Diagnosis and Management of Acute Coronary Syndrome

One (1.0) Contact Hours

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… Kim Maryniak, PhD(c), MSN, RNC-NIC…

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Purpose

The purpose of this continuing education course for nurses and APRNs is to provide an in-depth review of acute coronary syndrome (ACS), with an emphasis on unstable angina (USA), non-ST-elevation myocardial infarction (NSTEMI) and ST-elevation myocardial infarction (STEMI).

The pharmacological management of ACS, coronary revascularization options, and nursing management of the patient is also covered in this presentation.

Learning Objectives

After successful completion of this course, you will be able to:

1. Define acute coronary syndrome (ACS).
2. Examine ACS modifiable and non-modifiable risk factors.
3. Differentiate between male and female signs and symptoms of ACS.
4. Describe the pathophysiology for unstable angina (UA), non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI).
5. Analyze the pharmacological management in the treatment of ACS.
6. Discuss coronary revascularization procedures and nursing care.

Introduction

Acute coronary syndrome (ACS) is a term used to describe three sub-conditions associated with cardiac ischemia:

- Unstable angina (UA)
- Non ST-elevation myocardial infarction (NSTEMI)
- ST-elevation myocardial infarction (STEMI)

ACS is a form of cardiovascular disease, and is considered a sub-category of coronary artery disease (Sanchis-Gomar, Perez-Quilis, Leischik, & Lucia, 2016).

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Cardiovascular Disease Statistics

- Coronary heart disease is the leading cause of death, and accounts for one in seven deaths in the US, killing over 360,000 people a year
- About 790,000 people in the U.S. have heart attacks each year. Of those, about 114,000 will die.
- About 2,200 Americans die of cardiovascular disease each day, an average of 1 death every 40 seconds.
- Cardiovascular diseases claim more lives each year than all forms of cancer and respiratory disease
- Cardiovascular disease is the leading global cause of death, accounting for more than 17.3 million deaths per year in 2013, a number that is expected to grow to more than 23.6 million by 2030
- For individuals at 40 years old, the lifetime risk of developing CHD is 49% in men and 32% in women; for those reaching age 70 years, the lifetime risk is 35% in men and 24% in women

(American Heart Association [AHA], 2017; Sanchis-Gomar et al., 2016).

Etiology of ACS

ACS can occur if there are embolic clots in the circulation; for example, caused by clots formed in the heart during atrial fibrillation. Arterial inflammation (infection) or vasospasm can also cause transient and/or sustained ischemia. Potentially, coronary arteries can spasm from the use of cocaine or methamphetamines; or increased cardiac workload secondary to thyrotoxicosis, anemia, hypoxemia or fever.

Coronary Anatomy: Layers of the Heart

To better understand the heart, coronary anatomy including layers of the heart and coronary arteries will be discussed.

The **epicardium** is the outer layer of the heart. It contains the coronary arteries and veins, autonomic nerves, and the lymphatic and fat tissues.

The **myocardium** is the thickest part of the heart. It possesses cardiac muscle fibers and cells which assist the heart in its contractility and conductivity.

The **endocardium** is the inner most layer of the heart and contains epithelial cells that form a smooth surface to keep blood from clotting.
(Mancini, 2015).

Coronary Anatomy: The Coronary Arteries

There are two main coronary arteries which impact the circulation to the heart: the right and the left. The coronary arteries then branch off to smaller and smaller arteries which feed the entire heart. The left coronary artery branches into two other vital coronary arteries. The four coronary arteries are: left main coronary artery (LCA), left anterior descending (LAD) artery, left circumflex (LCX) artery, and the right coronary artery (RCA)

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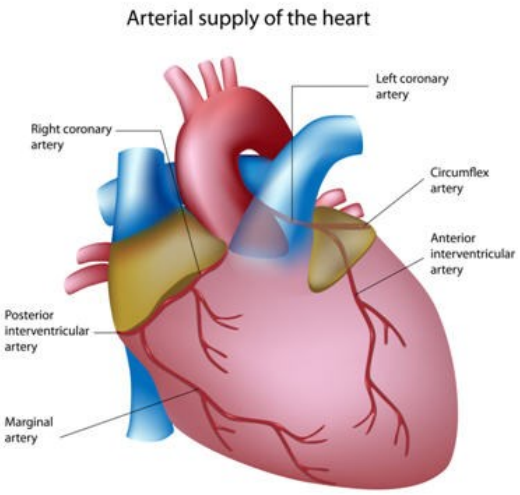
The coronary arteries receive a high oxygen content because the blood is supplied via the coronary sinus. The coronary sinus is located above the aortic valve in the ascending aorta. Milliseconds before the blood makes its way to the coronary arteries, the oxygen-rich blood is pumped out of the lungs, into the left atrium/ventricle and out to the aorta.

The coronary arteries feed each layer of the heart through microvascular vessels which branch off the main coronary arteries. As a result of their anatomical positions, the endocardium is the last layer of the heart to receive oxygen and nutrients.

The safest times for the coronary arteries to receive blood is during diastole, which is when the heart not contracting.
(Mancini, 2015)

Left Main Coronary Artery

The left main coronary artery supplies blood and nutrients to the left side of the heart. The LCA supplies most of the left atrium, left ventricle, interventricular septum, and AV bundles. This artery branches into the left anterior descending artery (LAD) and the left circumflex artery (LCX). Note that the left anterior descending artery (LAD) is also referred to as the anterior interventricular branch of the left coronary artery (see image on the right).



The **LAD** supplies blood and nutrients to the anterior wall of the left ventricle. The LAD artery is mostly epicardial, but can be intramuscular in places. An important identifying characteristic of the LAD artery during angiography is the identification of four to six perpendicular septal branches. These branches, approximately 7.5 cm in length, supply the interventricular septum.

The **LCX** supplies blood and nutrients to the lateral wall of the left ventricle and the entire left atrium. The sinoatrial (SA) node, which is the primary pacemaker of the heart, is supplied by the LCX in approximately 40% of the population.
(Mancini, 2015)

Right Coronary Artery

The right coronary artery (RCA) branches off from the right side of the aorta.

The RCA supplies blood and nutrients to the SA node in 60% of the population as well as the right atrium and ventricle. It also supplies the inferior and posterior walls of the left ventricle; including the atrioventricular (AV) node, which is the back-up pacemaker of the heart.

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The AV node is supplied by the RCA in 90% of the population. An occlusion to the RCA may cause an AV block.
(Mancini, 2015)

Anatomy of the Coronary Arteries

Coronary arteries are made up of three distinct layers: the intima, media, and adventitious layers.

The **intima**, the inner most lining of the artery, has a thin lining of endothelium which contains epithelial cells. In a normal artery, blood flows smoothly through the arterial walls. In ACS, the endothelium is damaged by plaque, microvascular bleeding and clotting takes place and narrows the diameter of the coronary artery.

In addition, the epithelial cells secrete nitrous-oxide to dilate and relax arteries and act as a magnet to the “bad” cholesterol called low-density lipids (LDLs).

The **media** is made up of smooth muscle and elastic connective tissue which allows for dilation and constriction of the vessel.

The **adventitia** is designed to protect the vessel and connect it to other vascular structures.
(Mancini, 2015)

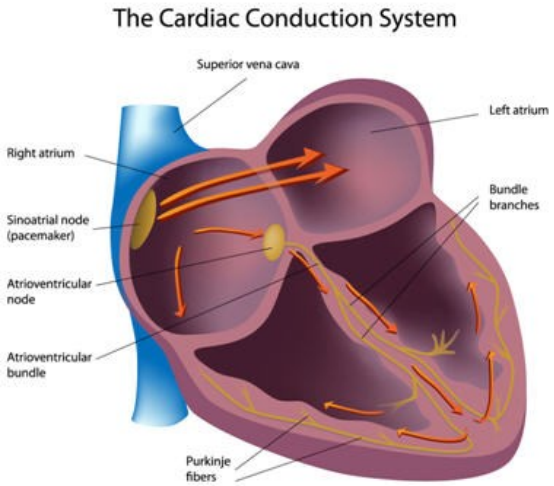
Pacemaker Nodes

Since the SA-node is the primary pacemaker of the heart and the AV-node is the back-up pacemaker to the SA-node, it is dependent on adequate blood flow from the RCA. If the RCA is occluded during ACS, the patient may exhibit atrioventricular (AV) blocks on their ECG.

