Guidelines for Diagnosis and Management of Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction

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Abstract

Heart disease continues to be the number one cause of death for men and women in the United States. New research findings and recommendations have made it necessary for the American College of Cardiology and the American Heart Association to revise the guidelines for treatment of unstable angina and non-ST-segment elevation myocardial infarction. By implementing these guidelines, health care providers can deliver quality care and improve outcomes for those patients with unstable angina and non-ST-segment elevation myocardial infarction.

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of death for men and women in the United States[1]. Acute Coronary Syndromes (ACS) is a term used for clinical symptoms consistent with myocardial ischemia. ACS includes acute myocardial infarction (AMI), which is both ST elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI), along with unstable angina (UA). UA/NSTEMI is usually caused by atherosclerotic disease, which is associated with an increased risk for cardiac death and myocardial infarction (MI). The pathological process of UA/NSTEMI is plaque rupture and thrombosis in the coronary arteries, which diminish the blood flow to the myocardium causing myocardial ischemia.

The purpose of these guidelines is to assist health care providers in the diagnosis and treatment of UA/NSTEMI. The American College of Cardiology (ACC) and the American Heart Association (AHA) formed a Task Force to make recommendations in the diagnosis and treatment of UA/NSTEMI. The current guidelines are an update from the 2000 guidelines; the 2002 update was initiated due to the recent evidence and results from clinical trials related to the treatment of UA/NSTEMI.

INITIAL EVALUATION AND MANAGEMENT

Atherosclerotic CAD usually causes UA/NSTEMI, which is associated with risk of cardiac death. Often it is difficult to discern clinically those patients with ACS and patients presenting with similar symptoms that do not have CAD. Patients presenting with signs and symptoms suggestive of ACS should be monitored with continuous electrocardiographic (ECG) monitoring. There must be capability to obtain a 12-lead ECG and defibrillation if necessary. Patients that are determined to have an AMI should receive immediate reperfusion therapy. During the initial evaluation there are four potential diagnoses: ACS, non-ACS cardiac condition, non-cardiac condition with another specific disease, or a non-cardiac condition that is unknown.[1]

UA/NSTEMI is a product of inadequate supply and/or increased demand of oxygen to the myocardium. There are 5 causes of UA/NSTEMI. First, the most common cause is decreased myocardial perfusion from coronary artery narrowing that may be caused by a non-occlusive thrombus. Second, a dynamic obstruction caused by a spasm in the coronary artery may be occurring. Third, there may be narrowing of the vessel without spasm or thrombus formation. Fourth, inflammation in the vessel related to infection may be the cause for UA/NSTEMI. Finally, there may be secondary UA that is caused by other factors such as fever, hypotension, thyrotoxicosis, or anemia.[1]

Along with the possible causes of UA/NSTEMI there are 3 common categories for the presentation of UA. First, rest angina occurs while the patient is at rest and lasts for more than 20 minutes. Second, new-onset angina causes marked

limitation of regular activity; finally, increasing angina is angina that occurs more often and lasts longer than normal.[1] Initial evaluation and management of patients suspected of having ACS should be done rapidly to determine early treatment. Patients that have symptoms of ACS should not be evaluated only via the telephone, but should be referred to a facility with the capability to perform 12-lead ECGs and where health care providers are knowledgeable in the evaluation of ACS. Patients with suspected ACS who have chest discomfort for more than 20 minutes, any hemodynamic instability, or a recent syncopal episode should be referred to an Emergency Department (ED) or a chest pain unit immediately.[1]

In order to determine a correct diagnosis of ACS and appropriate interventions patients must undergo early risk stratification. All patients presenting with chest discomfort suspicious for ischemia that is related to CAD should be classified as high, intermediate, or low. Those patients categorized as high likelihood for ACS are those patients with chest pain or left arm pain, known history of CAD or MI, hypotension, diaphoresis, pulmonary edema, ST-segment deviation, T-wave inversion, elevated troponins (TnI or TnT) or elevated creatinine kinase (CK).[1]

