# DOWNLOAD PDF UNSTABLE ANGINA: DIAGNOSIS AND MANAGEMENT

## Chapter 1 : Diagnosis of Acute Coronary Syndrome - - American Family Physician

*Braunwald E, Mark DB, Jones RH, et al. Unstable angina: diagnosis and management Rockville, MD: Agency for Health Care Policy and Research and the National Heart, Lung, and Blood Institute, US Public Health Service, US Dept of Health and Human Services, AHCPR Publication No. ().*

Description Cardiovascular disease is the leading cause of death in the United States for men and women of all racial and ethnic groups. Angina pectoris is a clinical syndrome usually characterized by episodes or paroxysms of pain or pressure in the anterior chest. The cause is insufficient coronary blood flow, resulting in a decreased oxygen supply when there is increased myocardial demand for oxygen in response to physical exertion or emotional stress. Classification There are five 5 classifications or types of angina. The symptoms increase in frequency and severity and may not be relieved with rest or nitroglycerin. Intractable or refractory angina. There is severe incapacitating chest pain. There is pain at rest, with reversible ST-segment elevation and thought to be caused by coronary artery vasospasm. There is objective evidence of ischemia but patient reports no pain. Pathophysiology Angina is usually caused by atherosclerotic disease. Almost invariably, angina is associated with a significant obstruction of at least one major coronary artery. Oxygen demands not met. Normally, the myocardium extracts a large amount of oxygen from the coronary circulation to meet its continuous demands. When there is an increase in demand, flow through the coronary arteries needs to be increased. When there is blockage in a coronary artery, flow cannot be increased, and ischemia results which may lead to necrosis or myocardial infarction. Schematic Diagram for Angina Pectoris via Scribd. Let us support them via Patreon to make more informative videos like this. Causes Several factors are associated with angina. This can precipitate an attack by increasing myocardial oxygen demand. This can cause vasoconstriction and elevated blood pressure , with increased oxygen demand. Eating a heavy meal. A heavy meal increases the blood flow to the mesenteric area for digestion, thereby reducing the blood supply available to the heart muscle ; in a severely compromised heart, shunting of the blood for digestion can be sufficient to induce anginal pain. Stress causes the release of catecholamines, which increased blood pressure, heart rate, and myocardial workload. Clinical Manifestations The severity of symptoms of angina is based on the magnitude of the precipitating activity and its effect on activities of daily living. The pain is often felt deep in the chest behind the sternum and may radiate to the neck, jaw, and shoulders. A feeling of weakness or numbness in the arms, wrists and hands. An increase in oxygen demand could cause shortness of breath. Inadequate blood supply to peripheral tissues cause pallor. The elderly person with angina may not exhibit the typical pain profile because of the diminished responses of neurotransmitters that occur with aging. Often, the presenting symptom in the elderly is dyspnea. Elderly patients should be encouraged to recognize their chest painâ€”like symptom eg, weakness as an indication that they should rest or take prescribed medications. Myocardial infarction is the end result of angina pectoris if left untreated. The heart pumps more and more blood to compensate the decreased oxygen supply, and. MI also predisposes the patient to cardiogenic shock. Assessment and Diagnostic Findings The diagnosis of angina pectoris is determined through: Often normal when patient at rest or when pain-free; depression of the ST segment or T wave inversion signifies ischemia. Dysrhythmias and heart block may also be present. Significant Q waves are consistent with a prior MI. Done to see whether pain episodes correlate with or change during exercise or activity. ST depression without pain is highly indicative of ischemia. Exercise or pharmacological stress electrocardiography: Provides more diagnostic information, such as duration and level of activity attained before onset of angina. A markedly positive test is indicative of severe CAD. Studies have shown stress echo studies to be more accurate in some groups than exercise stress testing alone. Usually within normal limits WNL ; elevation indicates myocardial damage. Usually normal; however, infiltrates may be present, reflecting cardiac decompensation or pulmonary complications. Pco2, potassium , and myocardial lactate: May be elevated during anginal attack all play a role in myocardial ischemia and may perpetuate it. May be elevated CAD risk factor. May reveal abnormal valvular action as cause of chest pain. Nuclear imaging studies rest or stress scan: Ischemic regions appear as areas of decreased thallium uptake. Evaluates specific and general ventricle performance, regional wall

motion, and ejection fraction. Cardiac catheterization with angiography: Definitive test for CAD in patients with known ischemic disease with angina or incapacitating chest pain, in patients with cholesterolemia and familial heart disease who are experiencing chest pain, and in patients with abnormal resting ECGs. Abnormal results are present in valvular disease, altered contractility, ventricular failure, and circulatory abnormalities. Ten percent of patients with unstable angina have normal-appearing coronary arteries. On occasion, may be used for patients who have angina at rest to demonstrate hyperspastic coronary vessels. Some patients may also have severe ventricular dysrhythmias. Medical Management The objectives of the medical management of angina are to increase the oxygen demand of the myocardium and to increase the oxygen supply. Oxygen therapy is usually initiated at the onset of chest pain in an attempt to increase the amount of oxygen delivered to the myocardium and reduce pain. Beta-blockers reduces myocardial oxygen consumption by blocking beta-adrenergic stimulation of the heart. Calcium channel blockers have negative inotropic effects. Antiplatelet medications prevent platelet aggregation; and anticoagulants prevent thrombus formation. Nursing Management The patient with angina pectoris should be managed by a cardiac nurse specifically. Nursing Assessment In assessing the patient with angina, the nurse may ask regarding the following:

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*This clinical practice guideline was developed by a member panel that included physicians, cardiovascular nurse specialists, a public health representative, & a consumer representative.*

Information from references 16 through  This widely available marker has low sensitivity and specificity for cardiac damage. Furthermore, CK levels may be elevated in a number of noncardiac conditions, including trauma, seizures, renal insufficiency, hyperthermia, and hyperthyroidism. The serum CK level rises within three to eight hours after myocardial injury, peaks by 12 to 24 hours, and returns to baseline within three to four days. Although CK commonly was measured serially along with CK-MB at the time of hospital admission and six to 12 hours after admission, this marker largely has been replaced by cardiac troponins and CK-MB. The CK-MB subform assay takes about 25 minutes to perform. However, the CK-MB subform assay is not yet widely available. Unlike troponin I levels, troponin T levels may be elevated in patients with renal disease, polymyositis, or dermatomyositis. The cardiac troponins typically are measured at emergency department admission and repeated in six to 12 hours. The cardiac troponins may remain elevated up to two weeks after symptom onset, which makes them useful as late markers of recent acute myocardial infarction. The troponins also can help identify low-risk patients who may be sent home with close follow-up. Bedside troponin assays are being developed. It can be detected in the serum as early as two hours after myocardial necrosis begins. Myoglobin has low cardiac specificity but high sensitivity, which makes it most useful for ruling out myocardial infarction if the level is normal in the first four to eight hours after the onset of symptoms. Combining a doubling of the baseline myoglobin level at two hours after symptom onset with an abnormal myoglobin test at six hours after symptom onset increases the sensitivity to 95 percent at six hours. A Practical Plan of Action No assessment protocol or constellation of tests is totally accurate in diagnosing acute coronary syndrome. From 1 to 4 percent of patients ultimately proven to have acute coronary syndrome are sent home from the emergency department. When a patient presents with chest pain or symptoms suggestive of acute coronary syndrome, vital signs should be obtained, the patient should be monitored, and a focused but careful history should be obtained. A lead ECG should be obtained within 10 minutes of presentation. Suggested approach to the evaluation of patients with chest pain or symptoms suggestive of ACS. Risk stratification then should be performed using the criteria in Table 1. Use of this instrument in an emergency department resulted in no change in appropriate admission of patients who had acute coronary syndrome. The benefit of its use was a significant reduction in hospital admissions of patients who did not have acute coronary syndrome. Patients who are at high risk for acute coronary syndrome should be admitted to a coronary care unit. Patients at intermediate risk may be monitored in a telemetry bed in an inpatient setting or a chest pain unit. A chest pain unit is a specialized unit within an emergency department or a medical center; the unit is dedicated to careful monitoring and aggressive implementation of diagnostic protocols clinical guidelines for the evaluation of acute coronary syndrome. Most low-risk patients may undergo early exercise testing or can be discharged with careful outpatient follow-up. Although protocols for chest pain units may vary somewhat, one protocol 28 that has been shown to be safe and cost-effective in an intermediate-risk population consists of the following: Event monitoring and continuous ST-segment monitoring; 2. Four patients staffed by one full-time nurse; 4. Admission to the cardiac care unit or a telemetry bed on the cardiology service for patients with elevated cardiac enzyme levels, recurrent chest pain consistent with unstable angina, or significant ventricular arrhythmias; 5. An exercise treadmill test for patients without abnormal findings on the initial tests, or a nuclear stress test or echocardiographic stress test; 6. Admission of patients with an equivocal or positive result. Use of this type of systematic approach has the potential to improve the ability of physicians to care for patients with possible acute coronary syndrome, as well as reduce the likelihood of medical error. In the future, advanced diagnostic modalities, such as myocardial perfusion imaging, may have a role in reducing unnecessary hospitalizations. Read the full article. Get immediate access, anytime, anywhere. Choose a single article, issue, or full-access subscription. Earn up to 6 CME credits per issue.