Collateral Circulation

Over the years, collateral circulation between the main coronary arteries will develop, providing an alternate source of blood supply to the myocardium. For example, if one of the coronary arteries is becoming smaller in diameter due to plaque build-up, the increase of pressure gradient will open the collateral circulation and assist in delivering oxygen and nutrients in and around where the artery is partially occluded

(Seiler, Stoller, Pitt, & Meier, 2013).



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Cardiac Veins

The cardiac veins run parallel to the cardiac arteries and like the superior and inferior vena cava, the cardiac veins return the unoxygenated blood to the right atrium (Mancini, 2015).

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Coronary Blood Composition

Blood contains about 50% plasma, which is primarily made up of water, and 50% blood elements called red and white blood cells, and platelets (Lichtin, 2016).

Risk Factors for Atherosclerosis

In this section, the pathophysiology of atherosclerosis and coronary artery disease (CAD) will be discussed. This will facilitate an understanding of the pathophysiological changes that can lead to acute coronary syndrome.

The risk factors for atherosclerosis include the following:

- Hyperlipidemia and dyslipidemia Hypertension
- Sedentary life-style
- Smoking
- Diabetes
- Increased body mass index (BMI)
- Familial history of heart disease or hypercholesterolemia
- Air pollution
- Age
- Gender
- Autoimmune disorders

(Boudi 2016a)

Plaque Build-Up

In healthy coronary arteries, platelets do not stick to the inner walls of the artery. However, when the intima of coronary artery has been injured through plaque build-up and rupture, the platelets become activated and stick to the site of injury.

Blood flow is significantly reduced or completely occluded and as a result there is more plaque and the diameter of the artery becomes smaller. Since plaque is perceived as a foreign substance in the artery, it has a high probability of rupturing.
(Boudi, 2016a)

Thrombosis Formation

Once the plaque has ruptured, it bleeds and initiates the release of platelets through the clotting process. There is thrombosis/clot formation which further occludes the diameter of the coronary artery and decreases blood supply to the heart.
(Boudi 2016a)

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The Clotting Cascade

To understand how platelets work, one must understand the basics of the clotting cascade. A platelet has a lifespan of approximately 5-10 days. In a healthy person, the normal amount of circulating platelets is 150,000-450,000 per micro liter of blood. The goal of the clotting cascade is to produce numerous chemical reactions to assist platelets to effectively stick to one another to form a fibrin clot/thrombus.

Any time there is active bleeding, a collagen plug is formed and platelets in the circulation adhere to it. Chemical reactions continue and the vessel constricts so more platelets are able to aggregate to one another on top of the collagen plug. This process continues until hemostasis is achieved.
(Boudi, 2016a; Lichtin, 2016)

Importance of Platelet Inhibition

So why is platelet inhibition so important in ACS?

Over time atherosclerotic lesions develop in the coronary artery. The endothelial cells become damaged or dysfunctional during atherosclerosis, which further create an inflammatory environment by attracting more platelets. In return, this makes the lesion larger.

Collagen is present in an injured and diseased atherosclerotic vessel. Collagen snags the platelets and they adhere to each other like Velcro. Chemical reactions continue, which make the platelet to platelet bond stronger.

In addition, if a percutaneous coronary intervention (PCI) is planned, the catheter which is introduced into the coronary arteries typically causes microvascular tears. If the patient is not appropriately anticoagulated prior to or during the PCI, dangerous clots will form.
(Boudi, 2016a)

Formation of Clots

The platelet to platelet bond strengthens during vascular injury when endogenous chemicals such as adenosine diphosphate (ADP) are released.

Thrombin, the result of conversion of fibrinogen to fibrin, and the release of von Willebrand factor from platelets lead to a strong fibrin clot/thrombus.
(Lichtin, 2016)

Test Yourself:

Atherosclerosis consists of the following except:

A. Lipids
B. **Insulin -** Correct!
C. Fibrin

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Symptoms of Coronary Artery Disease

When plaque continues to occlude the coronary artery and/or the coronary artery is constricted, blood carrying the necessary oxygen and nutrients cannot pass through, thus causing ischemia. As you know, ischemia is tissue which is starving for oxygen and therefore causes pain.

Since the coronary perfusion starts at the epicardium and ends at the endocardium, you will see ischemia affected areas in the endocardium first.
(National Heart, Lung, and Blood Institute [NHLBI], 2016)

Presenting Symptoms

Any time a patient presents with right or left sided chest pain, discomfort, pressure or tightness around the chest, they need to be immediately triaged in the emergency department.

Other symptoms that call for immediate attention include pain or discomfort in the shoulder, neck, jaw or back. Atypical symptoms of ischemic chest pain are dyspnea, nausea, vertigo, and diaphoresis. Additional atypical symptoms may include fatigue and upper back and neck pain.

If a person is exhibiting these symptoms out of the hospital setting, 911 should be called immediately. (NHLBI, 2016)

Gender Disparity in Presenting Symptoms

For years, researchers have debated whether the presentation of ACS symptoms differ in men and women.

The symptoms the media has been portraying on television usually have a man grabbing his chest in pain. While this is a common finding in ACS symptoms, females may present with atypical symptoms such as fatigue, upper back and neck pain, shoulder pain, abdominal pain, and nausea. Although angina is a common finding in both men and women, it is important to thoroughly assess for ACS when the presentation includes those atypical symptoms (Gulati, Shaw, & Bairey Merz, 2012).

Studies on Symptoms in Women

There have been numerous studies on gender differences in ACS and CAD, including the Women's Ischemia Syndrome Evaluation (WISE) study. Researchers found women present to the emergency department with angina, just as men do. However, women may also present with vague, atypical symptoms during acute ischemia. The lack of concrete or typical symptoms may lead to women being treated less aggressively with atypical symptoms compared to patients coming in with substernal chest pain.

The results of the trial concluded when females present with vague, multiple symptoms they need to be worked up aggressively (the same as substernal chest pain) for possible cardiac ischemia. Secondly, it found despite the woman’s activity tolerance, frequent chest pain symptoms do signify cardiac ischemia. Finally, more female-based studies are warranted to further the understanding of the symptoms in cardiac ischemia.

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It is important to note that women who present with symptoms of possible myocardial ischemia have a lower probability of obstructive CAD. However, women with ACS have a poorer prognosis (Gulati, Shaw, & Bairey Merz, 2012).

Take Away Points

The take away point from difference in gender symptoms is learning how to appropriately treat the ACS patient. It is important to remember, chest pain should be noted as the cardinal symptom in cardiac ischemia. Atypical symptoms as noted in the research may equally reflect the amount of cardiac ischemia as typically seen in sub-sternal chest pain (Gulati, Shaw, & Bairey Merz, 2012).

Test Yourself:

Atypical chest pain symptoms include right or left side chest pain?

A. True
B. **False –** Correct. Typical chest pain is right and left chest pain. Atypical pain is described as back and shoulder pain as well as nausea

Modifiable vs. Non-Modifiable Risk Factors

We cannot modify our gender, familial history, or age, but the older we become, the more calcification there is in our coronary arteries and they become less elastic.

Modifiable risk factors are avenues where patients can change their lifestyle and reduce their risk of ACS.
(Boudi, 2016b)

Modifiable Risk Factors Modifiable Risk Factors: Smoking

By itself cigarette smoking increases the risk of coronary heart disease (CHD) because it increases blood pressure and the tendency of blood to clot.

Additionally, women who take oral contraceptives and who smoke increase their risk of CHD more than those women who take oral contraceptives and do not smoke.
(Boudi, 2016b)

Modifiable Risk Factors: Hypercholesterolemia

Two types of cholesterol are low-density lipoprotein (LDL) and high-density lipoprotein (HDL). LDL cholesterol is considered “bad” and when there is too much, it will slowly build up on the intima of the

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arteries. As a result, because LDLs are ‘sticky,’ it is more likely circulating platelets will adhere to them.

