Emergency Preparedness in the OR

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Acute Coronary Syndrome—Part III

Darlene Hutton, BScN, MSN, RN

Acute coronary syndrome (ACS) is a diagnosis made on the basis of two criteria: patient presentation and electrocardiographic (ECG) changes. Prompt recognition and treatment of the patient with this diagnosis is essential to reduce the risk of associated complications. This article will address the pathophysiology, signs and symptoms, ECG changes, and treatment of an ACS including the unique situation of the peri- and postoperative patient experiencing this potentially fatal condition.

CASE STUDY 1

You are caring for a 52-year-old female postoperative patient who is recovering from breast reduction surgery. She suddenly starts complaining of difficulty breathing and chest heaviness. You assess her operative site to be clear and intact. Her vital signs are blood pressure 86/90 mmHg, heart rate 40/min, and respirations 20/min, oxygen saturation is 92%, and chest is clear. You promptly start oxygen 4 L by nasal prongs, ensure that her intravenous is intact, call for help, and obtain a 12-lead ECG. You assess the ECG and note that the patient is in a sinus bradycardia, normal PR, QRS, and QT intervals. Upon assessing each lead for ST-segment deviation, you note that the ECG has ST-segment elevation in leads II, III, aVF, and V_1, with ST-segment depression in the majority of the remaining leads (Figure 1). You notify the physician stat of the patient's symptoms and the ST-segment changes on the ECG. A diagnosis of an acute inferior wall myocardial infarction (MI) is made and the patient is treated accordingly. Treatment will be discussed in detail later in the article.

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CASE STUDY 2

During surgery for an inguinal hernia repair, a 60-year-old male patient suddenly develops ventricular fibrillation on the monitor. The patient is pulseless. Compressions are started at 100/min while the defibrillator is obtained. The patient is defibrillated using a biphasic defibrillator twice over a 3-min time frame using 200 J and returns to sinus rhythm after the second shock. The surgery is halted while investigation into the cause of this cardiac arrest is explored. An ECG is performed, which demonstrates ST-segment elevation in leads V_1–V_6, I, and aVL (Figure 2). A diagnosis of an acute anterior wall MI is made. The patient is transferred to the intensive care unit where management of the MI takes place. Treatment will be discussed in detail later in the article.

Acute Coronary Syndrome—Pathophysiology and Definition

An ACS is a condition in which one of the coronary arteries has either completely occluded or almost occluded with a clot. The occlusion of the artery results in no blood flow or a significantly reduced blood flow to the part of the heart that the artery supplies. When blood flow is reduced to the cardiac muscle, the cardiac muscle will become ischemic and often necrotic within 4–6 hr unless the process can be halted. Synonymous terms used for ACS are acute MI, unstable angina, cardiac ischemia, ST-segment elevation MI, and non-ST-segment elevation MI (Anderson et al., 2007). The definitive diagnosis of an MI is made once the cardiac blood markers become elevated, which may take up to 6 hr after the onset of coronary artery occlusion (Alpert, Thygesen, Antman,

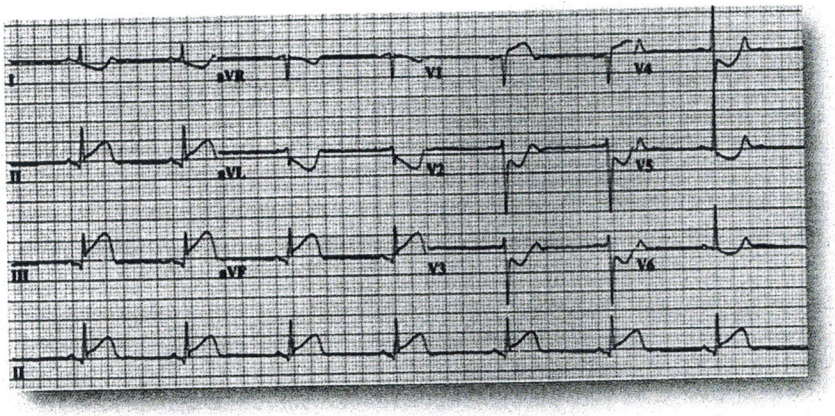


Figure 1. ST-segment elevation in leads II, III, aVF, and V_1. ST-segment depression in leads I, aVL, and V_2–V_6.

& Bassand, 2000). Treatment of an ACS is based on the ECG findings and the patient presentation, and not dependent on the results of the cardiac markers. The cardiac markers used to identify whether MI has occurred are troponin or creatinine kinase MB (Anderson et al., 2007; Schreiber & Miller, 2011). An ACS is diagnosed with the presence of symptoms and changes on the ECG in the form of ST-segment elevation, depression, or T-wave inversion (Anderson et al., 2007).

