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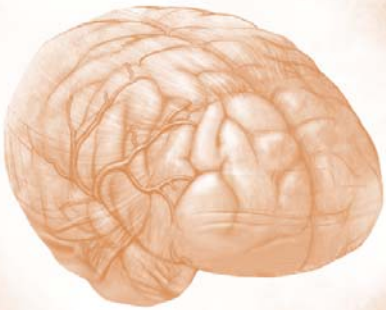
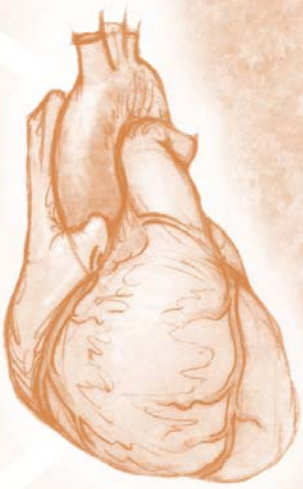
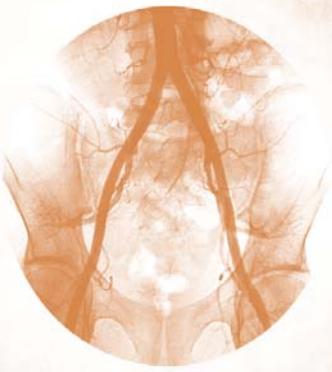
Future Prospects in the Management of Acute Coronary Syndrome and Thrombosis

2006 Update Newsletter







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TARGET AUDIENCE

This activity has been designed to meet the educational needs of registered nurses involved in the management of patients with acute coronary syndrome.

PURPOSE

To provide the latest information on the pathophysiology of acute coronary syndrome and thrombosis and results of recent research on the use of antithrombotic medications in the management of patients with, or at risk for, acute coronary syndrome.

STATEMENT OF NEED/PROGRAM OVERVIEW

Coronary heart disease is the leading cause of death in the United States, accounting for more than 500,000 deaths annually. Acute coronary syndrome (ACS) is a constellation of clinical symptoms related to acute myocardial injury. ACS represents a spectrum of life-threatening disorders that are a major cause of emergency medical care and hospitalizations in the US. Annually, an estimated 2 million Americans are admitted to the hospital with acute myocardial infarction, and more than 250,000 others die before reaching a hospital, within 1 hour of the onset of symptoms.

Despite the use of aspirin, heparin, and anti-ischemic therapy, current research has shown that the prognosis of patients presenting with ACS remains poor. Within 4 to 6 weeks of presentation, the risk of death or myocardial infarction is 8% to 14%. The rate of death, MI, or refractory ischemia is even more excessive (approximately 15% to 25%).

Advances in the understanding of the pathophysiology of ACS and thrombosis have led to the development of novel therapeutic agents and treatment strategies that hold promise for better clinical outcomes. Trials investigating the use of antithrombotics in patients with ACS are currently comparing the efficacy of new and established agents in preventing death, myocardial infarction, and refractory ischemia.

The educational goal of this program is to increase awareness of the results of recent research on the use of antithrombotic medications in the management of patients with, or at risk for, ACS.

EDUCATIONAL OBJECTIVES

After completing this series, the participant should be better able to:

- Discuss acute coronary syndrome and its classifications
- List the cornerstones of current treatment and the recommended usage of antithrombotic therapies for acute coronary syndrome and other thrombotic events
- Implement the treatment recommendations from the ACC/AHA guidelines for acute coronary syndrome
- Discuss ongoing studies, late-breaking trial results, and their relevance to clinical practice

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A statement of credit will be issued only upon receipt of a completed activity evaluation form and a completed posttest with a score of 70% or better. Your statement of credit will be mailed to you within 3 weeks.

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ISSUE 1 — Acute Coronary Syndrome: Introduction and Overview of Classifications

LEARNING OBJECTIVES

After completing this activity, the participant should be better able to:

- Understand the pathophysiology of acute coronary syndrome (ACS), particularly the progression of coronary atherosclerosis and the mediators of plaque rupture, thrombosis, and myocardial ischemia
- Describe the incidence and prevalence of different types of ACS, particularly ST elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina (UA)
- Identify the clinical features that distinguish STEMI and NSTEMI
- Recognize the distinctive clinical presentations and pathophysiologic causes of UA
- Describe the roles of various cardiac biomarkers in the diagnosis and risk-stratification of patients with ACS
- Describe the role of electrocardiographic and other diagnostic imaging in the evaluation of patients with suspected ACS

CASE STUDY: Unstable Angina/ Non-ST-segment Elevation Myocardial Infarction

A 58-year-old white man with a history significant for diabetes and hypertension presents to the emergency room complaining of chest and epigastric pain that began after dinner and has lasted more than 30 minutes. He states that after his morning walks, he has had similar episodes of pain, which are relieved by rest. He denies a history of myocardial infarction.