HDL cholesterol is considered “good” and studies have shown high levels of HDL are actually cardioprotective. Clinicians believe HDL helps remove LDL from the blood stream (coronary arteries as well).
(Boudi, 2016b)

Modifiable Risk Factors: Hypertension

Over time high blood pressure causes damage to the arteries and the heart, specifically the coronary arteries. The increased pressure on the arteries causes microscopic tearing. The result of the body fixing these tears is scar tissue. The more scar tissue the arteries have the more likely cholesterol and platelets are able to stick to the walls.

Studies have shown a strong link between hypertension and myocardial infarction (MI). In the United States, even individuals with high normal blood pressure (systolic readings of 130-139 mmHg, and/or diastolic readings of 85-89 mmHg), have two times the risk of developing cardiovascular disease (Boudi, 2016b).

Modifiable Risk Factors: Diabetes

People with diabetes (especially type II) are greatly at risk for developing CHD because type II diabetic patients typically are already obese and do not regularly exercise. Diabetics are two to eight times as likely to have heart disease or stroke as those who do not (Boudi, 2016b).

Modifiable Risk Factors: Obesity

A closer look at obesity statistics shows over one-third of US adults are obese, which correlates to approximately 75 million adults. The statistic grows grimmer as you look at the 2—19-year-old population. It is estimated 23,500,000 children are overweight or obese (Centers for Disease Control and Prevention [CDC], 2016). Obesity is linked to heart disease, stroke, type II diabetes, and cancer. Obese patients are at risk for STEMI at younger ages, and also have higher mortality rates in hospital following a STEMI (Boudi, 2016b; CDC, 2016).

Body Mass Index (BMI)

Body mass index (BMI) is used to measure body fat based on height and weight in adults. It should be noted it may overestimate the body fat in athletes who have a muscular build. Conversely, it may underestimate body fat in older persons who have muscle loss. To compute your BMI go to https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/english_bmi_calculator/bmi_calculator.html

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Weight	BMI
Underweight	Below 18.5
Normal	18.5-24.9
Overweight	25.0-29.9 **high risk for CHD**
Obese	30.0 and above **high risk for CHD**

https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/english_bmi_calculator/bmi_calculator.html

Chest Pain: Standard Orders

Any time a patient presents with chest pain/discomfort a number of tests are ordered. Oxygen should be applied and intravenous (IV) access should be established immediately. Standard orders usually include labs such as:

- Cardiac enzymes
- Complete blood count (CBC)
- Comprehensive metabolic profile (CMP)
- Cholesterol and coagulation studies

The physician will also order a stat ECG and a chest x-ray. A brief history and assessment is required. All of these will assist in determining if there has been acute ischemia and then further differentiating which sub-condition of ACS the patient is presenting.
(Davis et al., 2012)

Cardiac Enzymes

The cardiac enzymes which leak into the circulation during cardiac ischemia or injury are:

- Troponin-I
- Creatine kinase (CK)
- Creatine kinase-MB (fraction of CK)

Cardiac Enzymes: Troponin-I

Troponin-I is the most sensitive cardiac enzyme. It spills into the blood when there is ischemia/injury to the cardiac cells, as seen in NSTEMI and STEMI. An elevated troponin will assist in risk stratification as well as appropriate treatment for the patient.

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Troponin-I can be detected in blood as soon as two to four hours after ischemia has started. However, serial tests are needed because for some patients, Troponin-I is not detectable until 8-12 hours after initial ischemic event and peak at 18-24 hours.

Even though troponin levels are specific to cardiac ischemia, alone they cannot be diagnostic for ACS.

The following is a list of conditions in which a troponin level may be elevated in the absence of acute coronary syndrome:

Heart failure, pulmonary embolus, myocarditis, pericarditis, acute stroke, severe pulmonary hypertension, arrhythmias, myocardial contusion, extreme exertion, chemotherapy, cardioversion, critical illness/sepsis, aortic dissection and burns.
(Davis et al., 2012; Schreiber, 2017)

Cardiac Enzymes: CK & CK-MB

CK is an enzyme that is present in brain, heart, and skeletal muscle. The rises in CK are not **specific** to myocardial damage but assist in diagnosing of ACS.

CK-MB enzymes are more specific to the heart and when this enzyme is elevated, it is caused by myocardial injury. CK-MB usually rises within four to six hours of damage and peaks at 18-24 hours. (Davis et al., 2012; Schreiber, 2017)

12 Lead ECG

As the labs are being drawn and resulted, the 12-lead ECG should be completed and interpreted within the first 10 minutes.

When the patient is triaged in the field by emergency medical services (EMS) personnel, a 12-lead ECG should be completed as soon as possible for those patients having signs and symptoms of ACS. (Davis et al., 2012)

Acute Coronary Syndrome

The sub-conditions of ACS are unstable angina (UA), non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). Since the treatment modalities are typically the same for UA and NSTEMI, the two sub-conditions will be discussed together. The last sub-condition to be discussed is a STEMI.

Angina

Chest pain is described as angina which is further classified as stable or unstable.

Stable angina is reproducible and occurs during physical exertion or emotional stress and can be relieved by resting or with nitroglycerin.

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Unstable angina occurs with minimal exertion or at rest and increased doses of nitroglycerin are needed to relieve the pain.
(American College of Cardiology/American Heart Association (ACC/AHA), 2014)

Unstable Angina (UA)

There are certain symptoms and clinical data the patient presents with that determine their specific UA, NSTEMI, or STEMI ACS diagnosis.

Unstable angina (UA) is associated with chest pain, without ST-segment elevation on the 12- lead ECG and the serial laboratory cardiac enzymes will remain within normal limits. However, it is suggestive that plaque has ruptured in the coronary artery, but did not occlude the artery to cause widespread cardiac ischemia.
(ACC/AHA, 2014)

NSTEMI

NSTEMI is defined as chest pain symptoms with **elevated troponin levels**.

The 12-lead may show ST-segment depression and T-wave inversions, but it will **NOT** exhibit ST-segment elevations.

Like UA, during NSTEMI there is a rupture of plaque within the coronary artery. The difference is that the heart is experiencing more ischemia as evidenced by abnormal troponin levels.
(ACC/AHA, 2014)

Management Options for UA/NSTEMI

Next is a detailed look at UA/NSTEMI invasive and noninvasive interventions as supported by the American College of Cardiology/American Heart Association (ACC/AHA) guidelines (2014).

Non-Invasive Management Options for UA/NSTEMI

Patients who present with UA/NSTEMI symptoms should be transported to an emergency department immediately. They should also be given an aspirin as soon as possible. If tolerated, they should continue on aspirin therapy (and dual antiplatelet therapy) indefinitely.

If non-invasive therapy, non-percutaneous intervention (PCI) is selected, then aspirin and antiplatelet therapy, such as clopidogrel (Plavix™) or ticagrelor (Brilinta™), should be initiated and administered up to at least 12 months.
(ACC/AHA, 2014)

Alternative Non-Invasive Management Options for UA/NSTEMI

If a patient cannot tolerate or has an allergy to aspirin, loading and maintenance dose of clopidogrel (Plavix™), pursagrel (Effient™) or ticagrelor (Brilinta™) should be administered (ACC/AHA, 2014).

Warning:

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The patients who present to the hospital with UA/NSTEMI and who have a prior history of stroke and/or transient ischemic attack (TIA) and a PCI is planned should NOT be given prasugrel (Effient™), because it has shown to cause harm in patients (ACC/AHA, 2014).

Invasive Management Options for UA/NSTEMI

When a patient is admitted with non-symptomatic or symptomatic UA/NSTEMI, a PCI is usually indicated. If the ischemia has not penetrated all the layers of the heart, the patient typically is considered low risk and may not need to go for a PCI immediately. However, they should have a PCI within 12-24 hours from admission. It is estimated that approximately half of all PCI procedures are done with patients experiencing UA or NSTEMI. If the patient starts to have chest pain and becomes hemodynamically unstable, the cardiologist should be notified and plan for the patient to have a PCI immediately.
(ACC/AHA, 2014)

Pharmacological Preparation for PCI

If the patient is medium to high risk and undergoing an invasive therapy such as a PCI, an aspirin should be given as soon as possible and continued indefinitely as patient tolerates.

AND

Before the PCI the patient should also receive a loading dose of antiplatelet therapy (ACC/AHA, 2014).

Pharmacological Management Post PCI

If patients were treated with a bare metal or drug eluting stent, aspirin should be continued indefinitely and antiplatelet therapy for at least 12 months (ACC/AHA, 2014).

