ACC/AHA Pocket Guideline Update

Management of Patients With Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction

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A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

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The following material was adapted from the *ACC/AHA Guidelines for the Management of Patients With Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction*. For a copy of the summary article, (J Am Coll Cardiol, 2002;40:1366-74; Circulation, 2002, 106:1893-1900) and full report, visit our Web sites at *http://www.acc.org* or *http://www.americanheart.org* or call the ACC Resource Center at 1-800-253-4636, ext. 694.

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I. Introduction

Coronary artery disease (CAD) is the leading cause of death in the United States. Unstable angina (UA) and the closely related condition non-ST-segment elevation myocardial infarction (NSTEMI) are very common manifestations of this disease and are responsible for approximately 1.5 million hospitalizations in the United States each year. UA and NSTEMI are acute coronary syndromes (ACSs) that are characterized by an imbalance between myocardial oxygen supply and demand. The most common cause is the reduced myocardial perfusion that results from coronary artery narrowing caused by a nonocclusive thrombus that has developed on a disrupted atherosclerotic plaque. UA and NSTEMI are considered to be closely related conditions whose pathogenesis and clinical presentations are similar but of differing severity; they differ primarily in whether the ischemia is severe enough to cause sufficient myocardial damage to release detectable quantities of a marker of myocardial injury.

The customary American College of Cardiology/American Heart Association (ACC/AHA) classifications I, II, and III are used herein to summarize the expert opinion and provide final recommendations for both patient evaluation and therapy:

Class I	Conditions for which there is evidence and/or general agreement that a given procedure or treatment is useful and effective.
Class II	Conditions for which there is conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of a procedure or treatment. Class IIa Weight of evidence/opinion is in favor of usefulness/efficacy. Class IIb Usefulness/efficacy is less well established by evidence/opinion.
Class III	Conditions for which there is evidence and/or general agreement that a procedure/treatment is not useful/effective and in some cases may be harmful.

II. Initial Evaluation and Management

A. Clinical Assessment

Recommendations for Initial Triage

Class I

1. Patients with possible ACS should not be evaluated solely over the telephone, but should be referred to a facility that allows evaluation by a physician and the recording of a 12-lead electrocardiogram (ECG).

2. Patients with a suspected ACS with chest discomfort at rest for >20 minutes, hemodynamic instability, or recent syncope or presyncope should be strongly considered for immediate referral to an emergency department or a specialized chest pain unit.

B. Early Risk Stratification (See Table 1)

Recommendations

Class I

1. Patients who present with chest discomfort should undergo early risk stratification that focuses on anginal symptoms, physical findings, ECG findings, and biomarkers of cardiac injury.

2. A 12-lead ECG should be obtained immediately in patients with ongoing chest discomfort.

3. Biomarkers of cardiac injury should be measured in all patients who present with chest discomfort consistent with ACS. A cardiac-specific troponin is the preferred marker, and if available, it should be measured in all patients. Creatine phosphokinase-MB isoenzyme (CK-MB) by mass assay is also acceptable. In patients with negative cardiac markers within 6 hours of the onset of pain, another sample should be drawn between 6 and 12 hours.

Class IIb

1. C-reactive protein (CRP) and other markers of inflammation should be measured.

Class III

1. Total CK (without MB), aspartate aminotransferase (AST), serum glutamic oxaloacetic transaminase (SGOT), beta-hydroxybutyric dehydrogenase, and/or lactate dehydrogenase for the detection of myocardial injury.

