Chapter 6
Acute Coronary Syndromes

Learning Objectives

- Define:
  - Acute coronary syndrome (ACS)
  - Angina
  - Unstable angina pectoris (UA)
  - Non-ST segment elevation myocardial infarction (NSTEMI)
  - ST segment elevation myocardial infarction (STEMI)

Learning Objectives

- Discuss out-of-hospital morbidity and mortality issues

- List signs/symptoms of chest pain that indicate ischemia

- List risk factors for acute myocardial ischemia
Learning Objectives

- Discuss medications used to treat stable angina pectoris
- Discuss medications used to treat unstable angina (UA) and NSTEMI

Learning Objectives

- List benefits of beta blockers on ischemic heart and discuss beta-blocking medications:
  - Metoprolol (Lopressor)
  - Atenolol (Tenormin)
  - Esmolol (Brevibloc)
  - Propranolol (Inderal)
  - Labetalol (Trandate)

Learning Objectives

- List benefits of antiplatelet agents on ischemic heart and discuss antiplatelet medication clopidogrel (Plavix)
- Discuss anticoagulant medications, including unfractionated heparin (heparin) and low-molecular-weight heparin (LMWH)
Learning Objectives

- Discuss role of angiotensin-converting enzyme (ACE) inhibitors and 3-hydroxy-3-methylglutaryl (HMG) coenzyme A reductase inhibitors
- Describe methods of reperfusion therapy: percutaneous coronary intervention (PCI) and fibrinolytics

Introduction

- ACS is leading cause of death in adults in United States
- **Ischemia**
  - Heart muscle is deprived of O₂
- **Stable angina pectoris**
  - Brief and temporary ischemia
- **Acute myocardial infarction**
  - Death to a portion of the cardiac muscle
- Morbidity and mortality for ACS in prehospital environment is due to arrhythmias
- Goal of prehospital therapy is directed at limiting size of infarct and preserving left ventricular function
Overview of ACS

- Progression of cardiac disease that results from ischemia
- Coronary artery disease begins with early plaque formation within artery
  - Inflammatory response caused by deposition of lipoproteins that carry cholesterol
  - Rupture can:
    - Cause obstruction of artery by thrombus formation
    - Interfere with blood delivery to heart muscle

Overview of ACS

- Ischemia
  - Reversible process
  - Temporary damage does not occur unless there is prolonged decrease of O₂ supplied to the heart

Overview of ACS

- Stable angina pectoris
  - When O₂ demand of the heart exceeds O₂ supplied to the heart
  - Pain may be relieved with rest or O₂ supplementation
Overview of ACS

- Unstable angina pectoris (UA)
  - Occurs when partially occluding thrombus produces symptoms of ischemia
    - Chest pain
    - May occur at rest or with little provocation
    - Longer in duration
    - May not be relieved by rest or medication
  - Requires immediate attention
  - May cause a non-ST segment elevation myocardial infarction (NSTEMI)

Overview of ACS

- NSTEMI
  - Acute ischemia affects myocardium with no elevation of ST segment on electrocardiogram (ECG)
  - Cardiac enzymes will be elevated
  - Necrosis of cardiac tissue

Overview of ACS

- ST segment elevation myocardial infarction (STEMI)
  - Actual infarction of cardiac tissue secondary to O$_2$ deprivation
  - Cell death occurs
  - Permanent, irreversible damage to cardiac muscle cells
  - Will have elevation of ST segments on ECG
Stable Angina Pectoris

- Angina means “tightening,” not pain
- May be described as squeezing, pressure, burning, heaviness, or bandlike pain
- Typically felt in center of chest or on left side of rib cage

Stable Angina Pectoris

- May radiate to jaw or to left or right arm
- Periodic chest discomfort that occurs when $O_2$ demand exceeds $O_2$ supply of cardiac muscle

Stable Angina Pectoris

- Attacks may become more frequent over time
- Causes of attack:
  - Physical activity
  - Psychologic stress
  - Anemia
  - Arrhythmias
  - Certain environmental conditions
Stable Angina Pectoris