Test Yourself:

The medication prasugrel (Effient) can be administered if a patient has a history of a stroke or transient ischemic stroke (TIA).

A. True
B. **False –** Correct. Studies have shown prasugrel has caused increased rate of bleeding in those patients with a history of stroke and TIA.

Progression of a UA/NSTEMI to STEMI

If UA/NSTEMI is not appropriately treated or ignored by the patient and they do not seek medical treatment, ischemia and a larger infarction may occur. This is known as a STEMI.

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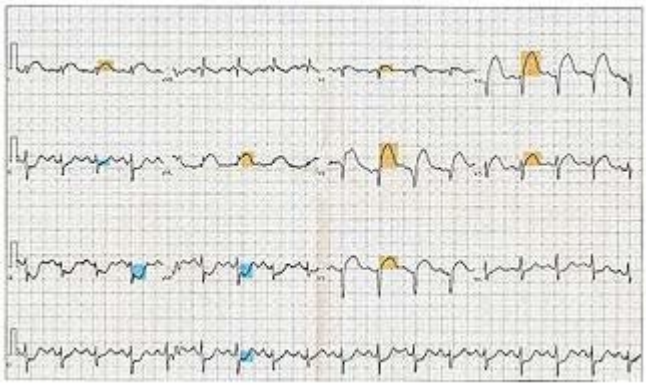
STEMI

The presentation of a STEMI may be similar to a NSTEMI; but on a 12-lead ECG, only STEMI presents with ST-segment elevation. This elevation reflects the ischemia that penetrates all three layers of the heart in STEMI, with indicators of myocardial necrosis.
(American College of Cardiology Foundation/American Heart Association [ACCF/AHA], 2013).

Presentation of STEMI

A patient admitted with a STEMI exhibits chest pain/cardiac symptoms, ST-segment elevation on a 12-lead ECG, and abnormal troponin cardiac enzyme is reflected in the lab work.

This is the most severe type of myocardial infarction and requires immediate pharmacological, percutaneous, or surgical intervention.
(ACCF/AHA, 2013)



12-lead ECG showing ST-Segment elevation (orange) in I, a VL and V1-V5. (Wikipedia, 2012)

STEMI Management

The goal is the same for resolving a NSTEMI as it is for a STEMI, which is to restore blood flow to the myocardium as soon as possible. However, with a STEMI, the occlusion of the artery is larger and the ischemia through the layers of the heart is more widespread.

The more time it takes to dilate and re-perfuse the artery, the greater mortality of the patient.

Three ways a blocked artery can be restored is through fibrinolytic therapy, PCI, or a coronary artery bypass graft (CABG) surgery.
(ACCF/AHA, 2013).

Diagnosing STEMI

When a patient is triaged in the field or in the emergency department for chest pain symptoms, a 12 lead ECG is taken immediately. First medical contact is considered either when assessed by

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emergency medical services or triage in a medical center. If there are indicators of a STEMI (positive troponin-I & ST-segment elevation), they are considered a high-risk ACS patient and should be taken to the cardiac catheterization (cath) lab immediately. If the patient is taken to a facility where they offer PCI, then the time from first medical contact to device time should be within 90 minutes (ACCF/AHA, 2013).

Many medical centers have what is called a “code STEMI,” which when activated, appropriate staff and resources are immediately available for the patient.

STEMI Risk Stratification

If a hospital does not have a PCI capability, the emergency physician must consider alternate options while weighing the risk to benefit ratio of the individual patient.

The healthcare team needs to assess duration of symptoms and the mortality risks of the STEMI versus the individual patient risk to receiving fibrinolytic therapy.

If the patient is a high risk STEMI, which is defined as an anterior MI, exhibits signs and symptoms congestive heart failure or cardiogenic shock, has a high bleeding risk and/or presents four hours after initial symptoms; then a decision to transfer this patient would be more beneficial than in prescribing anti-thrombolytic.
(ACCF/AHA, 2013)

Fibrinolytic Therapy

Fibrinolytic therapy is appropriate to use in situations where the patient will not be able to have a PCI within 90 minutes from time they had first medical contact.

Tecectplase (TNKase™), reteplase (rPA), and alteplase (tPA), are examples of fibrinolytics which are fibrin-specific agents, preferred for use in a STEMI which result in rapid coronary reperfusion. It has been studied that patients have better outcomes if fibrinolytics are administered earlier, within one to two hours of symptom onset. However, they can be given within 12 hours of the onset of symptoms. If use of fibrinolytics is the chosen therapy, the goal for the patient is to receive it within 30 minutes from when they were had first medical contact.

If the patient has a contraindication to fibrinolytics and is not able to be transferred to a PCI within 90 minutes, then it is appropriate for the emergency physician to transfer the patient to the PCI facility anyway, with the goal of PCI within 120 minutes of first medical contact.
(ACCF/AHA, 2013)

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STEMI: Stenting vs. CABG

Historically, if the patient had occlusion in the LAD, they were usually sent to surgery for a coronary artery bypass graft (CABG). The reasoning behind this is the LAD perfuses a large portion of the left ventricle and if re-occluded would cause significant myocardial damage.

However, it is recommended the cardiologist perform the PCI and if anatomy of the LAD is associated with low risk PCI complications and the patient will be compliant with long-term antiplatelet therapy, then the LAD may be stented. It is also recommended that the patient be followed up with coronary angiography between two and six months after their LAD was stented.

CABG generally has a limited role in the acute phase of STEM, but may be indicated for cardiogenic shock, failed PCI, for coronary anatomy not amenable to PCI, with the surgical repair of a mechanical defect, with ongoing or recurrent ischemia, severe heart failure, or other high-risk features.
(ACCF/AHA, 2013)

Test Yourself:

Which ACS sub-condition is defined as having positive troponin-I, but not ST-segment elevation?

A. Unstable angina
B. **NSTEMI –** Correct.
C. STEMI

12 Lead ECG

As stated earlier, the 12-lead ECG should be completed and interpreted within the first 10 minutes. When the patient is triaged in the field by emergency medical services (EMS) personnel, a 12-lead ECG should be completed as soon as possible for those patients having signs and symptoms of ACS. If the 12-lead ECG shows ST-segment elevation, advanced notification (e.g.. code STEMI) to the emergency department is warranted.
(Davis et al., 2012)

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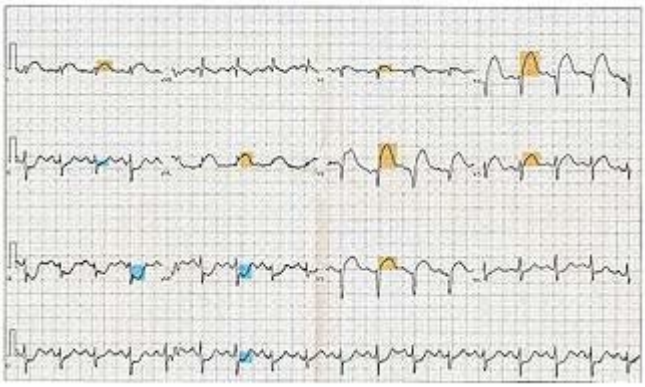

12-lead ECG showing ST-Segment elevation (orange) in I, a VL and V1-V5. (Wikipedia, 2012)

ST-Segment Changes on 12-Lead ECG

Any ST-segment depression or elevation indicate some type of ischemia and should be considered a "red flag." Routine 12-lead ECGs should be ordered as well if the patient experiences any chest discomfort.

ST-segment changes in an ECG reflect ischemia to the heart. ST-segment depression shows a smaller degree of blood supply deficit compared to ST-segment elevation which encompasses a larger ischemic area. In either ST-segment depression or elevation requires immediate medical evaluation.

Other changes in the ECG which signify cardiac ischemia are T-wave inversions, peaked positive T-waves, or Q waves.
(ACCF/AHA, 2013; Davis et al., 2012)

Summary of 12-Lead ECG Changes

ST-segment changes are considered substantial when there is a change greater or equal to 1 mm in two contiguous leads (or there is a new left bundle branch block); however, elevation of greater or equal to 0.5 mm is considered a risk.