VITAL SIGNS

- *Temperature: 37.1°C (98.8°F)*
- *Blood pressure: 140/90 mm Hg*
- *Pulse: 98/min*

Physical examination shows no jugular venous distension, a clear chest bilaterally, normal heart sounds on auscultation, a benign abdomen, and no peripheral edema.

INITIAL DIAGNOSTIC WORK-UP

- *Electrocardiogram: 1 mm ST-segment depression in leads II, III, and VF*
- *Chest x-ray: Normal*
- *Troponin: Within normal limits*

INTRODUCTION

Acute coronary syndrome (ACS) is a term used to describe a group of conditions, ranging from unstable angina to acute myocardial infarction, in patients with ischemic heart disease.[1] Decisions regarding medical and interventional treatments are based on the presence or absence of ST segment elevation on the presenting electrocardiogram and abnormal elevations of myocardial enzymes, such as troponins. Based on these diagnostic criteria, a patient is clinically classified into one of 3 categories:[1]

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

Therefore, this newsletter series begins with the epidemiology, pathophysiology, initial presentation, and electrocardiographic (ECG) and laboratory findings associated with ACS. Further issues will include case studies and in-depth discussions regarding conservative and aggressive management of ACS and thrombosis, including pulmonary embolism (PE) and deep vein thrombosis (DVT).

UA/NSTEMI

Epidemiology

ACS represents a significant public heath burden. According to the American Heart Association, preliminary estimates indicate that for 2003, approximately 1.6 million patients were hospitalized for ACS, including 946,000 patients diagnosed with MI and 650,000 with UA. Approximately 28,000 of these patients were discharged with both diagnoses. UA/NSTEMI is the more common diagnosis, with only 30% to 45% of MI patients diagnosed with acute STEMI.[2]

Typically, UA presents as chest pain or equivalent ischemic discomfort with at least one of 3 features:[3]

- Pain occurs at rest (or with minimal exertion), usually lasting >10 minutes
- Pain is severe and of new onset (ie, within the prior 4 to 6 weeks)
- Pain occurs with a crescendo pattern (ie, distinctly more severe, prolonged, or frequent than previously)

UA and NSTEMI often present in a similar manner, and the distinction is often made hours or days later, when the results of diagnostic tests become available.[5] A diagnosis of NSTEMI is not established until elevated cardiac biomarkers—evidence of myocardial necrosis—are detected in a patient with the clinical features of UA.[3]

Pathophysiology

UA/NSTEMI is triggered by a reduction in oxygen supply and/or an increase in myocardial oxygen demand, often in the presence of a coronary obstruction.[3] Researchers believe that there are major pathophysiologic processes that may contribute to the development of UA, more than one of which may affect many patients.

Angiographic studies of patients with UA/NSTEMI have shown that 40% have single-vessel disease, 30% have two-vessel disease, 15% have three-vessel disease, 10% have no critical coronary stenosis, and 5% have left main stenosis. On angiography, the "culprit lesion" has the distinct features of eccentric stenosis with scalloped or overhanging edges and a narrow neck. In contrast to the "red" thrombi often seen in patients with acute STEMI, patients with UA/NSTEMI are more likely to have "white" (platelet-rich) thrombi.[3]

TABLE 1

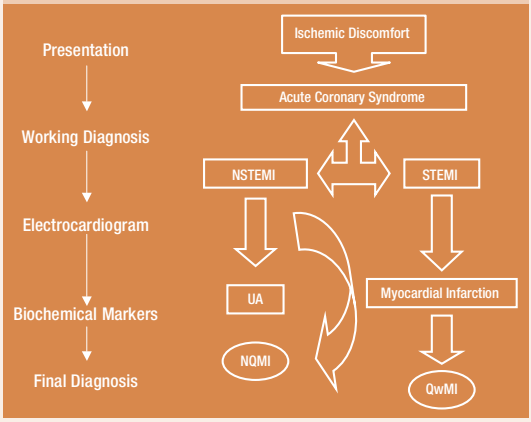
Causes of Unstable Angina[3,4]