Table 1. Short-Term Risk of Death or Nonfatal MI in Patients With Unstable Angina

Feature	High Risk (At Least 1 of the Following Features Must Be Present)	Intermediate Risk (No High-Risk Feature but Must Have 1 of the Following Features)	Low Risk (No High- or Intermediate-Risk Feature but May Have any of the Following Features)
History	Accelerating tempo of ischemic symptoms in preceding 48 h	Prior MI, peripheral or cerebrovascular disease, or CABG; prior aspirin use	
Character of pain	Prolonged ongoing (>20 min) rest pain	Prolonged (>20 min) rest angina, now resolved, with moderate or high likelihood of CAD Rest angina (<20 min or relieved with rest or sublingual nitroglycerin)	New-onset or progressive CCS Class III or IV angina in the past 2 weeks with moderate or high likelihood of CAD
Clinical findings	Pulmonary edema, most likely related to ischemia New or worsening MR murmur S_3 or new/worsening rales Hypotension, bradycardia, tachycardia Age >75 yrs	Age >70 yrs	
ECG findings	Angina at rest with transient ST-segment changes >0.05 mV Bundle-branch block, new or presumed new Sustained ventricular tachycardia	T-wave inversions >0.2 mV Pathological Q-waves	Normal or unchanged ECG during an episode of chest discomfort
Cardiac markers	Elevated (eg, TnT or TnI >0.1 ng/mL)	Slightly elevated (eg, TnT >0.01 but <0.1 ng/mL)	Normal

CABG indicates coronary artery bypass graft; CAD, coronary artery disease; CCS, Canadian Cardiovascular Society; ECG, electrocardiogram MI, myocardial infarction; MR, mitral regurgitation; TnT, troponin T; and TnI, troponin I.

Recommendation for the Diagnosis of Noncardiac Causes of Symptoms

The major objectives of the physical examination are to identify potential precipitating causes of myocardial ischemia (eg, uncontrolled hypertension or thyrotoxicosis), evidence of other cardiac disease (eg, aortic stenosis or hypertrophic cardiomyopathy), and comorbid conditions (eg, pulmonary disease) and to assess the hemodynamic impact of the ischemic event.

Class I	1. The initial evaluation of the patient with suspected ACS should include a search for noncoronary causes that could explain the development of symptoms.

Tools for Risk Stratification

The 12-lead ECG lies at the center of the decision pathway for the evaluation and management of patients with ischemic discomfort. A recording made during an episode of the presenting symptoms is particularly valuable. Importantly, transient ST-segment changes (>0.05 mV) that develop during a symptomatic episode at rest and that resolve when the patient becomes asymptomatic strongly suggest acute ischemia and a very high likelihood of underlying severe CAD.

Biomarkers are of critical importance in the evaluation of patients with UA/NSTEMI *(Table 2)*. The troponins offer great diagnostic sensitivity because of their ability to identify patients with lesser amounts of myocardial damage. Nevertheless, these lesser amounts of damage are associated with a high risk in patients with ACSs because they are thought to represent microinfarctions that result from microemboli from an unstable plaque.

Table 2. Biochemical Cardiac Markers for the Evaluation and Management of Patients Suspected of Having an ACS but Without ST-Segment Elevation on 12-Lead ECG

Marker	Point-of-Care Test Available	Advantages	Disadvantages	Clinical Recommendation
Cardiac troponins	Yes	1. Powerful tool for risk stratification 2. Greater sensitivity and specificity than CK-MB 3. Detection of recent MI up to 2 weeks after onset	1. Low sensitivity in very early phase of MI (<6 h after symptom onset) 2. Limited ability to detect late minor reinfarction	Useful as a single test to efficiently diagnose NSTEMI (including minor myocardial damage), with serial measurements.
CK-MB	Yes	1. Rapid, cost-efficient, accurate assays 2. Ability to detect early reinfarction	1. Loss of specificity in setting of skeletal muscle disease or injury, including surgery 2. Low sensitivity during very early MI (<6 h after symptom onset) or later after symptom onset (>36 h) and for minor myocardial damage (detectable by troponins)	Prior standard and still acceptable diagnostic test in most clinical circumstances
Myoglobin	Yes	1. High sensitivity 2. Useful in early detection of MI 3. Detection of reperfusion 4. Most useful in ruling out MI	1. Very low specificity in setting of skeletal muscle injury or disease 2. Rapid return to normal range limits sensitivity for later presentations	Should not be used as only diagnostic marker because of lack of cardiac specificity