Coronary Artery	Myocardial Infarction	Leads Affected
Right Coronary Artery (RCA)	Inferior Wall	II, III, aVF
Left Anterior Descending (LAD)	Anterospetal Wall	V1 – V3
Left Circumflex	Anterolateral Wall	I, aVL, V4 – V6

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Left Main	Anterior Wall	V1 – V6
Proximal Right	Posterior Wall	V1 –V3

(ACCF/AHA, 2013; Davis et al., 2012)

Test Yourself:

If there is an occlusion of the LAD, then the nurse would see ST-segment elevation in which of the following leads:

A. II, III, aVF
B. **V1-V3 -** Correct! If there is an occlusion of the LAD, then leads V1-V3 will show ST-segment elevation. aVL,
C. V4-V6

Risk Stratification

The nurses play a vital role in gathering information from the patient which helps determine the risk for poor outcomes. An astute assessment can mean the difference between minutes to beneficial treatment instead of hours.

Risk Stratification: Using TIMI

The Thrombolysis in Myocardial Infarction (TIMI) is the most commonly used scoring method to determine patients cardiac risk.

The TIMI risk score helps the physician quickly and accurately identify which unit the patient should be admitted to, and identify and order appropriate medications. The higher the score the greater risk of death, therefore early and aggressive treatments may be considered with a high score in comparison to a low score.

Each of the following TIMI selected risk factors carries a risk of one point. The risk factors are:

Risk Factors
Greater or equal to 65 years of age
History of coronary stenosis (greater or equal to 50%)
ST-segment changes greater or equal to 0.5mm
At least two episodes of ischemic symptoms in 14 hours
Use of aspirin in the last seven days

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Elevated cardiac biomarkers
3 or more risk factors of CAD

For more information on TIMI scoring go to:www.TIMI.org

(ACCF/AHA, 2013; Davis et al., 2012)

Pharmacological Management

As the treatment for ACS continues, let's take a look at the pharmacological management of ACS.

When a patient presents with cardiac symptoms, labs are drawn and a 12-lead ECG is completed. Through these labs and ECG interpretation, the healthcare team can obtain more detailed clinical information which will determine the sub-condition of ACS. Thereafter, more specific and appropriate treatment can then be initiated.

The primary goal is to stop myocardial ischemia and restore blood flow to the myocardium. In part, this can be done with certain types of medications.

Types of Drugs Used

The primary medications which are administered during the course of an ACS event are:

- Oxygen
- Aspirin
- Nitrates
- Analgesics
- Beta-blockers
- ACE-inhibitors/ARBs
- Antiplatelets
- Anti-fibrinolytics

(ACCF/AHA, 2013; Davis et al., 2012)

Oxygen Therapy

Oxygen should be administered to patients with shortness of breath, signs of heart failure, or cardiogenic shock.

Routine oxygen therapy has not been proven beneficial in those patients who are admitted with uncomplicated ACS and who are WITHOUT signs and symptoms of hypoxemia and heart failure.

Always remember to use oxygen cautiously in patients with chronic obstructive pulmonary disease (COPD), as the respiratory drive could be suppressed by high levels of oxygen, although this issue is controversial.
(ACCF/AHA, 2013; Davis et al., 2012)

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Aspirin Therapy

Aspirin inhibits platelet aggregation, which is the process of platelets sticking or clumping together. In ACS, platelet aggregation is dangerous because it can make the plaque or thrombosis larger, therefore occluding more blood flow to the heart.

The AHA recommends an aspirin dose between 160 – 325 mg chewable/non-enteric coated. Aspirin should be administered to all patients who are suspected to have ACS, unless they are allergic or intolerant to aspirin or have active gastrointestinal bleeding.

If the patient does complain of nausea and vomiting and is unable to take medication by mouth, aspirin 300 mg suppository is recommended.
(ACCF/AHA, 2013; Davis et al., 2012)

Nitroglycerin Therapy (NTG)

Nitroglycerin (NTG) is a vasodilator which has a greater affinity for veins than arteries. It helps to reduce both preload and afterload in the vasculature system, which will decrease the overall workload of the heart.

Nitroglycerin is contraindicated in patients whose systolic blood pressure (SBP) is < 90 mmHg or >30 mmHg below baseline heart rate (HR) <50 or >100 in the absence of heart failure, or with right ventricular infarction. In addition, nitroglycerin should not be administered if the patient has taken a phosphodiesterase inhibitor (e.g. Viagra™) within 24-48 hours. Phosphodiesterase inhibitors keep nitric oxide in the veins (which help cause vasodilation) and administering nitroglycerin would potentiate a substantial if not life-threatening hypotension.

Nitroglycerin should be used cautiously in patients with an inferior myocardial infarction. Patients with an inferior MI need to have adequate hydration (preload) on board so they do not become hypotensive.

Nitroglycerin preparation varies and can be administered sublingual, aerosol, topical, by mouth and intravenously.

The effectiveness of nitroglycerin is not an indicator ACS or does not exclude ACS. It is known to rid patients of both chest and GI discomfort.
(ACCF/AHA, 2013; Davis et al., 2012)

Morphine Sulfate

The next medication to consider in ACS is intravenous morphine sulfate. Morphine sulfate is the narcotic of choice for chest discomfort especially in STEMI patients who are not responding to nitroglycerin. If morphine sulfate does alleviate the patient's chest pain, it is important that healthcare providers do not assume adequate perfusion to the heart has been restored. In other words, morphine sulfate may have stopped the pain, but may not have necessarily dilated the coronary artery and restored adequate perfusion.

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Studies in patients who were diagnosed with UA/NSTEMI, have noted that morphine sulfate may have some potentially adverse side effects, although this controversial. Therefore, it is recommended to use cautiously with this patient population, but may be used in the absence of contraindications. (ACCF/AHA, 2013; Davis et al., 2012)

How Do Antiplatelets Work?

A thrombus in the coronary artery leads to an imbalance of oxygen supply and demand. The imbalance is based on the size of the thrombus (lesion).

Antiplatelets work on the clotting cascade to inhibit or block chemical reactions from happening to keep platelets from sticking to one another and as a result, form a thrombus (making the occlusion larger). Therefore, it makes the platelets “slide” right on past the thrombus.

Potent platelet inhibitors are used in combination with aspirin for high risk ACS patients and those undergoing PCI.

The current FDA approved antiplatelets are:

- Aspirin
- Ticlopidine (Ticlid™)
- Clopidogrel (Plavix™)
- Prasugrel (Effient™)
- Ticagrelor (Brilinta™)

(ACC/AHA, 2014; ACCF/AHA, 2013)

Risks of Antiplatelet Therapy

There is a risk of bleeding for any antiplatelet prescribed.

Compared to aspirin, gastrointestinal bleeds are less common with the use of clopidogrel (Plavix™).

It is recommended that a complete blood count (CBC) be monitored at discharge as well as every six months.
(ACC/AHA, 2014; ACCF/AHA, 2013)

Thienopyridines: First and Second Generation

Ticlopidine (Ticlid™) was the first-generation thienopyridine approved by the FDA. However, it possesses potential adverse effects such as neutropenia and thrombotic thrombocytopenia purpura (TTP). In addition, it requires a complete blood count (CBC) to be monitored every two weeks for the first three months. For these reasons, ticlopidine is not widely used.
(Willerson & Holmes, 2015)

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Plavix

A second–generation thienopyridine called clopidogrel (Plavix™) was created and approved by the FDA, which did not require frequent CBC monitoring.

Clopidogrel (Plavix™) one time loading dose is given in the event the patient is going to the cath lab for a PCI. If a stent is placed, the patient will be prescribed to take this once a day for at least 12 months.

Plavix™ is the most commonly prescribed antiplatelet.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Winning Combinations: Aspirin and Plavix

Aspirin and clopidogrel (Plavix™) are seen prescribed together because dual platelet therapy is more effective than monotherapy. Aspirin only targets one part of the clotting cascade and has little or no effect on the other clotting cascades.

Clopidogrel (Plavix™) does not have a reversal agent and should be stopped five days before coronary artery bypass grafting (CABG).
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Plavix Resistance

There have been instances of clopidogrel (Plavix™) resistance that have been noted in ACS patients. This resistance carries high probabilities of restenosis. The resistance is caused by how it is broken down in the body.