- Plaque rupture or erosion with superimposed nonocclusive thrombus (probably the most common cause)
- Dynamic obstruction (eg, coronary spasm or vasoconstriction)
- Progressive mechanical obstruction (eg, rapidly advancing atherosclerosis or restenosis following percutaneous coronary intervention [PCI])
- Secondary UA related to increased oxygen demand or decreased oxygen supply (eg, tachycardia, hypotension, or anemia)
- Arterial narrowing caused by inflammation and/or infection

Initial Clinical Evaluation

Disruption of a vulnerable or high-risk plaque leads to a reduction in blood flow through the affected epicardial coronary artery and subsequent ischemic discomfort, the clinical hallmark of ACS. For patients with ischemic discomfort and suspected ACS, the initial clinical evaluation is heavily dependent upon ECG monitoring

FIGURE 1
Evaluation of Patients with ACS



and testing for cardiac biomarkers (Figure 1).[4]

Ischemia may be caused by an incompletely or completely occlusive thrombus. Among STEMI patients, the majority ultimately develop Q-wave MI (QwMI), whereas a minority develop non–Q-wave MI (NQMI). The reverse is true in NSTEMI patients, with the majority developing NQMI, and a minority developing QwMI.[6]

Early diagnosis and aggressive clinical management are critical for achieving optimal outcomes in patients with ACS. Such patients should be evaluated with continuous ECG monitoring in an environment with defibrillation capability.[4] In addition, a definitive interpretation of a 12-lead ECG should be obtained within 10 minutes. The most crucial priority of early evaluation is to identify patients with STEMI who should be considered for immediate reperfusion therapy, and to recognize potentially life-threatening complications, such as aortic dissection.[4] Additional preliminary assessment goals are to:

- Recognize or exclude MI using cardiac markers[3]
- Evaluate for significant coronary artery disease using standard treadmill ECG stress testing in most patients
  - Perfusion or echocardiographic imaging in patients with fixed ECG abnormalities
  - Pharmacologic stress testing in patients with mobility restrictions[3]

Electrocardiography The electrocardiogram remains a crucial diagnostic tool in the management of patients with ACS. A detailed analysis of patterns of ST-segment elevation may inform the decision to use reperfusion therapy and perhaps the type of reperfusion therapy. For example, identifying infarcted arteries allows one to predict the amount of myocardium at risk and assess the urgency of revascularization. The electrocardiogram can also show signs of reperfusion, providing information about microvascular blood flow and subsequent prognosis. Analysis of the electrocardiogram can also reveal new conduction abnormalities and arrhythmias—additional important variables in prognosis and management strategies.[7]

Depending on the severity of ischemia, approximately 30% to 50% of patients with UA/NSTEMI can experience ST-segment depression, transient ST-segment elevation, and/or T-wave inversion.[3] ST-segment or T-wave changes are typically persistent in patients with NSTEMI, whereas if they occur in patients with UA, they are usually transient.[4]

Even minor ST-segment deviation—as low as 0.05 mV—can predict a poor outcome in patients with UA. T-wave changes are relatively sensitive, but less specific for ischemia, with the exception of new, deep, T-wave inversions (≥0.3 mV).[3]

Large variations in coronary anatomy may limit the specificity of electrocardiographic findings, particularly in patients with a history of coronary artery disease, MI, collateral circulation, or coronary-artery bypass surgery. However, electrocardiogram analysis is the best means of identifying proximal occlusion of the coronary arteries—the source of the most extensive and severe MIs—and for determining optimal treatment strategies for patients with ACS.[7]

Cardiac Biomarkers UA and NSTEMI are closely related conditions with very similar pathogenesis and clinical presentation. However, these conditions differ in severity, particularly in whether the degree of ischemia is severe enough to cause myocardial injury and the subsequent release of cardiac enzymes. Therefore, measurements of the markers of myocardial injury, particularly creatine kinase (CK), its MB isoenzyme (CK-MB), troponin I (TnI), and troponin T (TnT), are critically important tools for distinguishing between UA and NSTEMI. In patients with ACS, a diagnosis of UA is established if no cardiac biomarkers are detected, whereas a diagnosis of NSTEMI is established if markers of myocardial necrosis have been released.[4]