Patients classified as having intermediate likelihood of ACS are those patients with: chest pain or left arm pain, greater than 70 years old, diabetes mellitus (DM), extracardiac vascular disease, no Q waves, no abnormal ST-segments, no abnormal T-waves, and normal cardiac markers. On the other hand, those patients with low likelihood of ACS are those patients with recent cocaine use, chest discomfort with palpation of the chest wall, and normal cardiac markers.[1]

Initial evaluation of patients suspected to have ACS should include differential diagnoses that could possibly explain presenting symptoms. Patients suspected of having UA/NSTEMI may be divided into 2 categories: those patients with known CAD and those patients with new ischemic signs and symptoms. There are 5 factors that need to be determined from the history and physical in order to discern the likelihood of CAD ischemia: 1) nature of the anginal symptoms, 2) history of CAD, 3) gender, 4) age, and 5) the number of risk factors for CAD.[1]

Angina is described as chest discomfort that is deep and not localized. Angina is usually associated with physical exertion or emotional distress that is relieved by nitroglycerin (NTG). Some patients may have angina at rest.

Jaw, neck, ear, arm, or epigastric pain may be the symptom associated with angina rather than chest discomfort alone. Some patients, especially the elderly and women, may present with atypical anginal symptoms such as nausea, vomiting, diaphoresis or fatigue. The following symptoms are not characteristic of myocardial ischemia: pleuritic pain; mid or lower abdominal pain; pain localized at the tip of one finger on the chest; pain that is reproducible by palpation; pain lasting for hours; brief pain lasting only seconds or pain that radiates to the lower extremities.[1]

Although heart disease continues to be the primary cause of death for men and women in the United States, there is a difference in the type of ACS they experience. More women than men present with STEMI, of those patients presenting with NSTEMI, men more than women will be diagnosed with NSTEMI.[2] Women with STEMI tend to have a worse outcome than men with STEMI. Women have better outcomes with UA and outcomes are very similar for men and women with NSTEMI.[3,4]

Elderly patients are more likely to present with atypical symptoms along with having an increased risk of an adverse outcome than younger patients. Elderly patients are more likely to have more severe, mulitvessel coronary disease. Also, the left ventricular function is more likely to be decreased in the elderly placing them at an increased risk for negative outcomes. The presence of traditional risk factors for heart disease such as hypertension and smoking are not always predictive of cardiac ischemia, but these risk factors along with a history of ACS is predictive of a poor outcome.[1] Diabetes is a major risk factor for patients with ACS to have poorer outcomes, including death and acute heart failure. Current smoking does not actually carry a higher risk of mortality, probably related to the less severe underlying CAD.[1]

In the immediate management of patients presenting with typical or atypical symptoms of ACS the health care provider should perform an initial history, physical examination, 12-lead ECG, and cardiac marker tests. This information will assist in assigning patients to one of the following categories: noncardiac diagnosis, chronic stable angina, possible ACS, or definite ACS. The history will provide information related to the patient's family history, current medical problems, risk factors for CAD, and the description of the type of chest pain or discomfort the patient is experiencing. The physical examination may reveal other causes for the patient's symptoms. Patients with CAD may

have exacerbation of their symptoms related to gastrointestinal bleeding, COPD, hypertension or hyperthyroidism. All patients should have vital signs obtained along with a thorough cardiovascular and chest examination. The 12-lead ECG will provide information related to dysrhythmias, ST-segment changes that may be indicative of myocardial ischemia or injury. Serial ECGs are beneficial to detect evolving myocardial ischemia.

The other useful clinical information includes cardiac markers. These markers detect myocardial necrosis and prognosis. Death of myocytes releases enzymes that can be detected in the blood. Cardiac markers will be positive if there has been myocardial necrosis and is helpful in the diagnosis, especially because the 12-lead ECG does not always detect myocardial necrosis. CK and CK-MB have been the primary serum markers to detect myocardial damage in the past. However, this marker has its limitations due to the increase in CK with skeletal muscle damage, making it less cardiac specific. The cardiac troponins have 3 subunits: TnT, TnI, and TnC. The TnT and TnI are cardiac specific and are the two that have immunoassays available at this time. Troponins are accurate in determining myocardial necrosis, but this necrosis may not be from CAD. Therefore, the diagnosis of an AMI cannot be made with positive troponins alone. Myoglobin is a protein found in both skeletal and cardiac muscle. It can be detected within 2 hours after the onset of myocardial necrosis, much more rapidly than the CK-MB or troponins, but it is not cardiac specific. The troponins are considered the cardiac marker to diagnose ACS at this time.[1] See Table 1 for comparison of cardiac markers.