Acute Coronary Syndrome–Patient Presentation

When blood flow is diminished to the cardiac muscle, the patient may experience a wide variety of symptoms, including pain, ache, heaviness, pressure, indigestion, and a tingling sensation. These symptoms may occur anywhere from the abdominal area to the jaw, down either arm, or in the back (Zafari et al., 2011). Patients who are diabetic, female, or elderly will often present with an ACS experiencing symptoms other than chest pain (Achar, Kundu, & Norcross, 2005). These patients may make the diagnosis of an ACS difficult, as the symptoms may be atypical. Another group of patients who may pose a challenge in quickly diagnosing ACS is the perioperative or postoperative patients. Patients in this setting represent a unique population when they are having an ACS for two reasons. The first reason relates to the paralytic and sedation medication used during and after surgery that may render the patient unable to verbalize any symptoms. Patients who are unable to verbalize their symptoms will often demonstrate signs of an ACS in other ways: decreasing oxygen saturation, a rapid change in their blood pressure or heart rate, and the development of pulmonary edema. They may also have cardiac rhythm changes ranging from ventricular irritability in the form of premature ventricular contractions or ventricular fibrillation to atrioventricular blocks. Any of these changes in a patient unable to verbalize symptoms should have an ECG done immediately. The second reason why the peri- and postoperative patients are unique is that they often meet exclusion criteria for the treatment of an ACS. Treatment will be discussed later in the article.

ECG REVIEW

In the two case studies presented, the diagnosis of an ACS was based on patient presentation and changes on the ECG consisting of ST-segment elevation, depression, or T-wave inversion (Table 1). When assessing an ECG, it is important to use a systematic approach. The approach used by this author is RIRI: rate, interval, rhythm, and infarct/ischemia (Hutton, 2005). For the purposes of this article, the only part of the ECG assessment that will be covered is the infarct/ischemia portion that involves looking at every single lead of the ECG and identifying whether there is any ST-segment or T-wave changes. When a patient is experiencing an ACS, the ST segment will deviate from baseline and/or the T wave may become flattened or inverted. The ST segment is the line immediately after the completion of the QRS complex to the T wave (Figure 3). In a normal ECG, every single lead of the 12-lead ECG should have ST segments at baseline and T waves upright. The 12-lead ECG takes a picture of three segments of the left ventricle: the anterior, inferior, and lateral wall.

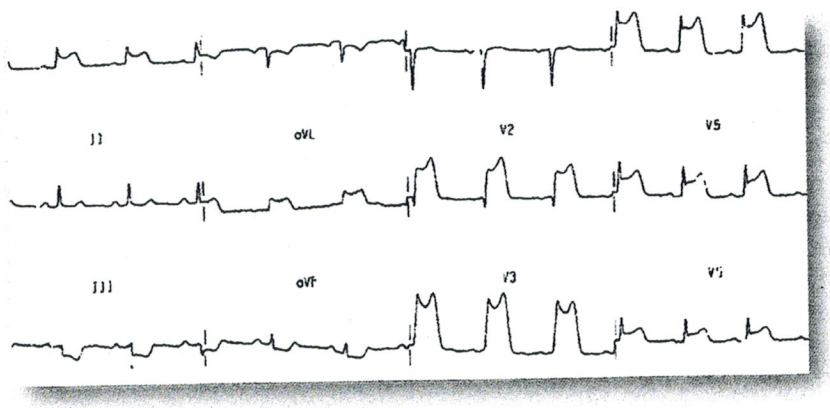
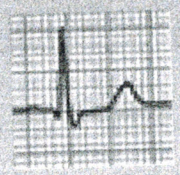
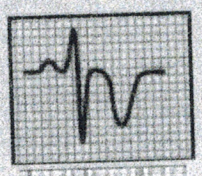
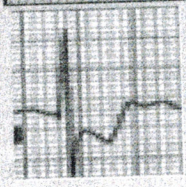
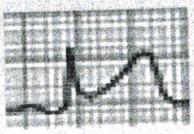
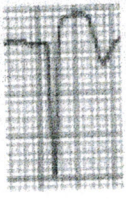


Figure 2. Acute anterior lateral wall MI. Note significant ST-segment elevation in V_2–V_4 (anterior) and leads I, aVL, V_5, and V_6 (lateral).