Clopidogrel (Plavix™) requires the metabolism of the cytochrome P450 to activate it in the body. If the patient has genetic varients of the cytochrome P450, then clopidogrel (Plavix™) may not be as effective and will have variability of efficiency.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Thienopyridines: Third Generation

Prasugrel (Effient™) was approved by the FDA as a third generation thienopyridine. It is similar to clopidogrel (Plavix™). However, it has shown to significantly benefit subpopulations of patients, such as those with diabetes).

Parsugrel (Effient™) has a loading dose and a maintenance dose is indicated for patients undergoing a PCI.

Compared to clopidogrel (Plavix™), prasugrel (Effient™) has a quick onset of 30 minutes and greater patient to patient predictability because it does not need the cytochrome 450 to metabolize the medication. Due to its quick onset, patients who immediately need to go to the cath lab, as in a STEMI, may be given prasugrel (Effient™) more than Plavix™.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

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Monitoring During Platelet Therapy

Patients undergoing antiplatelet therapy should always be monitored carefully. Some studies have shown an increase of bleeding with prasugrel (Effient™).

The FDA added a black box warning on specific patient populations such as low-weight or elderly patients (75 years old or older) and those with a previous or current stroke or TIA.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Discontinuing Prasugrel

If a CABG (or other surgery) is indicated for a patient during the 12 months they are on prasugrel (Effient™), therapy should be stopped at least seven days before the surgery, as there is no reversal agent for this medication. The patient's cardiologist should be contacted prior to discontinuing this medication.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Ticagrelor

Ticagrelor (Brilinta™) is the newest antiplatelet approved by the FDA in the summer of 2011. Like the other antiplatelet medications, its purpose is to decrease further myocardial damage and death in patient with ACS.

Tricagrelor (Brilinta™) is similar to prasugrel (Effient™) in that it does not need the cytochrome P450 to become active. Therefore, it has consistent patient to patient predictability.

Studies have shown ticagrelor (Brilinta™) is superior to clopidogrel (Plavix™) when aspirin was added to the anitplatelet regime. It should be noted that aspirin dosages greater than 100 mg/day should not be prescribed, because it may decrease the effectiveness of ticagrelor (Brilinta™) .

Ticagrelor (Brilinta™) compared to clopidogrel (Plavix™) had slightly more incidence of bleeding in the overweight patient, dyspnea (without changes in pulmonary functioning) as well as ventricular pauses (greater or equal to three seconds). The mechanism causing dyspnea and ventricular pauses was not able to be identified.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Glycoprotein (GP) IIb/IIa

It is recommended that glycoprotein (GP) IIb/IIIa inhibitors should be started at the time of the PCI (Georger & Dangas, 2010). GP IIb/IIa inhibitors are considered antiplatelets and are used in the treatment of ACS.

Three common GP IIb/IIIa inhibitors are:

- Abciximab (Reopro™)
- Tirofiban (Aggrastat™)

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- Eptifibatide (Integrilin™)

These intravenous medications are used during ACS. Specifics for administration are dependent upon the patient's ACS diagnosis and physician preference.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Fibrinolytic Therapy: t-PA

Fibrinolytic therapy is only appropriate if the patient is diagnosed with a STEMI. It should be only considered if the patient cannot undergo a PCI (due to transfer time) or there are no contraindications to fibrinolytics.

T-PA is administered through an intravenous line. It is relatively easy to administer, causes less noncerebral bleeding, and results in quicker coronary reperfusion than fibrinolytics.

With any fibrinolytics there are contraindications. T-PA has the following absolute contraindications:

- Any known intracranial hemorrhage or a known structural vascular lesion (AVM)
- An ischemic stroke with three months EXCEPT acute ischemic stroke within three hours
- Suspected aortic dissection
- Any active bleeding/bleeding disorders (except menses)
- Significant closed head trauma within three months

(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

The Use of Beta Blockers

Beta-blockers can be administered during the initial work up process because this type of medication decreases myocardial oxygen consumption by lowering the heart rate and contractility.

It is recommended that a beta-blocker be prescribed on discharge, and may be reportable for accreditation. If it is contraindicated due to a slow heart rate or low systolic blood pressure, then the physician must be the one to document the contraindication.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

Ace Inhibitors and Angiotensin Receptor Blockers

Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) prevent the constriction of vessels as well as the reabsorption of sodium and water by the kidneys.

As a result, there is less preload and afterload, and the workload of the heart is decreased.

Clinically the patient will have decreased blood pressure. It is recommended that an ACE-inhibitor or ARB be prescribed if there is left ventricular systolic dysfunction, and may be reportable for accreditation.

(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

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Test Yourself:

A patient may have resistance to colegipril (Plavix) due to _____.

A. **Cytochrome P450 -** Correct! Cytochrome P450 assists in the metabolism of Plavix, therefore dysfunction of cytochrome P450 will affect the drug availability in the body.
B. Mitochondria dysfunction
C. Continued aspirin therapy

Cardiac Catheterization

Cardiac catheterization is a surgical option for the management of ACS. PCIs include, but are not limited to the following:

- Percutaneous coronary angioplasty
- Coronary stent placement
- Coronary atherectomy

Percutaneous Coronary Angioplasty

Angioplasty enlarges the diameter of the coronary artery as well as compressing the plaque against the artery wall.

This procedure is appropriate in patients who have single or double vessel blockages greater than 70% stenosis.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

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Angioplasty Demonstration

Click on the video below to view an angioplasty demonstration. (Video available in the online version of the course, slide 126)

Stent Placement

Successful coronary stent placement will provide complete revascularization, without the need for open heart surgery.

The stents are composed of stainless steel mesh which assist in keeping the artery patent.

Bare metal stents (BMS) and drug eluting stents (DES) are the two categories of stents used in patients who require antiplatelet therapy for an extended period of time.
(ACC/AHA, 2014; ACCF/AHA, 2013; Willerson & Holmes, 2015)

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PCI With Stent Placement

This slide exhibits a video of RCA before and after a PCI with stent placement. (Video available in the online version of the course, slide 127)

Coronary Atherectomy

Coronary atherectomy is the removal of atheromatous, which is a mixture of lipids and cellular material occluding the coronary artery.

Post-Catheterization Hemostasis

After a cardiac catheterization, it is necessary to restore hemostasis. This can be challenging when multiple medications are used to inhibit platelet aggregation. Additionally, cardiac catheterization can be done either through the femoral or radial artery. There are two ways to achieve hemostasis, that is by manual compression or the use of an artery closure device.

Manual Compression & Artery Closure Devices

Manual compression is a type of procedure to obtain hemostasis after a PCI.

Before manual compression an activated clotting time (ACT) must be drawn and if prolonged, notify the cardiologist before removing the catheter.

Manual pressure should be held over the puncture site for at least 10 minutes and up to 20 minutes or greater depending if the vessel is venous or arterial and the amount of anticoagulants the patient has received.

Artery closure devices have had varying studies demonstrating their superiority to manual compression in clinical trials, however real-world practice continues to reflect closure device success rates.

There are manyFDA-approved artery closure devices currently on the market. Some of these include:

- Perclose®
- Angiosea®
- Starclose®
- VASCADE Vascular Closure System (VCS)®
- Celt ACD®
- QuickSeal®

Each of these are deployed differently. Some devices use sutures or clips at the insertion site, some use a collagen plug which is inserted into the catheter insertion site, and others use a sealant. If a device is used at your facility, it is important to know which device is utilized, and the post-management strategies.

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Assessments Post Heart Catheterization

The nurse needs to assess the catheter insertion site, which may be a groin or wrist, depending on the type of catheterization done. At the puncture site it is important to assess for tenderness, pain, swelling, bleeding and new bruits. These signs and symptoms are indicative of bleeding within the site that needs to be addressed quickly.

Many hospitals have post-PCI orders which include when to call the physician as well as when to apply a compression device.
(Naidu, et al., 2012)

Assessments of Peripheral Pulses

A nurse should always assess peripheral pulses. There should always be a pulse, even if a compression device is used.

The peripheral leg or arm should appear blanchable and warm to the touch.

Marking the peripheral pulses and noting their strength before the procedure is helpful to determine baseline and should be reported off to the nurse taking care of the patient.
(Naidu, et al., 2012)

Cardiac Cath Complications

During a cardiac catheterization, there are many femoral artery complications that the nurse needs to be able to recognize and know how to intervene (Bhimji, 2016).