Figure 1

Table 1: Comparison of Cardiac Markers

Marker	Advantages	Disadvantages
CK-MB	Rapid Cost effective Detected early in infarctions	Loss of specificity with skeletal muscle damage Detection after 6 hours of myocardial necrosis
Myoglobin	Highly sensitive Early detection of MI, within 2 hours Detects reperfusion Most useful in ruling out MI	Low specificity with skeletal muscle injury Rapid return to normal
Troponins	Powerful tool for risk stratification Greater sensitivity and specificity than CK-MB Detects recent MI up to 2 weeks Helpful to determine therapy Detection of reperfusion	Low sensitivity in MI of less than 6 hours Require repeat measures at 8-12 hours if first result is negative Less able to detect late, minor MIs

Due to the varied time release of the cardiac enzymes, it is important to determine how long the patient has been experiencing ischemic symptoms in order to correlate the potential rise in enzymes. Table 2 contains the categories of how patients may be triaged in determining treatments for ACS. Those patients that have been determined to have definite or possible ACS, but their initial cardiac markers and 12-lead ECG are normal, should be monitored in a facility such as a chest pain unit. The cardiac markers and 12-lead ECG should be repeated in 6 to 12 hours after the patient first experienced ischemic symptoms.[1]

Figure 2

Table 2: ACS Triage Categories

ACS Category	Symptoms	ECG	Cardiac Markers
Possible ACS	Recent chest discomfort No chest discomfort at time of evaluation	Normal or unchanged	No elevation
Definite ACS	Typical chest pain or accelerated angina	ST changes	Positive

If the follow up ECG and cardiac markers are normal for those patients who are known to have a history of CAD or suspected ischemic disease, they should then have a stress test, either exercise or pharmacological. The stress test may be performed in the ED, chest pain unit, or on an outpatient basis shortly after discharge. For those patients that are unable to undergo an exercise stress test or that have an abnormal 12-lead ECG, they should have a pharmacological stress test. Those patients with a low risk for ischemic

disease and a negative stress test can be managed as outpatients. Patients with the following should be admitted to the hospital: definite ACS, ongoing chest pain, positive cardiac markers, new ST-segment deviations, hemodynamic instability, or a positive stress test. Patients presenting with definite ACS and ST-segment elevation should be assessed for immediate reperfusion therapy, primary angioplasty or thrombolytics.[1]

HOSPITAL CARE

Patients who are hemodynamically stable should be cohorted in a step down unit with continuous ECG monitoring and careful observation. Patients hemodynamically unstable or those with continued discomfort should be monitored in a coronary care unit until they are free of major complications, such as ventricular tachycardia, ventricular fibrillation, sinus tachycardia, atrial fibrillation, atrial flutter, high degree atrioventricular (AV) blocks, sustained hypotension, recurrent ischemia, ST-segment changes, new mechanical defect such as ventricular septal defect, mitral regurgitation or heart failure for the first 24 hours.[1]

The goal of care is to relieve ischemia and prevent serious adverse outcomes and complications. Combination of treatments and interventions are selected based on recurrence of symptoms and risk stratification. The following are recommendations for anti-ischemic treatment. First, bedrest is insisted upon while ischemia is ongoing, but once ischemia is resolved patients can be allowed to sit in a chair or use a bedside commode.

Second, nitrates are administered to dilate coronary arteries and increase venous pooling, this in turn decreases myocardial preload and oxygen consumption. NTG also promotes collateral flow and redistributes blood flow to the ischemic regions. Nitrates should be initiated in the ED sublingually or by spray 5 minutes apart for three doses. If symptoms are not relieved a continuous intravenous (IV) NTG infusion should be started at 10 mcg per minute and increased every 3 to 5 minutes until symptoms are relieved or a decreased blood pressure response is noted. The maximum dose for NTG is usually 200 mcg per minute. Patients often develop a tolerance after 24 hours of administration of NTG; therefore, it is beneficial to decrease the dose gradually once symptoms have been relieved. Do not use nitrates within 24 hours of sildenafil (Viagra) because it can cause profound hypotension, MI and even death.[5]

Third, supplemental oxygen should be administered if arterial oxygen saturation decreases to less than 90%. There is no clinical evidence for routine use of oxygen.