- **Treatment**
  - Rest
  - Supplemental O₂
  - Nitroglycerin administration

- Usually lasts 2 to 5 min, no more than 20 min

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Stable Angina Pectoris

- **Pathophysiology**
  - Caused by reduction in diameter of coronary arteries that supply blood to the heart
  - Long-term deposition of atherosclerotic plaque

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Stable Angina Pectoris

- When heart muscle is stressed by exertion, increased need for O₂ occurs

- Because of decreased diameter of vessels, delivery of oxygenated blood cannot be increased to accommodate heart’s demands

- When patient rests, heart’s O₂ demand is met and ischemia and pain subside
Management of Stable Angina Pectoris

- Follow protocols

- After completing primary assessment, administer O₂
  - Based on presentation, administer via nasal cannula or nonrebreather mask
  - Red blood cells become loaded with O₂ for delivery to the heart muscle
  - Has immediate onset of action
  - Duration is less than 2 min

Management of Stable Angina Pectoris

- Place on cardiac monitor

- Obtain IV access

- 12-lead ECG should be completed

Management of Stable Angina Pectoris

- Aspirin
  - Antiplatelet medication that prohibits platelets from adhering to each other
  - Used for analgesic, antiinflammatory, and antipyretic effects
  - In acute MI, reducing clot formation in coronary arteries is primary concern
  - Prevents clot from growing larger
  - Inhibits platelets from adhering to one another and preventing clots from forming
UA and NSTEMI

- **UA**
  - Chest pain/discomfort that occurs with minimal exertion or at rest
  - May not follow pattern
  - New-onset angina
  - Episode may be longer in duration, more than 20 min

- **NSTEMI**
  - Acute MI with ST segment elevation

UA and NSTEMI

- **Characteristics**
  - Symptoms of angina at rest
  - New-onset exertional angina
  - Recent acceleration of angina
  - Variant angina
  - Post-MI angina (more than 24 hours)
  - May not follow pattern
  - Women may present atypically

UA and NSTEMI

- **Variant or Prinzmetal angina**
  - Form of UA
  - Caused by coronary artery spasm at rest
  - Little atherosclerotic disease is noted
  - Pain is often severe
  - Can display characteristics of a STEMI
UA and NSTEMI

- Pathophysiology
  - Same as STEMI
  - Atherosclerotic changes of coronary artery disease form plaques
    - May rupture and platelet-rich thrombus forms
    - Arterial occlusion causes decreased blood flow to portion of cardiac muscle
    - Ischemia occurs, can lead to infarction

Management of UA and NSTEMI

- Perform thorough history and physical assessment
- Supplemental O₂
- IV access
- 12-lead ECG

Management of UA and NSTEMI

- Aspirin
- Monitor with SCG and pulse oximeter monitor
Management of UA and NSTEMI

- Nitroglycerin
  - Administered by sublingual route
  - Once in body, converted to nitric oxide, a potent vasodilator
  - Venous system dilation reduces amount of blood returning to heart with each heartbeat

- Nitroglycerin
  - Reduces workload of the heart
  - Decreases tension on walls
  - Changes result in amount of O₂ consumed by the heart
  - Causes both arterial and venous dilation

- Vital signs must be monitored before and after each administration

Management of UA and NSTEMI

- Morphine sulfate
  - Used for pain control
  - Opioid agonist
  - Treats refractory pain in ACS
  - Has some vasodilating properties that reduce preload
  - Class I recommendation for STEMI
  - Class IIa recommendation for UA and NSTEMI
### Management of UA and NSTEMI

- **Fentanyl and hydromorphone**
  - Narcotics used for pain management in ACS

- **Beta blockers**
  - Helps ischemic heart by reducing chronotropic and inotropic activity
  - Reduce consumption by the heart
  - Should be administered for UA and NSTEMI unless contraindicated
  - When administered, decreased myocardial damage and decreased long-term complications
  - Should be administered as soon as possible