ACS indicates acute coronary syndrome; CK-MB, creatine phosphokinase-MB isoenzyme; ECG, electrocardiogram

MI, myocardial infarction; NSTEMI, non-ST-segment elevation myocardial infarction

Initial Evaluation

C. Immediate Management (Figure 1)

Recommendations

Class I

1. The history, physical examination, 12-lead ECG, and initial cardiac marker tests should be integrated to assign patients with chest pain to 1 of 4 categories: a noncardiac diagnosis, chronic stable angina, possible ACS, and definite ACS.

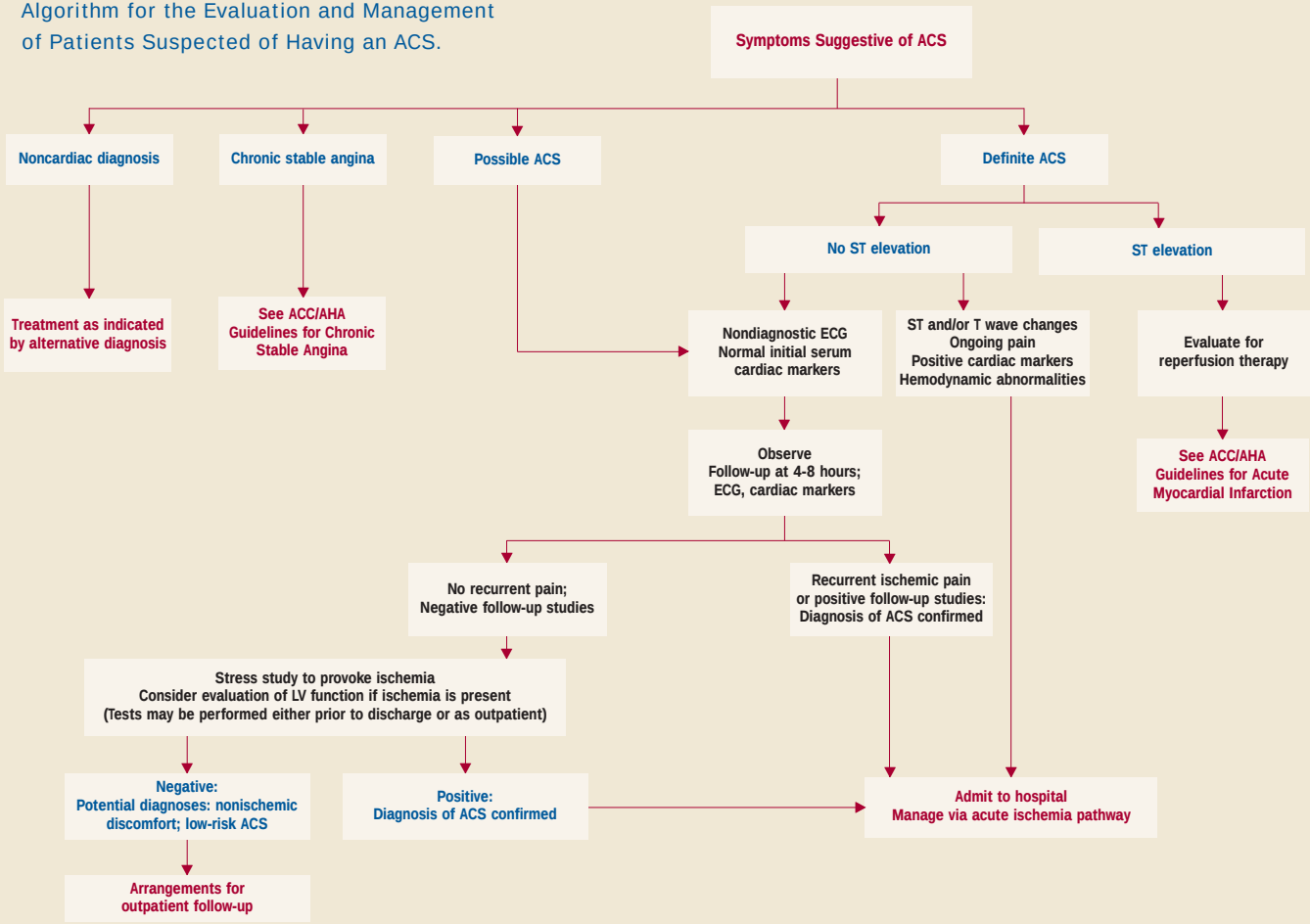
2. Patients with definite or possible ACS whose initial 12-lead ECG and cardiac marker levels are normal should be observed in a facility with cardiac monitoring, and a repeat ECG and cardiac marker measurement should be obtained 6 to 12 hours after the onset of symptoms.