Fourth, morphine relieves pain unrelieved by NTG and decreases myocardial oxygen demand. Morphine also causes vasodilatation and mild reduction in heart rate. One to 5 mg IV push may be administered every 3 to 5 minutes unless contraindicated due to hypotension, respiratory depression, nausea or vomiting.[1]

Fifth, beta adrenergic blockers reduce the cardiac workload and myocardial oxygen demand. These medications decrease myocardial contractility, sinus node rate and AV node conduction. They also inhibit catecholamine responses at the beta adrenergic receptor in the myocardium. Activation of the sympathetic nervous system over time can cause ventricular hypertrophy, increased heart chamber volumes and vasoconstriction. By giving patients beta blockers the risk of AMI decreases, mortality decreases, and ventricular remodeling decreases.[1] The goal is to decrease heart rate to 50 to 60 beats per minute. Initially, beta blockers should be given IV push every 5 minutes for a total of 3 doses or 15 mg. Then the patient should be started on an oral dose of 25 to 50mg every 6 hours for 48 hours and then 100 mg twice daily. Beta blockers should not be given to patients with 2nd or 3rd degree heart blocks or to patients with a history of severe left ventricular dysfunction associated with heart failure during the acute ischemic phase. Beta blockers should be used cautiously in chronic obstructive pulmonary disease (COPD) patients.[1]

Sixth, calcium antagonists such as verapamil and diltiazem can be used to control continued ischemia in patients not responding to beta blockers and nitrates or to manage hypertension in UA patients. Finally, angiotenson converting enzyme (ACE) inhibitors prevent the conversion of angiotenson I to angiotenson II, a potent vasoconstrictor. ACE inhibitors alleviate the symptoms of HF, reduce death and hospitalization. ACE inhibitors should be given to all patients with left ventricular dysfunction and can also be used to relieve angina in high risk chronic CAD patients or hypertensive patients who do not respond to nitrates or beta blockers.[1]

ANTIPLATELET AND ANTICOAGULATION THERAPY

Because ACS are caused by plaque rupture and thrombosis, antithrombotic therapy is essential to modify the disease

process and its progression to death, MI, or recurrent MI.[1] Antithrombotic therapy is designed to stop platelet aggregation and interfere with the coagulation process. Use of a combination of antiplatelets, anticoagulants, and glycoprotein (GP) IIB/IIIA inhibitors is recommended.[1] The antiplatelets include aspirin (ASA), which inhibits cycloxygenase-1 within platelets and prevents the formation of thromboxane A2 that diminishes platelet aggregation. The ISIS-2 trial, the second international study of infarct survival, indicated an ASA dose of 160 mg chewed on arrival to the ED as soon as a diagnosis of ACS is suspected or made decreases mortality.[6] Subsequent doses of ASA 75-325 mg should then be prescribed everyday indefinitely unless contraindicated due to allergy, active bleeding, uncontrolled hypertension or peptic ulcers.[1]

Thienopyridines or adenosine dephosphate receptor antagonists such as clopidogrel (plavix) and ticlopidine (ticlid) work by inhibiting platelet aggregation. Clopidogrel should be given in the ED if the patient has an allergy to ASA. There is strong evidence to support it being used concurrently with ASA in UA/NSTEMI patients who the non-interventional approach is intended. Clopidogrel should also be used routinely in patients who are undergoing percutaneous coronary interventions (PCI). Clopidogrel has shown a 34% reduction in cardiovascular death or recurrent MI when the patient is given a loading dose of 300 mg and then 75 mg orally daily. Clopidogrel should not be used if there is strong suspicion that the patient may need coronary artery bypass grafting (CABG). Clopidogrel should be held 5-7 days prior to surgery. Ticlopidine significantly reduces the rate of fatal and non-fatal MIs but its adverse effects such as diarrhea, abdominal pain, vomiting, neutropenia and thrombocytopenia purpura limit its usefulness.[1]