- **Beta blockers**
  - For IV beta blockers, frequently evaluate patient’s heart rate, BP, ECG monitor
  - Avoid with cocaine abuse
Management of UA and NSTEMI

- Beta blockers
  - Contraindications
    - Signs of heart failure
    - Evidence of low output state
    - Cardiogenic shock or increased risk for cardiogenic shock
    - PR interval more than 0.24 sec
    - Second- or third-degree heart block
    - Active asthma or reactive airway disease

Management of UA and NSTEMI

- Antiplatelets
  - Platelet plugs within coronary arteries cause decreased blood flow to cardiac muscle
  - Aspirin
  - Clopidogrel (Plavix)

Management of UA and NSTEMI

- Anticoagulants
  - Unfractionated heparin or low-molecular-weight heparin (LMWH) inhibit thrombin
  - Heparin
  - LMWH
  - Blood thinners, but do not reduce existing clots
  - Used in ACS
  - Typically administered in ED
Management of UA and NSTEMI

- Glycoprotein IIb/IIIa inhibitors
  - Used in patients with UA, NSTEMI, STEMI
  - Used in conjunction with heparin or aspirin to prevent clotting before and during invasive heart procedures
  - Inhibit platelet aggregation by inhibiting the integrin glycoprotein IIb/IIIa receptor
  - Inhibit final common pathway of platelet aggregation

- HMG coenzyme A reductase inhibitors (statins)
  - When administered within 24 hours after onset of ACS, reduces incidence of:
    - Morbidity
    - Reinfarction
    - Recurrent angina
    - Rehospitalization
    - Stroke

Management of UA and NSTEMI

- Overview of ST segment MI
  - Chest pain with diaphoresis is suggestive of ACS and infarction
  - ST segment elevation in leads I, aV1, V5, and V6
    - Specific for lateral wall STEMI
    - Aggressively treat and transport
ST Segment MI

- Pathophysiology
  - Complete blockage of an artery by ruptured plaque and thrombus formation
  - Decreased blood flow can occur secondary to vasospasm
  - Thrombus or vasospasm actually causes blood flow to the heart to stop or diminish considerably
  - Decreased O₂ delivery causes ischemia
  - After heart cells are damaged, are unable to respond to electrical stimulus

Management of STEMI

- O₂
- Start IV
- Cardiac monitor
- Administer:
  - Aspirin
  - Nitroglycerin
  - Morphine sulfate

Management of STEMI

- Reperfusion therapy
  - Pharmacologic (fibrinolytics) indications:
    - Onset of symptoms less than 12 hours
    - ST segment elevation more than 1 mm in 2+ contiguous leads
    - History suggesting STEMI
  - PCI indications:
    - If PCI can be accomplished within 90 min of onset of symptoms
    - Increased bleeding risk
ACS Primary Goal

● Minimize damage to myocardial cells
  ➢ Can be accomplished by the following medications:
    • O₂ is used to help increase supply of O₂ to ischemic cells
    • Nitroglycerin is vasodilating medication that increases blood flow to coronary arteries
    • Morphine sulfate, fentanyl, and hydromorphone are narcotic pain medications that reduce sympathetic response to pain
    • Aspirin is antiplatelet medication that reduces platelet clumping and formation of thrombus

ACS Primary Goal

● Minimize damage to myocardial cells
  ➢ Can be accomplished by the following medications:
    • Clopidogrel is antiplatelet medication that also inhibits clumping of platelets and affects platelet for its entire life
    • Heparin and LMWH affect clotting cascade and inhibit further clotting at site of thrombus
    • Glycoprotein IIb/IIIa inhibitors also prevent terminal step in platelet aggregation

ACS Primary Goal

● Minimize damage to myocardial cells
  ➢ Can be accomplished by the following medications:
    • Beta blockers reduce heart rate and BP, lowering shear stress on myocardial muscle cells
    • ACE inhibitors reduce volume and BP
    • Fibrinolytics are the only medications that will break an existing thrombus
    • PCI is mechanical opening of blocked artery by either balloon or stent placement