Elevated cardiac biomarkers are associated with an increased risk of death or recurrent MI (Table 2).[3] Among the cardiac biomarkers, troponins are a particularly specific marker of myocardial necrosis and provide the greatest diagnostic value in patients with normal CK-MB levels.[3,5] In these patients, elevated troponin T levels are associated with a nearly 4-fold risk of coronary events within the following 6 months.[5]

Diagnostic Imaging As an adjunct to exercise or pharmacologic stress testing, nuclear imaging improves both sensitivity and specificity, providing important information on patient risk. For example, during a stress test, altered thallium uptake—measured as an increased ratio of the amount of thallium in the lungs to that in the heart—is suggestive of stress-induced left ventricular (LV) dysfunction. Compared to patients with normal lung reuptake, patients with abnormal thallium uptake have lower LV function, reduced exercise capacity, and a higher prevalence of exercise-induced angina. Moreover, despite a higher revascularization rate, patients with abnormal lung reuptake have a higher rate of subsequent cardiac events.[5]

Dipyridamole or adenosine sestamibi tomography, another form of diagnostic imaging, can distinguish between high-risk and low-risk patients with UA whose clinical findings alone suggest intermediate risk. In such patients, normal dipyridamole sestamibi scanning results are associated with a lower rate of subsequent cardiac events at 2-year follow-up, whereas the presence of either a reversible or fixed perfusion defect suggested a markedly higher risk of future events.[5]

In addition to providing prognostic information, a major benefit of diagnostic imaging with sestamibi or thallium is reducing unnecessary hospitalizations by demonstrating normal myocardial perfusion and excluding acute ischemia.[3]

STEMI

Prevalence and Epidemiology

STEMI occurs in 30% to 45% of MI patients, suggesting a conservative estimate of at least 500,000 STEMI events per year in the US.[1,8] Over the past several decades, the mortality rate associated with STEMI has progressively declined, caused in part by an associated fall in the overall incidence of MI and an increased post-MI survival rate. Moreover, there has been a steady decrease in the proportion of ACS patients presenting with STEMI versus NSTEMI.[8]

Pathophysiology

STEMI can be triggered by an abrupt decrease in

coronary blood flow following the thrombotic occlusion of an atherosclerotic coronary artery. STEMI typically occurs when a thrombus develops quickly at a site of vascular injury, such as those secondary to lipid accumulation, hypertension, or cigarette smoking. Infarction is most likely to occur when an atherosclerotic plaque ruptures, fissures, or ulcerates in a pro-thrombogenic environment, which is ideal for the development of a coronary artery occlusion.[6]

Coronary plaques with a rich lipid core and thin fibrous cap are prone to rupture. Several agonists, including collagen, epinephrine, and serotonin, promote platelet activation at the site of the ruptured plaque.[6] Agonist stimulation of the platelets leads to a series of deleterious events, including the release of thromboxane A2, a potent local vasoconstrictor, additional platelet activation, and a conformational change in the glycoprotein IIb/IIIa receptor.

Converted to its functional state, the glycoprotein IIb/IIIa receptor develops a high affinity for soluble adhesive proteins, attracts multivalent molecules, and triggers platelet cross-linking and aggregation.[6] Following repeated activation of the coagulation cascade, platelet aggregates and fibrin strands conspire with the thrombus to occlude the culprit coronary artery. Indeed, coronary thrombus occlusion of the infarcted artery is a signature finding among patients with STEMI. Angiography reveals coronary thrombus formation in more than 90% of patients with STEMI, in 35% to 75% of patients with UA or NSTEMI, and in 1% of patients with stable angina.