The anticoagulants include unfractionated heparin (UFH) and low molecular weight heparin (LMWH) which work by accelerating the action of circulating thrombin, a proteolytic enzyme that inactivates factor IIA (thrombin), factor 1Xa and factor Xa. It prevents the formation of the thrombus but does not lyse clots. Several clinical studies between 1986-1988 tested ASA and UFH against ASA and placebo and the results showed an 89% decrease in AMI and a 63% reduction in refractory angina in the groups that used ASA and UFH.[7] Heparin has limitations in that it non-specifically binds the cells and proteins and it does it differently in people. Smoking, diabetes and age alter the availability of binding so the doses have to be adjusted according to

prothrombin time (PTT) laboratory values. Heparin is also known to have a rebound effect. Once heparin is discontinued, thrombin is reactivated and increased clotting can occur. A severe side effect includes thrombocytopenia in 1-2% of patients; therefore, platelet counts must be closely monitored.[1]

LMWHs were developed through depolyerization of polysaccharide chains of heparin. All LMWHs inactivate factor Xa, but only some inactivate thrombin. The advantages of using LMWHs are that they have a longer half-life, more predictable, sustained anticoagulation and do not require frequent laboratory monitoring. The disadvantages of using LMWHs are that anticoagulation cannot be readily reversed if the patient begins to bleed or has to go emergently to CABG. Interventional cardiologists also like to monitor activated clotting time (ACT) levels during PCI and maintain them around 350 seconds; this cannot be done with LMWH. It has been suggested that patients with UA/NSTEMI can be initially stabilized with LMWH prior to PCI and UFH can be administered during PCI. Recent studies have shown a moderate benefit when using enoxaparin (lovenox) versus UFH in UA/STEMI.[8]

Hirudin is a direct thrombin inhibitor that binds to the catalytic site of thrombin to produce predictable anticoagulation. Hirudin has been extensively studied in trials against UFH and there are no clear benefits over UFH.[9] An excess of major bleeds requiring transfusions was noted in the hirudin groups. Currently hirudin is only indicated for heparin induced thrombocytopenia (HIT) and prophylaxis for deep vein thrombosis in patients undergoing a hip replacement operation.[1]

GP IIB/IIIA receptors are found on the surface of platelets. Once stimulated the receptor undergoes a chemical change that increases its affinity for binding to fibrinogen. This binding of fibrinogen to platelets helps platelets aggregate. GPIIB/IIIA inhibitors bind to this receptor site and prevent fibrinogen from binding and altercating other platelets. There are currently three drugs used including abciximab (ReoPro), eptifibatide (Integrilin) and tirofabin (Aggrastat). Studies have shown that these inhibitors bind to greater than 80% of the receptor sites and the result is protein anticoagulation. Current recommendations are that GPIIB/IIIA inhibitors are beneficial in patients with UA/NSTEMI who undergo PCI and are of modest benefit in patients who are not routinely scheduled to undergo PCI.[1]

The management of ACS requires continuous risk assessment. Assessment of risks drives medical interventions, pinpoints patients who would benefit from coronary angiography and predict mortality. Risk is assessed by looking at presenting 12-lead ECG, troponins, initial assessments, response to anti-ischemic/anti-thrombotic therapy and by results of non-invasive tests. Non-invasive tests determine the presence or absence of ischemia in patients with low likelihood of CAD and estimate prognosis. Known high risk patients should not undergo non-invasive tests such as those patients who present with severe left ventricular dysfunction; recurrent rest angina despite intensive therapy; and those with hemodynamic compromise, previous PCI within last 6 months or prior CABG.[1]

Two theories exist to manage UA/NSTEMI early conservative and early invasive strategies. Early conservative strategies include coronary angiography and should be reserved for high risk patients and not used routinely on all patients. Experts believe invasive strategies expose patients to undo risk and cost. Patients should be assessed by response to treatments. The results of non-invasive tests and echocardiogram need to be reviewed. If the patient is determined to be high risk then they should undergo coronary angiography.[1] The early invasive theorists believe that patients should be taken to coronary angiography after stabilization unless otherwise contraindicated. The rationale for this approach is that: 1) coronary angiography provides an invasive approach to risk stratification; 2) quickly identifies 10-15% of patients with no CAD; 3) identifies the 20% of patients with 3 vessel disease that would benefit from CABG; 4) quick revascularization of the lesion decreases risk of recurrent hospitalization and the need for antianginal drugs; 5) use of LMWH and GP IIB/IIIA inhibitors has significantly improved outcomes; and 6) the ability to discharge patients sooner or to alter management strategy if non-cardiac origin is determined.[1]