3. In patients in whom ischemic heart disease is present or suspected, if the follow-up 12-lead ECG and cardiac marker measurements are normal, a stress test (exercise or pharmacological) to provoke ischemia may be performed. Low-risk patients with a negative stress test can be managed as outpatients.

4. Patients with definite ACS and ongoing pain, positive cardiac markers, new ST-segment deviations, new deep T-wave inversions, hemodynamic abnormalities, or a positive stress test should be admitted to the hospital.

5. Patients with possible ACS and negative cardiac markers who are unable to exercise or who have an abnormal resting ECG should have a pharmacological stress test.

Figure 1.
Algorithm for the Evaluation and Management of Patients Suspected of Having an ACS.



Initial Evaluation

III. Hospital Care

A. Anti-Ischemic Therapy

Recommendations

Class I

1. Bed rest with continuous ECG monitoring for ischemia and arrhythmia detection in patients with ongoing rest pain.

2. Sublingual followed by intravenous nitroglycerin (NTG) for immediate relief of ischemia and associated symptoms.

3. Morphine sulfate intravenously when symptoms are not immediately relieved with NTG or when acute pulmonary congestion is present.

4. A beta blocker, with the first dose administered intravenously if there is ongoing chest pain, followed by oral administration, in the absence of contraindications.

5. A nondihydropyridine calcium antagonist (eg, verapamil or diltiazem) in the absence of severe left ventricular (LV) dysfunction or other contraindications in patients with continuing or frequently recurring ischemia when beta blockers are contraindicated.

6. An angiotensin converting enzyme inhibitor (ACEI) when hypertension persists despite treatment with NTG and a beta blocker in patients with LV systolic dysfunction or congestive heart failure (CHF) and in ACS patients with diabetes.

Class IIa

1. Oral long-acting calcium antagonists for recurrent ischemia in the absence of contraindications and when beta blockers and nitrates are fully used.

2. An ACEI for all post-ACS patients.

Class IIb

1. Extended-release form of nondihydropyridine calcium antagonists instead of a beta blocker.

2. Immediate-release dihydropyridine calcium antagonists in the presence of a beta blocker.

Class III

1. NTG or other nitrate within 24 hours of sildenafil (Viagra) use.

2. Immediate-release dihydropyridine calcium antagonists in the absence of a beta blocker.

Table 3 shows the recommended doses of the various antiplatelet and antithrombotic agents.