The vascular complications which will be discussed are:

- Hemorrhage/vessel laceration
- Hematoma
- Retroperitoneal hemorrhage
- Pseudoaneurysm
- Arteriovenous fistula
- Vessel thrombus
- Neural damage
- Infection

Hemorrhage

Procedural factors which increase the risk of bleeding include increased catheter size, the use of GB IIa/IIIa inhibitors, longer procedure duration, use of a vascular closure device, and heparin use after procedure (which is currently not recommended).

Patient-associated factors which increased the risk of bleeding were age (older), female, severe renal impairment. Interestingly, diabetes is linked to a lower risk of bleeding.

Material protected by copyright

(Bhimji, 2016)

Post-Operative Complications: Vessel Laceration

Vessel laceration is caused from the catheter entering and puncturing through the artery. Spasms of the vessel may occur during catheterization. Strategies to prevent spasms, as well as time for the spasm to resolve are important to avoid vessel laceration.

If this is not identified at the time, a patient may develop more severe vascular complications such as a retroperitoneal hemorrhage or an arteriovenous fistula.
(Bhimji, 2016)

Post-Operative Complications: Hematomas

A hematoma is a bleed into the groin or thigh. It is usually hard on palpation. The patient will usually complain of pain at the insertion site. A large hematoma is defined as > 5 cm in diameter.
(Bhimji, 2016)

Post-Operative Complications: Pseudoaneurysms

A pseudoaneurysm is caused by a puncture to one of the weaker walls of the branch in the femoral artery. This causes the area to bleed in the tissue forming a wall around the artery. The patient will commonly complain of pain and the nurse may auscultate a new bruit or feel swelling at the puncture site.
(Bhimji, 2016)

Post-Operative Complications: Arteriovenous Fistulas

An arteriovenous fistula is caused by a puncture to both the vein and the artery from which they “communicate” with each other. This complication is best diagnosed with an ultrasound. (Bhimji, 2016)

Post-Operative Complications: Neural Damage

Neural damage can be inadvertently caused by nurse applying excessive force to the nerve, while the cardiologist is accessing the femoral site or when there is compression to the tissue and nerves as seen in pseudoaneursym and retroperitoneal hematomas. The patient will exhibit pain and tingling at the groin or wrist site. The patient may have difficulty ambulating or decreased patellar tendon reflex for a groin site, or difficulty moving the hand or wrist with a radial site.
(Bhimji, 2016)

Post-Operative Complications: Hospital Acquired Infections (HAIs)

An infection in puncture site is rare, but possible. This may be caused by a compromise is sterile technique or increased dwell time. The patient will complain of pain, fever, chills and may have purulent drainage from the puncture site.
(Bhimji, 2016)

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Test Yourself:

What causes potential bleeding complication in post heart catheterization?

A. Patient exhibits abnormal WBCs
B. **Intensive antiplatelet therapy** - Correct! There are several causes of bleeding complications, but multiple antiplatelet therapy inhibits clotting of the blood for a period of time.
C. Patient's activity level before heart cath

Continuous Telemetry Monitoring

Potential causes of chest pain are stent sensation, coronary artery spasm and distal occlusion from debris. Stent thrombosis can happen within 24 hours up to 30 days after a PCI.

All chest pain/discomfort should be reported to the physician and documented.

Additional assessments post cardiac catheterization include continuous ECG monitoring of the patient.

It is standard practice to obtain a 12-lead ECG post PCI and with any chest pain or discomfort. The physician will need to be notified if there is any change, especially further ST-segment elevation in their ECG from pre-cardiac catheterization ECG.

It is important to continue to monitor cardiac rhythms with telemetry, specifically their ST-segment for elevation post-heart cath. Early intervention in cardiac ischemic decreases mortality and improves patient outcomes.
(Naidu, et al., 2012)

Open Heart Surgery

In some cases, a PCI will be completed and the cardiologist will decide not to place any stents. Instead they will consult a cardiothoracic surgeon about possible open-heart surgery.

Coronary Artery Bypass Graft (CABG)

Coronary artery bypass graft surgery is recommended for high risk patients whose LAD is occluded with or without the patient's dual antiplatelet therapy. Secondly, if there is multi-vessel disease or a failed PCI there is an indication for a CABG. The documentation of antiplatelet medication therapy is important because it will determine when the patient can safely go to surgery.

There are two ways a CABG can be performed: “on pump”, using a cardiopulmonary bypass or “heart-lung machine”, or “off pump”, where the heart continues to beat during the surgery. The surgeon will determine which type of surgery is warranted, based on patient condition and risk factors.

During “on pump” coronary artery bypass surgery, the right coronary artery is mobilized (freed) from its surrounding adipose tissue (yellow). The patient's heart is stopped, the cardiopulmonary bypass

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machine is used for continued oxygenation and blood flow, and the aorta is cross-clamped. For a brief video demonstrating a conventional CABG, go to https://www.youtube.com/watch?v=GvGAgQOhQqY

With an “off pump” coronary artery bypass surgery, the areas of the heart that are the focus of the bypass are stabilized. The remainder of the heart continues beating while the one area is stabilized. For a brief video demonstrating an off-pump CABG, go to https://www.youtube.com/watch?v=klbpfryB08E

Patient Education and Discharge Planning

Patient education is a priority during the rest of the time in the hospital. Patients and families need education and reinforcement of post-PCI orders such as bedrest and limb immobility. It is important to discuss their compliancy with anti-platelet medication and diet modification, as well as cardiac rehab.

During the patient’s hospital admission for UA/NSTEMI or STEMI, they should be given education on smoking cessation, nutritional counseling, pharmacological management, insertion site care and management if PCI, surgical site management if CABG, pain management, and physical activity (including cardiac rehabilitation).

Cardiac rehab should start as soon as possible. Cardiac rehab is a multidisciplinary effort to assist the patient with how to change to a heart healthy diet, appropriate exercise routines and risk factor management.

Discharge instructions should include medication teaching. When discussing antiplatelets you should advise patients to seek immediate medical attention if they have dark or bloody urine, dark or bloody stools, vomiting, or various sizes of bruises on their skin.

Most importantly, patients need to be advised to continue their antiplatelet therapy every day as prescribed. In addition, the patient needs to talk to their cardiologist if they are told or if they need to discontinue their antiplatelet therapy for any reason.
(Davis et al., 2012)

Case Study #1: Cath Lab

A 36-year-old female, Mrs. P., complained of upper back pain and nausea at a physician's clinic. A 12-lead ECG was completed at the clinic and showed ST-segment elevation in leads II, III, and aVF. EMS was called and the paramedics brought her to the hospital as a “code STEMI.”

The patient is a one pack/day smoker, who drinks socially, and the only medication she takes is birth control pills. She weighed 136 kg (300 lbs.) on admission. Labs were drawn and cardiac biomarkers were all elevated with a troponin- I of 8.2. The physician gained informed consent for coronary angiography with possible angioplasty and stent placement. Mrs. P. denied allergies to any medication or food. The patient was sent to the cardiac cath lab. Multiple attempts were attempted on the right and left groin without success. The cardiologist took a right radial approach and was able to

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gain access. The patient had 95% blockage to her right coronary artery (RCA) and it was stented with a drug-eluding stent.

Case Study #1, continued: Decline in Status

Mrs. P. was closely monitored through the night and did not develop any hematomas in the groin. The groin and radial sites were bruised. The patient spent the next day in the hospital. As night shift was coming on duty, the nursing assistant showed the nurse the patient had a blood pressure of 84/50 mmHg, with a pulse of 90 beats/minute. The nurse looked at the previous blood pressures, which showed a trend of 130/80s and the heart rate in the 70s at rest throughout the day.

What do you think is happening to this patient? What should the nurse consider next?

Case Study #1, continued: Nursing Assessment

The nurse assessed Mrs. P. and noted she was dizzy when walking to the bathroom. The nurse assisted her to the bed and checked all the sites again for hematomas. She had the patient turn to the side and the nurse noted there was slight bruising above her hips. In looking at the documentation she only voided 300 mL that day, with her 12-hour input and output being 2200 mL in/300 mL out. The hemoglobin and hematocrit were low normal this am.

What should be the nurse's next step?

The nurse called the physician and informed him of her assessment findings. The physician ordered a CT scan to rule out retroperitoneal hematoma and a complete blood count (CBC).