CORONARY ANGIOGRAPHY AND CARE OBJECTIVES

The purpose of coronary angiography is to provide detailed information about the size and distribution of coronary vessels; the location of atherosclerotic obstruction; assessment of left ventricular function; and the suitability for revascularization. UA/NSTEMI patients appear to have better outcomes when an early conservative approach is used with ischemia guided revascularization. Lesions in this population of patients are often eccentric with irregular borders and intracoronary thrombosis. These patients often have multiple, complex lesions.[1]

The goals of coronary revascularization are to: 1) improve prognosis, 2) relieve symptoms, 3) prevent ischemic complications and 4) improve functional capacity.[1] Revascularization should not be done on patients with distal lesions on large, irreversibly damaged myocardium. Comorbidity should also be considered and used to eliminate the decision for revascularization in cases of advanced metastatic malignancy, intracranial pathology, end-stage cirrhosis and CAD known not to be amenable to revascularization. PCI with GP IIB/IIIA inhibitors added to the standard regimen of ASA, heparin and anti-ischemic medications have significantly improved the safety and success rate in patients. PCI with GPIIB/IIIA inhibitors prevent acute vessel closure and periprocedure MI.[1]

Studies done in the 1970's and early 1980's showed improved survival rates of patients with depressed left ventricular function undergoing CABG, regardless of the vessels bypassed, rather than medical therapy. All randomized trials of CABG versus medical therapy have reported improved symptom relief and functional capacity with CABG, but by ten years results begin to alternate. New studies must be done comparing CABG with PCI, stents and GP IIB/IIIA inhibitors. Current recommendations are that CABG is preferred for 1) patients with significant left main CAD, 2) patients with 3 vessel disease, 3) patients with 2 vessel disease with proximal CAD and abnormal ejection fraction less than 50%. Diabetic patients treated with insulin or hypoglycemic agents appear to have better outcomes and survival rates with CABG for multivessel disease.[10]

HOSPITAL DISCHARGE AND POST-HOSPITAL DISCHARGE CARE

Patients need to have a comprehensive discharge that includes resuming activities to an optimal level and minimizing present lifestyle risk factors for CAD. Those medications that are required to control ischemia in the hospital should be continued when the patient is discharged home for those patients that do not have revascularization, failed revascularization, or patients that have recurrent symptoms after revascularization. Medications may need to be titrated once the patient is discharged home. Medical therapy can be guided by using the following mnemonic ABCDE: aspirin and antianginals, beta blockers, cholesterol

and cigarettes, diet and diabetes, and education and exercise.[11] All patients with CAD should be given sublingual or NTG spray along with instructions on its use. Patients should be given instructions on signs and symptoms of AMI and how they should seek help.[1]

Long-term medical therapy should be implemented for those patients with CAD. The following medications are recommended to prevent death and AMI in patients with known CAD. First, ASA 75 to 325 mg per day should be prescribed unless contraindicated. In addition, clopidogrel 75 mg daily if aspirin is not tolerated and there is no contraindication for clopidogrel. ASA and clopidogrel should be prescribed for 9 months for those patients diagnosed with UA/NSTEMI. Beta blockers should be included in the medical regimen. Lipid lowering agents should be initiated and diet modification for patients with ACS who have a low-density lipoprotein (LDL) greater than 130 mg/dL or for those patients with current diet modification and a LDL greater than 100 mg/dL. ACE inhibitors should be prescribed for patients with heart failure, left ventricular dysfunction, hypertension, or diabetes.[1]