B. Antiplatelet and Anticoagulation Therapy

Antithrombotic therapy is essential to modify the disease process and its progression to death, myocardial infarction (MI), or recurrent MI. A combination of aspirin (ASA), clopidogrel, and unfractionated or low molecular weight

Hospital Care

Table 3.
Clinical Use of Antiplatelet and Antithrombotic Therapy

Oral Antiplatelet Therapy	
Aspirin	Initial dose of 162-325 mg nonenteric formulation followed by 75-160 mg/d of an enteric or a nonenteric formulation
Clopidogrel (Plavix)	75 mg/d; a loading dose of 4-8 tablets (300-600 mg) can be used when rapid onset of action is required
Ticlopidine (Ticlid)	250 mg twice daily; a loading dose of 500 mg can be used when rapid onset of inhibition is required; monitoring of platelet and white cell counts during treatment is required
Heparins	
Dalteparin (Fragmin)	120 IU/kg subcutaneously every 12 h (maximum 10,000 IU twice daily)
Enoxaparin (Lovenox)	1 mg/kg subcutaneously every 12 h; the first dose may be preceded by a 30 mg IV bolus
Heparin (UFH)	Bolus 60-70 U/kg (maximum 5000 U) IV followed by infusion of 12-15 $U \cdot kg^{-1} \cdot h^{-1}$ (maximum 1000 U/h) titrated to aPTT 1.5-2.5 times control
Intravenous Antiplatelet Therapy	
Abciximab (ReoPro)	0.25 mg/kg bolus followed by infusion of 0.125 $mcg \cdot kg^{-1} \cdot min^{-1}$ (maximum 10 mcg/min) for 12 to 24 h
Eptifibatide (Integrilin)	180 mcg/bolus followed by infusion of 2.0 $mcg \cdot kg^{-1} \cdot min^{-1}$ for 72 to 96 h*
Tirofiban (Aggrastat)	0.4 $mcg \cdot kg^{-1} \cdot min^{-1}$ for 30 minutes followed by infusion of 0.1 $mcg \cdot kg^{-1} \cdot min^{-1}$ for 48 to 96 h*

**Different dose regimens were tested in recent clinical trials before percutaneous interventions.*

heparin, represents the most effective therapy. A platelet glycoprotein GP IIb/IIIa receptor antagonist should be used in patients with continuing ischemia or with other high-risk features in whom an early invasive strategy is planned.

For patients in whom there are contraindications for ASA use, clopidogrel should be administered. In the absence of a high risk for bleeding, aspirin and clopidogrel should be administered prior to PCI and clopidogrel should be continued for at least one month after stenting. Aspirin should be continued for an indefinite period.

Heparin (either UFH or low molecular weight heparin [LMWH]) is a key component in the antithrombotic management of UA/NSTEMI. The dose of UFH should be titrated to an activated partial thromboplastin time that is 1.5 to 2.5 times control. Advantages of LMWH preparations are the ease of subcutaneous administration and the absence of a need for monitoring. Furthermore, the LMWHs stimulate platelets less than UFH does and are less frequently associated with heparin-induced thrombocytopenia. However, they appear to be associated with significantly more frequent minor (but not major) bleeding.

When platelets are activated, the GP IIb/IIIa receptor undergoes a change in configuration that results in binding of fibrinogen to platelet receptors, resulting in platelet aggregation. The efficacy of GP IIb/IIIa antagonists in prevention of the complications associated with percutaneous coronary intervention (PCI) has been documented in numerous trials, many of which were composed entirely or in large part of patients with

UA. Trials with tirofiban and one trial with eptifibatide have also shown their efficacy in UA/NSTEMI patients, only some of whom underwent interventions. In PCI trials, the administration of abciximab consistently showed a significant reduction in the rate of MI and the need for urgent revascularization.

Treatment with GP IIb/IIIa blockers increases the risk of bleeding, which is typically mucocutaneous or involves the access site of vascular intervention. Blood hemoglobin and platelet counts should be monitored, and patient surveillance for bleeding should be performed daily during the administration of GP IIb/IIIa blockers.

Recommendations

Class I

1. Antiplatelet therapy should be initiated promptly. ASA should be administered as soon as possible after presentation and continued indefinitely.

2. Clopidogrel should be administered to hospitalized patients who are unable to take ASA because of hypersensitivity or major gastrointestinal intolerance.