Case Study #1, continued: Retroperitoneal Hematoma

The CT scan was completed and the radiologist called the physician and confirmed there was a large retroperitoneal hematoma.

Her hemoglobin was 8.5 g/dL, hematocrit was 25.5%, and platelets were 180,000/mL.

The physician orders two units of blood to be given through the night and to recheck the patient's CBC two hours after the last unit of blood is transfused.

Case Study #1, continued: Monitoring, Observation and Outcomes

In the am, Mrs. P's hemoglobin was 11.1 g/dL, and her hematocrit was 33.2%. Her blood pressure was consistently 120/80's and her heart rate was in the 70s. The patient stayed one more night and was released from the hospital the next day without any further complications.

Due to the nurse's astute observation and assessment skills, she was able to intervene quickly when evidence of a thrombosed radial artery presented.

The nurse was able to critically evaluate her observations and conduct nursing assessments which led to a diagnosis of a retroperitoneal hematoma.

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Case Study #2: STEMI

The following STEMI case study will assist you in applying the knowledge gained so far.

The patient is Mr. R., a 60-year-old male who came to the emergency room with sub-sternal chest pain in which he rated a 9/10 and diaphoretic. The nurse immediately took him to a treatment room and informed the physician of his symptoms of sub-sternal chest pain. The nurse anticipates he needs labs and a 12lead ECG. The hospital does provide PCI.

Case Study #2: Collecting Labs

The nurse puts the patient into a gown and she calls the lab to get to room #10 immediately. She also completes a 12-lead ECG within 10 minutes of arrival to the emergency room.

Which of the following labs should be drawn at this time?

- CBC
- CMP
- Coagulations
- Estimated Sedimentation Rate
- Cardiac Enzymes
- Lipid Profile
- Glomerular Filtration Rate

Answer: CBC, CMP, Coagulations, Cardiac Enzymes, Lipid Profile are all appropriate labs to be drawn when a patient has cardiac symptoms.

Case Study #2: Provider Assessment

The provider assesses the patient and ascertains that Mr. R. has a history of type II diabetes and hypertension, smokes ½ pack/day for 42 years, and his father died in his 40s from a “heart attack.”

He is currently on atenolol 25 mg po daily and metformin 500 mg po daily for his hypertension and diabetes respectively. His blood pressure on admission is 160/88 mmHg, heart rate is 90 beats/minute, respirations are 22 breaths/minute and temperature of 98.4. He rates his pain at an 8/10.

He is connected to continuous ECG monitoring and his 12-lead ECG confirms he is in sinus rhythm with ST-segment elevation in leads II, III, and aVf and in V4, V5 and V6 with ST-segment depression in V1, V2 and V3. The provider reviews his labs.

Case Study #2: ECG Interpretation

The ECG shows what kind of MI?

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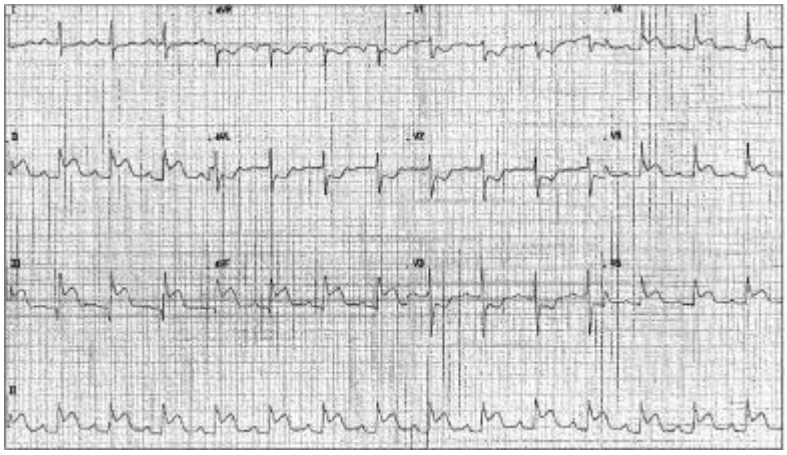

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A. Apical
B. NSTEMI
C. **Inferior -** Correct! Segment Elevation in leads II, III, avF

Test Yourself:

Due to where the ST-segment elevation is located, there is a high probability the affected coronary artery is:

A. LAD
B. **RCA -** Correct! During an occlusion in the RCA, leads II, III, aVF will be elevated.
C. LCX

Interpreting Lab Values

According to the 12-lead ECG and lab results, what kind of sub-condition of ACS is patient X experiencing?

Lab	Value	Normal
CK	517	55-170 ng/ml
CKMB	27.4	0.00 – 2.37 ng/ml
Troponin-I	14.9	0.00 – 0.34 ng/ml
Total Cholesterol	169	Desirable <200 mg/dl
Triglycerides	117	Desirable <150 mg/dl
HDL	46	Desirable >50 mg/dl
LDL	100	Desirable <100 mg/dl

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A. Unstable Angina
B. NSTEMI
C. **STEMI** - Correct! A STEMI is defined as an elevated troponin and ST-segment elevation

Case Study #2: Management Options

Since Mr. R.'s past history included a history of hypertension, familial history of heart attack, diabetes, lipid dysfunction, currently smokes, AND his ECG has ST-segment elevation and troponin-I is abnormally high, the patient is high risk and experiencing a STEMI.

What is the next step in this patient's treatment?

A. Fibrinolytics
B. **PCI** - Correct! PCI is indicated because a STEMI is a MI which goes through all layers of the heart and to decrease the patient's mortality, reperfusion must happen immediately.
C. CABG Surgery

Case Study #2: Antiplatelet Administration

The patient's next step in ACS treatment is to administer antiplatelets. The medication clopidogrel (Plavix™) was administered to the patient as well as a GB IIb/IIIa inhibitor in preparation for the PCI.

Case Study #2: Drug-Eluting Stents (DES)

The cardiologist and the cath lab team was called and the patient entered the cath lab with stable vital signs, but with chest pain 8/10.

The patient gave informed consent to the cardiologist and a time out was performed prior to the procedure.

The cardiac angiography showed >95% occlusion to the proximal and distal RCA. A DES was placed and patient tolerated the procedure without becoming hemodynamically unstable.

The patient's right femoral artery was closed successfully with an artery closure device.

Case Study #2: Post Op Assessments

What post-care assessment would require calling the cardiologist?

A. Patient complaining of dull headache
B. Patient has +2 pedal pulses to the right foot

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C. **Patient has an increased groin bleeding with a hematoma > 5 cm** - Correct! A hematoma greater than 5cm requires further compression and the cardiologist should be notified.

Case Study #2: Monitoring the Patient's Status

Mr. R. returned to the critical care unit with a GB IIIa/IIb infusing. Routine post-cath orders were reviewed and signed by the cardiologist.

Upon the nurse's assessment, the groin was soft and without a hematoma. There was minimal drainage on the dressing. Peripheral pulses were present and right foot is warm, blanchable.

The patient was able to ambulate five hours later.

Case Study #2: Discharge Medications

If not contraindicated, which of the following prescriptions would you anticipate the patient receiving on discharge?

A. **ARB/ACE-I; beta-blockers** - Correct! ARB/ACE-I; beta-blockers – This is what the CMS/TJC says the patient should be prescribed after a MI.
B. Nitroglycerin; beta-blockers
C. Aspirin; beta-blockers

Case Study #2: Discharge Instructions

The nurse was able to spend time with the patient and spouse reviewing the new medications he would be taking. The nurse also stressed the importance of NOT stopping the antiplatelet regime without discussing it with the cardiologist.

Mr. R. was given information about when he will start cardiac rehab as well as recommended diet modifications.

Mr. R. did mention he was overwhelmed with all the new information, but was very thankful the nurse took the time to go over all the new information.

Conclusion

Time is muscle and the immediate attention to a patient with ACS symptoms is required. A careful physical assessment, labs and 12-lead ECGs as well as risk stratification will assist in determining which ACS sub-condition that patient is suffering from. After the patient is diagnosed, then appropriate medical and/or interventional treatment is initiated. Successful patient outcomes are contingent on the specific interventions as well as the education the healthcare team provides the patient. As healthcare continues to learn more about ACS, our treatments will continue to evolve.

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