Discharge instructions should include when the patient should follow-up with their health care provider. For those patients considered low-risk who received medical treatment and those patients that were revascularized, they should follow-up in 2 to 6 weeks. Higher risk patients should follow-up within 1 to 2 weeks. Patients that experience UA who were initially treated conservatively should undergo coronary angiography to determine severity of the CAD and possible revascularization. Patients that have no anginal symptoms at follow-up should be maintained on long-term medical therapy for CAD.[1] Patients and the patient's caregiver should be given detailed instructions regarding medication type, purpose, dose, frequency, and adverse effects. If the patient's anginal symptoms change from the patient's usual pattern, the patient should be instructed to contact their health care provider for possible additional testing or treatment. Patients should also receive specific information on smoking cessation, optimal weight, daily exercise, and diet. Some patients may benefit from referral to a smoking cessation program or an outpatient cardiac rehabilitation program. Patients should receive a HMG-CoA reductase inhibitor for LDLs greater than 130 mg/dL. A fibrate or niacin should also be prescribed if the high-density lipoprotein (HDL) is less than 40 mg/dL. Hypertension

should be maintained at 130/85 mm Hg or less and tight glycemic control is also recommended in diabetic patients.[1]

SPECIAL GROUPS

There are several special groups that need to be addressed with regards to management, including: women, DM, post-CABG, the elderly, cocaine users, variant (Prinzmetal's) angina, and Syndrome X patients. Women who have been determined to have UA/NSTEMI should be managed similar to men. Women develop CAD at an older age than men and generally have more comorbidities than men. Women should receive ASA and clopidogrel along with the same non-invasive and invasive testing as men. 12-lead ECGs tend to be less predictive in women than men.[12,13,14,15] Stress echocardiography is a more useful tool in women than is thallium stress testing due to the breast tissue present in women.[1]

DM is an independent risk factor for UA/NSTEMI. Medical treatment in diabetic patients in the acute phase should be the same as those patients that are not diabetic, including stress testing and revascularization. Diabetic patients should be maintained with a tight glucose control or normoglycemia. Those patients with diabetes and multivessel disease should be considered for CABG with use of the internal mammary artery for grafting rather than PCI. Those patients post CABG that present with ischemic symptoms should be treated the same as those patients that are not post CABG. Due to the multiple causes of ischemia in patients post CABG, angiography should be considered early in UA/NSTEMI.[1]

When assessing and treating elderly patients with UA/NSTEMI several things need to be considered. First, what is the general health of the patient. The health care provider needs to determine the patient's wishes regarding medical treatment and the quality of life that the patient currently has and wishes to maintain. The health care provider must also weigh the patient's comorbidities, cognitive status, and reasonable life expectancy. Elderly patients need to have special attention to the medications prescribed with regard to therapeutic levels and adverse effects. Elderly patients may be more prone to hypotension or bleeding. If the health care provider and patient decide for intensive medical and/or interventional therapy for ACS, there must be careful follow-up assessments for adverse effects of these therapies.[1]

Those patients that experience chest discomfort, along with

ST-segment elevation or depression due to ingestion of cocaine, may be treated with NTG and oral calcium antagonists. If this patient population has persistent ST-segment elevation immediate coronary angiography should be performed with PCI if indicated. There is also a subpopulation of patients that experience ischemia related to coronary artery spasm or Printzmetal's angina. This type of angina usually occurs spontaneously with ST-segment elevation and resolves before progression to an AMI. Anginal pain is usually resolved by NTG or calcium antagonists. Coronary angiography should be performed to determine abnormal coronary anatomy or plaque formation. If the angiogram is found to be normal, patients should be treated with NTG and calcium antagonists.[1]

Finally, patients with Syndrome X are found to have angina with exercise, ST-segment depression on treadmill testing, and normal coronary arteries. The cause of this syndrome in unknown at this time. Health care providers need to give reassurance to these patients along with prescriptions for NTG, beta blockers, and calcium antagonists alone or in combination. These patients should be assessed for CAD risk factors and effective risk factor modification if needed.[1]

CONCLUSION

These guidelines are recommended by the ACC/AHA to provide a framework to treat those patients with UA/NSTEMI. Proper assessment and recognition of ischemic disease are necessary to utilize these guidelines UA/NSTEMI appropriately. At this time these guidelines are the best clinical evidence and/or expert opinion in management of UA/NSTEMI.

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