3. In hospitalized patients in whom an early noninterventional approach is planned, clopidogrel should be added to ASA as soon as possible on admission and administered for at least 1 month and for up to 9 months.

4. In patients for whom a PCI is planned, clopidogrel should be started and continued for at least 1 month and up to 9 months in patients who are not at high risk for bleeding.

5. In patients taking clopidogrel in whom CABG is planned, if possible the drug should be withheld for at least 5 days, and preferably for 7 days.

6. Anticoagulation with subcutaneous LMWH or intravenous unfractionated heparin (UFH) should be added to antiplatelet therapy with ASA and/or clopidogrel.

7. A platelet GP IIb/IIIa antagonist should be administered, in addition to ASA and heparin, to patients in whom catheterization and PCI are planned. The GP IIb/IIIa antagonist may also be administered just prior to PCI.

Class IIa

1. Enoxaparin is preferable to UFH as an anticoagulant in the absence of renal failure and unless CABG is planned within 24 h.

Class III

1. Intravenous fibrinolytic therapy in patients without acute ST-segment elevation, a true posterior MI, or a presumed new left-bundle-branch block (LBBB).

2. Abciximab administration in patients in whom PCI is not planned.

C. Risk Stratification

The management of patients with an ACS requires continuous risk stratification. The goals of noninvasive testing are to determine the presence or absence of ischemia in patients with a low likelihood of CAD and to estimate prognosis.

Because of simplicity, lower cost, and widespread familiarity with performance and interpretation, the standard low-level exercise ECG stress test remains the most reasonable test in patients able to exercise who have a resting ECG that is interpretable for ST-segment shifts. Patients with an ECG pattern that would interfere with interpretation of the ST segment should have an exercise test with imaging. Patients who are unable to exercise should have a pharmacological stress test with imaging.

Recommendations

Class I	1. Noninvasive stress testing in low-risk patients *(Table 1)* who have been free of ischemia at rest or with low-level activity and free of CHF for a minimum of 12 to 24 hours.
	2. Noninvasive stress testing in patients at intermediate risk *(Table 1)* who have been free of ischemia at rest or with low-level activity and of CHF for a minimum of 2 or 3 days.
	3.Choice of stress test is based on the resting ECG, ability to perform exercise, local expertise, and technologies available.
	4. Prompt angiography without noninvasive risk stratification for failure of stabilization with intensive medical treatment.
Class IIa	1. A noninvasive test (echocardiogram or radionuclide angiogram) to evaluate LV function in patients with definite ACS who are not scheduled for coronary arteriography and left ventriculography.

D. Early Conservative Versus Invasive Strategies

Two different treatment strategies, termed “early conservative” and “early invasive,” have evolved for patients with UA/NSTEMI. In the early conservative strategy, coronary angiography is reserved for patients with evidence of recurrent ischemia (angina or ST-segment changes at rest or with minimal activity) or a strongly positive stress test despite vigorous medical therapy. In the early invasive strategy, patients without clinically obvious contraindications to coronary revascularization are routinely recommended for early coronary angiography and angiographically directed revascularization, if possible.

In patients with UA/NSTEMI without recurrent ischemia in the first 24 hours, the use of early angiography provides a convenient approach to risk stratification. It can identify the patients with no significant coronary stenoses and those with

3-vessel disease with LV dysfunction or left main disease. The former group has an excellent prognosis, whereas the latter group may derive a survival benefit from coronary artery bypass graft surgery (CABG). In addition, early PCI of the culprit lesion has the potential to reduce the risk for subsequent hospitalization and the need for multiple antianginal drugs compared with the early conservative strategy. In patients without high-risk features, coronary arteriography is optional and can be safely deferred.