Initial Clinical Evaluation

The initial clinical evaluation of patients with suspected STEMI is similar to that in patients with NSTEMI/UA (Figure 1). When evaluating patients with suspected or confirmed STEMI, it is important to consider the temporal aspects of the condition. Myocardial infarction typically progresses through several distinct stages:

ACUTE: Encompassing the first few hours to 7 days following infarct onset

HEALING: Occurring during days 7 to 28

HEALED: Beginning on day 29 and continuing thereafter[6]

Electrocardiography Electrocardiographic findings are critical for distinguishing Q-wave from non–Q-wave MI in patients with STEMI. In patients with suspected STEMI, total occlusion of an epicardial artery produces ST-segment elevation in the initial stage of the acute phase of MI. In the majority of patients presenting with ST-segment elevation, the development of Q waves on the ECG leads to a diagnosis of sustained Q-wave MI. Only a small proportion of patients in this population maintain a non–Q-wave MI.[6]

The Role of Cardiac Biomarkers Following a STEMI event, the necrotic heart muscle releases large quantities of cardiac biomarkers into the blood. The rate of release of specific proteins is directed by several factors, including their intracellular location and molecular weight, and the local blood and lymphatic flow. Therefore, the pattern of protein release can reveal important diagnostic information for patients following an episode of STEMI. Given the urgency of reperfusion strategies, treatment decisions must be made before results of serum cardiac biomarker assays are available from a central laboratory. However, with the advent of whole-blood bedside assays, patterns of cardiac biomarker release are becoming a prominent component of treatment decisions, especially in patients with nondiagnostic ECGs.[6]

One of the major cardiac biomarkers in patients with ACS is CK. Following an episode of STEMI, CK levels increase within 4 to 8 hours and return to baseline by 48 to 72 hours. However, total CK measurements have a low specificity for STEMI. Elevated CK levels may be due to multiple causes:[6]

- Skeletal muscular diseases, including muscular dystrophy and myopathies
- Electrical cardioversion
- Hypothyroidism
- Stroke
- Surgery
- Skeletal muscle damage secondary to trauma, convulsions, and prolonged immobilization

Given the range of other possible causes of increased CK levels, relying solely on CK measurements may lead to an erroneous diagnosis of STEMI.[6] Unlike total CK, the MB-CK isozyme is generally not present in extracardiac tissue, a characteristic that gives it considerably higher specificity for cardiac events. Still, myocardial disruption due to cardiac surgery, myocarditis, or electrical cardio-version can increase serum MB-CK levels and potentially lead to a false STEMI diagnosis. Some clinicians use a CK-MB mass:CK activity ratio to clarify the diagnosis in patients with elevated CK-MB. For example, a ratio ≥2.5 suggests a myocardial infarction rather than a skeletal muscle source of CK-MB elevation). This ratio is less useful when levels of total CK are high because of skeletal muscle injury or when the total CK level is within the normal range but CK-MB is elevated.

TABLE 2

Cardiac Biomarkers and Short-term Risk of Death or Nonfatal MI in Patients With Unstable Angina[4]

RISK OF DEATH	CARDIAC BIOMARKER LEVEL
HIGH	Elevated (eg, TnT or TnI >1.0 ng/mL)
INTERMEDIATE	Slightly elevated (eg, TnT >0.01 ng/mL but <1.0 ng/mL)
LOW	Normal (eg, TnT <0.01 ng/mL)

Cardiac-specific troponin T (cTnT) and troponin I (cTnI) differ from the skeletal muscle forms of troponin proteins by several amino acid sequences, a feature that allowed for the development of a quantitative assay with highly specific monoclonal antibodies. In the blood of a healthy individual, cTnT and cTnI are normally undetectable. Following an episode of STEMI, cTnT and cTnI levels increase more than 20-fold and remain elevated for 7 to 10 days, imparting considerable diagnostic utility. Given their high cardiac specificity, cTnT and cTnI are now the preferred biochemical markers for MI.[6] Although many hospitals are using cTnT or cTnI rather than CK-MB as the routine serum cardiac marker, any of these are useful for diagnosing STEMI.

Myoglobin, another biomarker released into the blood within a few hours of STEMI, is not very useful for an unequivocal diagnosis of the condition. In addition to myoglobin's lack of cardiac specificity, it is rapidly excreted in the urine, returning to normal levels within 24 hours of the onset of STEMI.[6]

To confirm the diagnosis of MI, serum cardiac markers should be measured upon hospital admission, 6 to 9 hours after admission, and, if the diagnosis remains uncertain, 12 to 24 hours after admission.[6] Although it is not cost-effective to measure several biomarkers at all time points, there are some situations when this becomes necessary. For example, in patients with cardiac-specific troponin levels that have been elevated for longer than 1 week, it may be useful to measure

CASE STUDY: ST-segment Elevation Myocardial Infarction

A 62-year-old African-American man with no significant medical history presents to the emergency room with severe, sharp, non-radiating substernal chest pain that began while rearranging furniture with his wife. He denies any prior episodes of pain. Vital signs are within normal limits. Physical exam is completely benign.