TABLE 1 ECG Changes Associated With Acute Coronary Syndrome

ECG Changes	
	*Normal ST segment and T wave*: The ST segment starts at the end of the QRS complex and ends at the start of the T wave. In the normal ECG, the ST segment should be at baseline and the T wave should be upright in all leads of the ECG (aVR is the exception).
	*T-wave inversion*: A patient presenting with symptoms plus T-wave inversion is having an ACS. Standard therapy for ACS needs to be initiated.
	*ST depression*: A patient presenting with symptoms plus ST-segment depression is having an ACS. If the cardiac markers become elevated, the discharge diagnosis is a non-ST-elevation MI.
	*ST elevation*: An acute ST-elevation MI usually occurs in the setting of a coronary artery that has become completely occluded because of a clot. Less frequently, ST elevation can occur in the setting of a coronary artery spasm.
	*Q wave*: A normal ECG should have no Q waves. A Q wave is noted when the first direction the QRS complex takes is a negative one. A Q wave is usually indicative of an ST-elevation MI where full myocardial wall necrosis has occurred. In the setting of an acute ST-elevation MI, Q waves will develop within the first 6 hr of symptom onset.

The three inferior leads are II, III, and aVF. The four anterior leads are V_1–V_4. The four lateral leads are I, aVL, V_5, and V_6 (Figure 4). To diagnose ACS changes on the ECG, there needs to be at least two adjacent leads within a particular grouping to have ST-segment or T-wave inversion.

In Case Study 1 (Figure 1), there is ST-segment elevation in leads II, III, aVF, and V_1 with ST-segment depression in the remainder of the leads. When looking at an ECG with a combination of ST elevation and depression, the infarction is occurring in the segment of the heart where the ST elevation is; in this case, the inferior wall of the left ventricle. ST-segment elevation indicates an acute MI. The leads with the ST depression, while not infarcting, are affected by the diminished blood supply.

In Case Study 2 (Figure 2), there is ST-segment elevation in three of the four anterior leads (V_2, V_3, and V_4), and the entire lateral leads (I, aVL, V_5, and V_6). There is ST-segment depression in leads III and aVF. This ECG demonstrates an acute anterior lateral wall MI. One of the most frequent complications to anticipate with an anterior wall MI is acute pulmonary edema, or if a low blood pressure exists with pulmonary edema and cardiogenic shock.

Complications with all MIs in the acute phase and for up to 48 hr afterward may include pulmonary edema, cardiogenic shock, bradycardia, complete atrioventricular block, ventricular rupture, papillary muscle rupture, and cardiac arrest usually in the form of ventricular fibrillation. Before the current guidelines for management of the patient with an acute MI, these complications occurred much more frequently than they do today.

TREATMENT

According to the American College of Cardiology and the American Heart Association (2007), the guidelines for the management of ACS stress prompt identification and rapid treatment as being most essential to reduce the potential risks and to, ideally, prevent tissue death from occurring. A very important agent used to rapidly start the process of dissolving the platelets found within the clot that cause the symptomatic narrowing is antiplatelet therapy: aspirin, nonenteric coated, and chewable; and plavix. Another important agent, if no contraindications exist, is nitroglycerin. Patients who should not receive nitroglycerin are those who are already hypotensive, who are having a

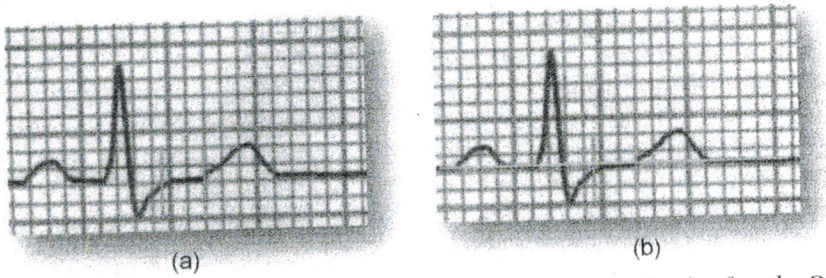


Figure 3. (a) The ST segment is the horizontal portion immediately after the QRS complex and ending at the start of the T wave. In every single lead of the ECG, the ST segment should be at baseline, which is defined as the isoelectric line (b).