Recommendations

Class I

1. An early invasive strategy is recommended in patients with UA/NSTEMI and any of the following high-risk indicators:

a) Recurrent angina/ischemia at rest or with low-level activities despite intensive anti-ischemic therapy.

b) Elevated TnT or TnI.

c) New or presumed new ST-segment depression at presentation.

d) Recurrent angina/ischemia with CHF symptoms, an S_3 gallop, pulmonary edema, worsening rales, or new or worsening mitral regurgitation.

e) High-risk findings on noninvasive stress testing.

f) Depressed LV systolic function (eg, ejection fraction [EF] < 0.40 on noninvasive study).

g) Hemodynamic instability or angina at rest accompanied by hypotension.

h) Sustained ventricular tachycardia.

i) PCI within 6 months.

j) Prior CABG.

2. In the absence of these findings, either an early conservative or an early invasive strategy in hospitalized patients without contraindications for revascularization.

Class IIa

1. An early invasive strategy in patients with repeated presentations for ACS despite therapy and without evidence of ongoing ischemia or high risk.

Class III

1. Coronary angiography in patients with extensive comorbidities (eg, liver or pulmonary failure, cancer), in whom risks of revascularization are not likely to outweigh the benefits.

2. Coronary angiography in patients with acute chest pain and a low likelihood of ACS.

IV. Coronary Revascularization

Coronary revascularization (PCI or CABG) is performed to improve prognosis, relieve symptoms, prevent ischemic complications, and improve functional capacity. Patients with high-risk coronary anatomy are likely to benefit from revascularization in terms of both symptom improvement and long-term survival. The indications for coronary revascularization in patients with UA/NSTEMI are similar to those for patients with chronic stable angina. The majority of current PCIs now involve balloon dilatation followed by coronary stenting. Stenting has contributed greatly to catheter-based revascularization by reducing the risk of both acute vessel closure and late restenosis. PCI can lead to angiographic success in most patients with UA/NSTEMI. An important advance in the treatment of patients with UA/NSTEMI undergoing PCI has been the introduction of platelet GP IIb/IIIa receptor inhibitors. This therapy takes advantage of the fact that platelets play an important role in the development of ischemic complications that may occur during PCI. The safety of these procedures in these patients is enhanced by the addition of intravenous platelet GP IIb/IIIa receptor inhibitors to the standard regimen of ASA, heparin, and anti-ischemic medications.

Recommendations for Revascularization With PCI and CABG in Patients With UA/NSTEMI

Class I	1. CABG for patients with significant left main CAD.
	2. CABG for patients with 3-vessel CAD; the survival benefit is greater in patients with abnormal LV function (EF < 0.50).
	3. CABG for patients with 2-vessel CAD with significant proximal left anterior descending CAD and either abnormal LV function (EF < 0.50) or demonstrable ischemia on noninvasive testing.
	4. PCI or CABG for patients with 1- or 2-vessel CAD without significant proximal left anterior descending CAD but with a large area of viable myocardium and high-risk criteria on noninvasive testing.
	5. PCI for patients with multivessel CAD with suitable coronary anatomy, with normal LV function, and without diabetes.
	6. CABG with the internal mammary artery for patients with multivessel CAD and treated diabetes mellitus.
	7. Intravenous platelet GP IIb/IIIa inhibitor in UA/NSTEMI patients undergoing PCI.

Class IIa	1. PCI or CABG for patients with 1-vessel CAD with significant proximal left anterior descending CAD.
Class IIb	1. PCI for patients with 2- or 3-vessel CAD with significant proximal left anterior descending CAD, with treated diabetes or abnormal LV function, and with anatomy suitable for catheter-based therapy.
Class III	1. PCI or CABG for patients with insignificant coronary stenosis (< 50% diameter). 2. PCI in patients with significant left main coronary artery disease who are candidates for CABG.

V. Hospital Discharge and Post-Hospital Discharge Care

The acute phase of UA/NSTEMI is usually over within 2 months. The risk of progression to MI or the development of recurrent MI or death is highest during this period. Most patients then resume a clinical course similar to that of patients with chronic, stable CAD.