INITIAL DIAGNOSTIC WORK-UP:

- *ECG: Sinus tachycardia with 2 mm of ST elevation in leads V2-V4*
- *Troponin level: Within normal limits*
- *Chest x-ray: Normal*
- *Arterial blood gases: 7.47/32/96/99% saturation on room air*

biomarkers that typically stay in the blood briefly—such as CK-MB or myoglobin—to evaluate the presence of recurrent MI.

Diagnostic Imaging Given its safety and ease of use, echocardiography represents a useful screening tool for patients with suspected STEMI, despite its limitations in distinguishing old myocardial scars from acute severe ischemia. In patients with STEMI, two-dimensional echocardiography nearly always reveals abnormalities of wall motion. Echocardiography findings, particularly the early detection of wall abnormalities, are used to develop management strategies. Other echocardiography findings, such as the estimation of LV function, can help determine patient prognosis. Doppler echocardiography can be used to identify serious complications of STEMI, including ventricular septal defect and mitral regurgitation.[6]

Although employed less often than echocardiography, several radionuclide imaging techniques are available to evaluate patients with suspected STEMI. For example, myocardial perfusion imaging with ^{201}TI or ^{99m}Tc-sestamibi—two radionuclides that are distributed by myocardial blood flow and concentrated by viable myocardium—can reveal defects during the first few hours after developing transmural infarction. Despite its high sensitivity, perfusion scanning is unable to distinguish acute infarcts from chronic scars, and therefore it is not specific for acute MI.[6] Using ^{99m}Tc-labeled red blood cells, radionuclide ventriculography is another radionuclide technique that can reveal wall motion disorders and reduced ventricular ejection fraction in patients with STEMI. Although this assay is useful in diagnosing RV infarction, it is also nonspecific for MI.

SUMMARY

The evaluation and management of acute coronary syndrome are undergoing rapid development. A key factor in ACS management will be the refinement of strategies to define and distinguish an intermediate-risk category of patients who need urgent admission and treatment, as opposed to those lower-risk patients who have not experienced acute ischemia and can be expeditiously discharged to outpatient follow-up. The methodology should include clinical evaluation tools that incorporate electrocardiographic data, cardiac biomarkers, and complementary use of imaging technology.

Controversy surrounds temporal intervention in the management of ACS. The next two issues of the newsletter series will address both conservative and early intervention, as well as long-term outcomes. ■

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POSTTEST

1. Which of the following parameters is used to classify patients with MI?
   A. Presence or absence of ST segment elevation on the presenting electrocardiogram
   B. Levels of cardiac biomarkers
   C. Findings of diagnostic imaging
   D. All of the above

2. Over the past several decades, the STEMI mortality rate has been declining. Which of the following is NOT a contributing factor in this decline?
   A. Decreased overall incidence of MI
   B. Decreased post-MI survival rate
   C. Decreased proportion of ACS patients who present with STEMI vs NSTEMI
   D. All of the above

3. Among patients with MI, approximately what percentage presents with acute STEMI?
   A. 5%-10%
   B. 10%-15%
   C. 30%-45%
   D. 75%-90%

4. Which of the following conditions describes chest pain or equivalent ischemic discomfort that occurs at rest or with minimal exertion and lasts longer than 10 minutes?
   A. Rest angina
   B. New-onset angina
   C. Increasing angina
   D. None of the above

5. The initial clinical features observed in patients with UA and those with NSTEMI are very similar, and until additional diagnostic test results become available, hours or even days may be necessary to accurately distinguish between the two conditions.
   A. True
   B. False

6. Which of the following is NOT a pathophysiologic process that is known to contribute to the development of UA?
   A. Nonocclusive thrombus on pre-existing plaque
   B. Dynamic obstruction (eg, coronary spasm or vasoconstriction)
   C. Progressive mechanical obstruction (eg, rapidly advancing atherosclerosis)
   D. Arterial widening due to inflammation and/or infection

7. Which of the following ECG findings is NOT associated with a high risk of short-term death or nonfatal MI in patients with UA?
   A. Angina at rest with transient ST-segment changes >0.05 mV
   B. New (or presumed new) bundle-branch block
   C. Unchanged ECG during an episode of chest discomfort
   D. Sustained ventricular tachycardia