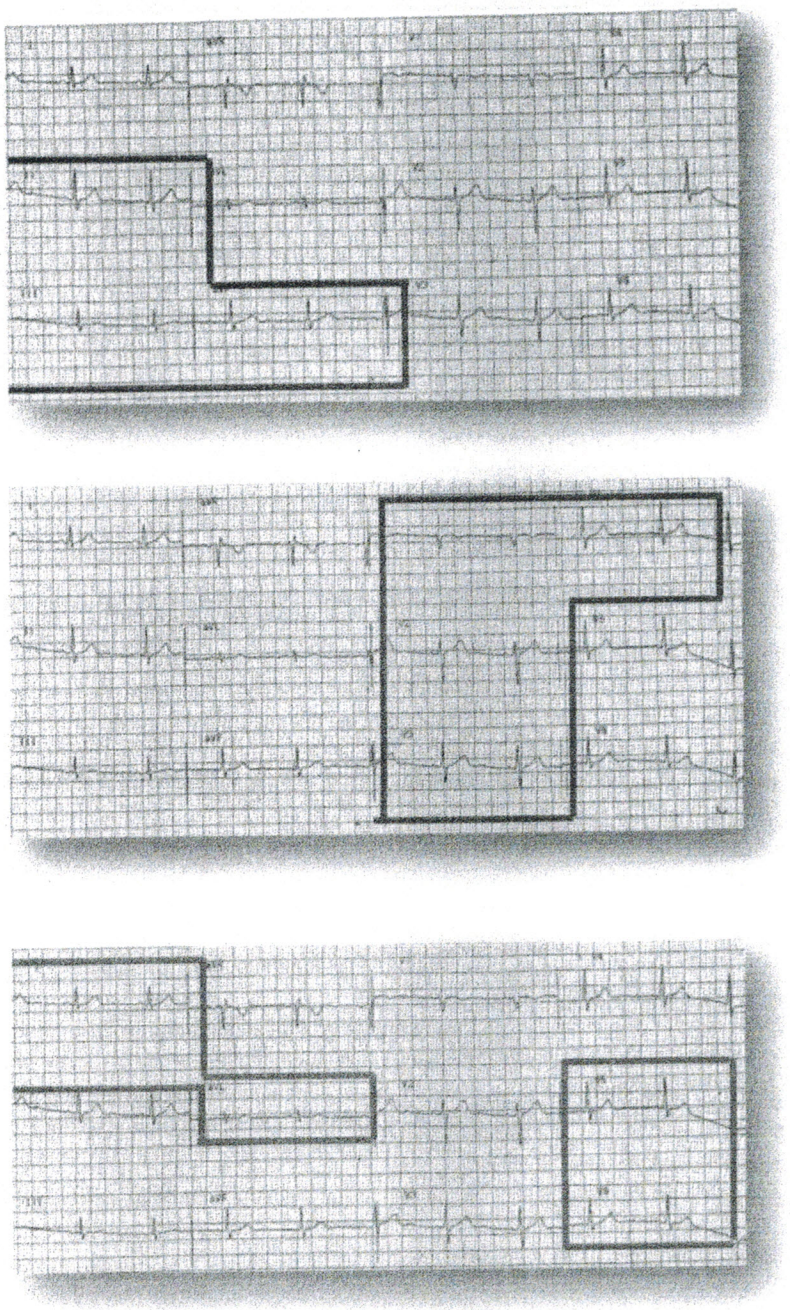


Figure 4. Top ECG: Inferior leads II, III, and aVF. Middle ECG: Anterior leads V_1–V_4. Bottom ECG: Lateral leads I, aVL, V_5, and V_6

right ventricular wall MI, or who have recently taken a drug for erectile dysfunction. Oxygen is to be administered as part of standard therapy; however, the recently published 2010 Advanced Cardiac Life Support guidelines recommend that oxygen is only to be started on patients with an ACS if they have an oxygen saturation level below 94%, they are in pulmonary edema, or they have dyspnea (American Heart Association, 2010). With ST-segment elevation, the coronary artery is completely occluded with a fibrin and platelet-rich clot. In addition to the previous treatments listed, the patient with ST-segment elevation also requires some form of reperfusion therapy. There are two treatment strategies to open up the occluded artery, also known as reperfusing the artery: fibrinolytic therapy and primary angioplasty. In the peri- and postoperative patient, administration of a fibrinolytic is contraindicated because of the high risk of bleeding associated with this medication in the surgical patient. Therefore, prompt transfer to a cardiac center with catheterization capability is ideal. Another medication routinely administered to patients having an ACS is an anticoagulant such as low-molecular-weight heparin (enoxaparin), intravenous heparin, or fondaparinux. These agents may also be contraindicated in the surgical patient because of the higher risk of bleeding.

CONCLUSION

The diagnosis of an ACS is based on the clinical presentation of the patient along with changes on the ECG in the form of ST-segment deviation from baseline or T-wave inversion. Complications associated with an ACS in the acute phase and for the first few days include cardiac arrest, muscle rupture, cardiac dysrhythmias, and pump failure such as pulmonary edema or cardiogenic shock. Prompt recognition of ACS in the surgical patient may pose a challenge because of the patient's inability to verbalize or because of their reduced pain sensation because of the medications used. For these patients, the nurse needs to be aware of the following in assessing for an ACS: reduced oxygen saturation; changes in the patient's blood pressure and heart rate; ventricular irritability such as frequent premature ventricular contractions or ventricular fibrillation; or bradycardic rhythms such as sinus bradycardia or complete atrioventricular block. Recognizing these changes should prompt the nurse to perform an ECG immediately. Once ACS is diagnosed, the surgical patient should have oxygen administered in most

of the cases, chewable aspirin given, and nitroglycerin administered, and the patient should be transferred to a center where cardiac catheterization and primary angioplasty can be performed.

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