A. Medical Regimen

An effort of the entire staff (physicians, nurses, dietitians, pharmacists, rehabilitation specialists, and physical and occupational therapists) is often necessary to prepare the patient for discharge. Direct patient instruction is important and should be reinforced and documented with written instruction sheets. Enrollment in a cardiac rehabilitation program after discharge may enhance patient education and compliance with the medical regimen.

Recommendations for Postdischarge Therapy

Class I

1. Before hospital discharge, the patients and/or a designated responsible caregiver should be provided with well-understood instructions with respect to medication type, purpose, dose, frequency, and pertinent side effects.

2. Drugs required in the hospital to control ischemia should be continued after hospital discharge in patients who do not undergo coronary revascularization. Adjustment of the dose may be required.

3. Anginal discomfort that lasts > 2 or 3 minutes should prompt the patient to discontinue the activity or remove himself or herself from the stressful event. If pain does not subside immediately, the patient should be instructed to take NTG. Pain that lasts >15 to 20 minutes or persistent pain despite 3 NTG doses should prompt the patient to seek immediate medical attention by calling 9-1-1 and going to the nearest hospital ED.

4. If the pattern of anginal symptoms changes (eg, pain that is more frequent or severe, is precipitated by less effort, or now occurs at rest), the patient should contact his or her physician to determine the need for additional treatment or testing.

5. ASA 75 to 325 mg/d in the absence of contraindications.

6. Clopidogrel 75mg daily (in the absence of contraindications) when ASA is not tolerated because of hypersensitivity or gastrointestinal intolerance.

7. The combination of ASA and clopidogrel for up to 9 months after UA/NSTEMI.

8. Beta-blockers in the absence of contraindications.

9. Lipid-lowering agents and diet in post ACS patients with low-density lipoprotein (LDL) cholesterol > 130 mg/dL, including after revascularization.

10. Lipid-lowering agents if LDL cholesterol level after diet is > 100 mg/dL.

11. ACEIs for patients with CHF, LV dysfunction (EF < 0.40), hypertension, or diabetes.

A reduction in mortality and vascular event rates was reported in one large trial, the Heart Outcomes Prevention Evaluation (HOPE) Study, with the long-term use of an ACEI in moderate-risk patients with CAD, many of whom had preserved LV function, as well as in patients at a high risk of developing CAD. ACEI use should be considered in these patients as well.

B. Risk Factor Modification

The healthcare team should work with patients and their families to educate them regarding specific target levels for cholesterol, blood pressure, and weight. The family may be able to further support the patient by also making changes in risk behavior (eg, cooking low-fat meals for the entire family, exercising together). Beyond instructions for daily exercise, patients require specific instruction on activities (eg, heavy lifting, climbing stairs, yard work, household activities) that are permissible and those that should be avoided. Specific mention should be made regarding when they can resume driving and return to work.

Recommendations

Class I	1. Specific instructions should be given regarding the following: a) Smoking cessation and achievement or maintenance of optimal weight, daily exercise, and diet. b) HMG-CoA (hydroxy-methylglutaryl coenzyme A) reductase inhibitors for LDL cholesterol of > 130 mg/dL and if LDL is > 100 mg/dL after diet. c) A fibrate or niacin if high-density lipoprotein (HDL) cholesterol is less than 40mg per dL, occurring as an isolated finding or in combination with other lipid abnormalities. d) Hypertension control to a blood pressure < 130/85 mm Hg. e) Tight control of hyperglycemia in diabetes. 2. Referral of patients who are smokers to a smoking cessation program or clinic and/or an outpatient cardiac rehabilitation program.
Class IIa	1. HMG-CoA reductase inhibitors and diet for LDL cholesterol greater than 100 mg per dL begun 24 to 96 h after admission and continued at hospital discharge.