8. In patients with ACS, a diagnosis of UA is established if no cardiac biomarkers are detected, whereas a diagnosis of NSTEMI is established if cardiac enzymes—markers of myocardial necrosis—have been released.
   A. True
   B. False

9. Which of the following cardiac enzymes is a particularly specific marker of myocardial necrosis?
   A. Creatine kinase
   B. Creatine kinase MB isoenzyme
   C. Troponin
   D. Myoglobin

10. Which of the following pathophysiologic processes has been implicated in the development of coronary artery occlusion in patients with STEMI?
    A. Agonist-mediated platelet stimulation
    B. Conformational change in the glycoprotein IIb/IIIa receptor
    C. Activation of the coagulation cascade
    D. All of the above

EVALUATION

(EVALUATION ID# - 3871 ES 13)

Postgraduate Institute for Medicine (PIM) respects and appreciates your opinions. To assist us in evaluating the effectiveness of this activity and to make recommendations for future educational offerings, please take a few minutes to complete this evaluation form. You must complete this evaluation form to receive acknowledgment of participation for this activity.

Please answer the following questions by circling the appropriate rating:
5 = Outstanding 4 = Good 3 = Satisfactory 2 = Fair 1 = Poor

1. Extent to Which Program Activities Met the Identified Objectives.
   a. Understand the pathophysiology of acute coronary syndrome (ACS), particularly the progression of coronary atherosclerosis and the mediators of plaque rupture, thrombosis, and myocardial ischemia 5 4 3 2 1
   b. Describe the incidence and prevalence of different types of ACS, particularly ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina (UA) 5 4 3 2 1
   c. Identify the clinical features that distinguish STEMI and NSTEMI 5 4 3 2 1
   d. Recognize the distinctive clinical presentations and pathophysiologic causes of UA 5 4 3 2 1
   e. Describe the roles of various cardiac biomarkers in the diagnosis and risk-stratification of patients with ACS 5 4 3 2 1
   f. Describe the role of electrocardiographic and other diagnostic imaging in the evaluation of patients with suspected ACS 5 4 3 2 1

2. Overall Effectiveness of the Activity.
   a. Was timely and will influence how I practice 5 4 3 2 1
   b. Will assist me in improving patient care 5 4 3 2 1
   c. Fulfilled my educational needs 5 4 3 2 1
   d. Avoided commercial bias or influence 5 4 3 2 1

3. Impact of the Activity.
   a. The information presented… **(select all that apply)**
      ❑ Reinforced my current practice/treatment habits
      ❑ Will improve my practice/patient outcomes
      ❑ Provided new ideas or information I expect to use
      ❑ Enhanced my current knowledge base
   b. Will the information presented cause you to make any changes in your practice? *Circle One:* YES NO
   c. If yes, please describe any change(s) you plan to make in your practice as a result of this activity. _____
   d. How committed are you to making these changes?
      Circle One: 5 4 3 2 1 *(5-Very committed, 1-NOT at all committed)*

4. Future Activities. Do you feel future activities on this subject matter are necessary and/or important to your practice?
   Circle One: YES NO

5. Please list any other topics that would be of interest to you for future educational activities._____

Follow-up: As part of our ongoing continuous quality-improvement effort, we conduct post-activity follow-up surveys to assess the impact of our educational interventions on professional practice. Please indicate your willingness to participate in such a survey:

❑ **YES, I would be interested in participating in a follow-up survey**
❑ **NO, I'm not interested in participating in a follow-up survey**

Additional comments about this activity: _____

POSTTEST ANSWER KEY

If you wish to receive acknowledgment of participation for this activity, please complete the posttest by selecting the best answer to each question, complete this evaluation verification of participation and FAX to: 303-790-4876

1: ☐ 2: ☐ 3: ☐ 4: ☐ 5: ☐ 6: ☐ 7: ☐ 8: ☐ 9: ☐ 10: ☐

REQUEST FOR CREDIT

Name:_____

Degree:_____ Specialty:_____

Address:_____

City:_____ State:_____ Zip: _____

Phone:_____ Fax:_____

Email:_____

❑ **I participated in the entire activity and claim 1.2 credits**

Signature:_____

Date Completed:_____