (cTn) T and I, creatinine kinase (CK) and CK-MB isoenzymes, myoglobin, lactate dehydrogenase (LDH) and LDH1 isoenzyme, homocysteine, and CRP—can be elevated, indicating cardiac muscle necrosis (see *Food for Thought*, below)
- CBC might reveal anemia, elevated WBCs, or elevated erythrocyte sedimentation rate (ESR), indicating inflammatory processes. Triglycerides and LDL and/or very low-density lipoprotein (VLDL) cholesterol might be elevated; levels of high-density lipoprotein (HDL) cholesterol might be low
- Complete metabolic panel might reveal abnormalities (e.g., BUN, creatinine, glucose) consistent with the patient's medical history, and to be ruled out as a possible ethology (e.g., hyperkalemia or hypokalemia, hypocalcemia)
- Brain natriuretic peptide (BNP) is elevated in AMI, and might also indicate heart failure
- Prothrombin time (PT) and partial thromboplastin time (PTT) may be ordered to establish coagulation baseline levels, for possible anticoagulation therapy

› **Other Diagnostic Tests/Studies That May Be Ordered**
- Repeat or serial 12-lead EKG might show ST-segment elevation (≥1mm)or depression in two contiguous leads and the presence of new Q waves or peaked T waves

- Echocardiography will evaluate for wall motion abnormalities and provide information on ventricular function; chest X-ray will evaluate for the presence of aortic dissection or heart failure
- Myocardial perfusion scanning, the most commonly used cardiac nuclear medicine procedure in patients with MI, visualizes blood-flowpatterns to the heart walls. Nuclear ventriculography studies (e.g., radionucleotideventriculography [RNV] or multiple gate acquisition [MUGA] scan) using the radioactive isotopes*technetium-99m* or *thallium-201* and 64-slice CT scans can be ordered to evaluate for damage to cardiac muscle
- MRI can assist in making a diagnosis of MI in patients experiencing chest pain and provide information on the degree of myocardial injury
- Arterial blood gases (ABG's) may be ordered if metabolic acidosis is suspected

Treatment Goals

› **Resuscitate as Appropriate and Reduce Risk of MI-Related Complications**
- Assist with resuscitation as appropriate, including providing supplemental oxygen via nasal cannula at moderate flow rates, as ordered. Place the patient on telemetry and obtain serial EKGs to monitor for arrhythmias. Closely monitor vital signs and fluid, nutritional, respiratory, and hemodynamic status for developing complications
  – Pulmonary artery (Swan-Ganz) hemodynamic monitoring is critical for detecting post-MI complications
- Administer prescribed medications for MI, including alteplase, reteplase, or tenecteplase, as fibrinolytic therapy, followed by an infusion of heparin or enoxaparin, as antithrombotic therapy to prevent rethrombosis; clopidogrel to inhibit platelet aggregation; beta-blocker to prevent ventricular arrhythmias and ischemia; nitroglycerin for angina; atorvastatin to reduce cholesterol levels; ALPRAZolam for anxiety; and morphine sulfate for pain
- Encourage bed rest with the use of a bedside commode for at least the first 24 hours after MI. Assist with range of motion exercises, as ordered
- Follow facility pre- and posttreatment protocols if patient becomes candidate for a procedure or surgery (e.g., CABG or other coronary reperfusion procedure)
  – Reinforce pre- and posttreatment education and verify completion of facility informed consent documents
  – Intensively monitor post-treatment for complications

› **Promote Emotional Support and Educate to Relieve MI-RelatedAnxiety**
- Assess patient's anxiety level and coping ability; provide emotional support and educate about MI etiology, potential complications, treatment risks and benefits, and individualized prognosis; request referral to a mental health clinician, as appropriate, for counseling on strategies for coping with having a life-threateningcondition

Food for Thought

› The chromosome 9 variant 9p21 has been identified in 2007 as an independent risk factor for CAD, particularly to heart failure, sudden death, ischemic stroke, and intracranial aneurysm. To date, there are a total of 50 genetic markers predisposing to CAD (Skhohlar et al, 2017)

› Women generally experience more lethal and severe first MIs than men, regardless of age, history of angina, or existing comorbidities; in addition, women and older adults often experience atypical symptoms (Mehta et al, 2016)

› Severe depression is common in patients following MI. Researchers in a recent empirical study of 310 patients hospitalized for MI, PCI or CABG surgery reported that 44%of the patients presented persistent and/or worsening depressive symptoms in the first year after the cardiac event. The researchers indicated that the depression symptoms were linked to individual factors such preexistent baseline depression, low or lack of social support, and loss of trust in the physicians. The investigators recommend vigilant follow-up and screening for depression immediately following cardiac events (Mittag et al., 2016)

› Cardiac markers (e.g., troponin, CK-MB, myoglobin) are used for the diagnosis of patients suspected of experiencing a cardiac event. Troponin as the preferred method for the diagnosis of AMI is more specific, as it is released into the bloodstream as a response to heart muscle injury. CK-MBand myoglobin levels can be elevated with either heart damage or skeletal muscle injury (Aydin et al, 2019)

Red Flags

› Absence of Q waves on 12-lead EKG in a symptomatic patient indicates partial occlusion or early infraction, and new ST segment elevation or T wave changes may indicate myocardial ischemia or injury

› Clopidogrel therapy should be discontinued for at least 5–7 days before elective CABG

› Nitrates should not be administered in patients who have recently used phosphodiesterase (PDE-5) inhibitors (e.g., sildenafil [Viagra]), and who have hypersensitivity to the drug (e.g., extreme drop in blood pressure)

› Closely monitor high-risk patients (e.g., those with pre-existing kidney disease, heart failure, volume depletion) for contrast-induced acute kidney injury by measuring serum creatinine levels once daily for 5 days after coronary angiography

What Do I Need to Tell the Patient/Patient's Family?

› Advise the patient to seek immediate medical attention or call emergency medical services (e.g., 9-1-1) for new or worsening signs and symptoms suggestive of MI, including crushing chest pain
› Encourage family members to attend CPR classes
› Suggest to consult with the cardiologist regarding purchasing a home defibrillator
› Emphasize the importance of attending post-hospitalization programs for cardiac rehabilitation, weight reduction, and/or smoking cessation, as appropriate
› Educate about the need to adhere to the prescribed treatment regimen of antihypertensive drugs and statins to reduce the risk for recurrent MI
› Encourage making lifestyle changes including smoking cessation, healthy diet, stress reduction strategies (e.g., meditation), and regular physical activity, as appropriate

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