## Chapter 3 : Unstable angina - Wikipedia

*ACC/AHA Guidelines for the Management of Patients With Unstable Angina and Non-ST-Segment Elevation Myocardial Infarction: Executive Summary and Recommendations A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Unstable Angina).*

Immediate access to this article To see the full article, log in or purchase access. After graduating from Harvard Medical School, Dr. He also completed a cardiology fellowship at Johns Hopkins Hospital, Baltimore Address correspondence to Stephen D. Reprints are not available from the authors. The authors indicate that they do not have any conflicts of interest. This article is one in a series developed in collaboration with the American Heart Association. Guest editor of the series is Sidney C. J Am Coll Cardiol. Accessed online May 11, , at: Wiviott SD, Braunwald E. Unstable angina and non-ST-segment-elevation myocardial infarction: Coronary revascularization, hospital discharge, and post-hospital care. Predictors of outcome in patients with acute coronary syndromes without persistent ST-segment elevation. Results from an international trial of patients. An integrated clinical approach to predicting the benefit of tirofiban in non-ST elevation acute coronary syndromes. N Engl J Med. The prognostic value of C-reactive protein and serum amyloid a protein in severe unstable angina. C-reactive protein is a potent predictor of mortality independently of and in combination with troponin T in acute coronary syndromes: Thrombolysis in Myocardial Infarction. Markers of myocardial damage and inflammation in relation to long-term mortality in unstable coronary artery disease. Fragmin during Instability in Coronary Artery Disease. The prognostic value of B-type natriuretic peptide in patients with acute coronary syndromes. Multimarker approach to risk stratification in non-ST elevation acute coronary syndromes: Aspirin, heparin, or both to treat acute unstable angina. Thienopyridine derivatives ticlopidine, clopidogrel versus aspirin for preventing stroke and other serious vascular events in high vascular risk patients. Cochrane Database Syst Rev ; 2: Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation [published corrections appear in N Engl J Med ; Effects of pretreatment with clopidogrel and aspirin followed by long-term therapy in patients undergoing percutaneous coronary intervention: Troponin concentrations for stratification of patients with acute coronary syndromes in relation to therapeutic efficacy of tirofiban. Invasive versus conservative strategies in unstable angina and non-Q-wave myocardial infarction following treatment with tirofiban: Bosch X, Marrugat J. Adding heparin to aspirin reduces the incidence of myocardial infarction and death in patients with unstable angina. Low molecular weight heparins versus unfractionated heparin for acute coronary syndromes. Combination therapy with tirofiban and enoxaparin in acute coronary syndromes. QTc interval and B-type natriuretic peptide levels predict death in patients with advanced heart failure. Percutaneous coronary intervention after subcutaneous enoxaparin pretreatment in patients with unstable angina pectoris. Enoxaparin and abciximab adjunctive pharmacotherapy during percutaneous coronary intervention. Thrombolysis in patients with unstable angina improves the angiographic but not the clinical outcome. Randomized trial of thrombolysis versus heparin in unstable angina. Effects of tissue plasminogen activator and a comparison of early invasive and conservative strategies in unstable angina andnon-Q-wave myocardial infarction. Thrombolysis in Myocardial Ischemia.

## Chapter 4 : management of unstable angina/non STEMI - General Practice Notebook

*Unstable angina (UA) is one of the acute coronary syndromes, a group of conditions that also includes non-ST elevation myocardial infarction (MI) and ST elevation MI. The underlying pathogenic substrate of all these entities is the unstable coronary plaque with an overlying intracoronary thrombus.*

It is important to remember that the CCS class assigned is the maximum limitation and is not fixed: Functional studies have also shown a weak correlation with objective measures of exercise performance. Classification of angina severity according to the Canadian Cardiovascular Society Class I Ordinary activity such as walking or climbing stairs does not cause angina. Angina with strenuous or rapid or prolonged exertion at work or recreation. Class II Slight limitation of ordinary activity. Angina on walking or climbing stairs rapidly; walking or stair climbing after meals; in cold wind; under emotional stress; or during the first few hours after awakening. Walking more than two blocks on the level or climbing more than one flight of ordinary stairs at a normal pace and in normal conditions. Class III Marked limitation of ordinary physical activity. Angina on walking one to two blocks on the level, or one flight of stairs in normal conditions and at a normal pace. Class IV Open in a separate window After obtaining a detailed description of chest pain, the presence of risk factors and co-morbid conditions should be determined. Hence, the clinician should identify conventional risk factors for the development of CAD such as the presence of hypertension, dyslipidemia, cigarette smoking, diabetes or impaired glucose tolerance, obesity and sedentary lifestyle. It is also important to reliably identify co-morbid conditions such as chronic heart failure HF , cerebrovascular disease, peripheral vascular disease, or chronic kidney disease, as these conditions may have an adverse influence on prognosis, presumably through their effect on the progression of atherosclerosis. The physical examination is often normal in patients with SIHD and is generally not useful for confirming the diagnosis. Nonetheless, a focused physical examination is important during the initial evaluation of patients. A focused physical examination may exclude other conditions associated with angina such as anemia, hypertension, valvular heart disease, hypertrophic obstructive cardiomyopathy or arrhythmias. One should search for evidence of non-coronary vascular disease such as auscultation for carotid or femoral bruit; palpation of an abdominal aneurysm; and detection of diminished peripheral pulses. Signs of co-morbid conditions such as thyroid disease, renal disease, hypertension or diabetes should be identified. The association between these findings and high-risk patients are well documented and portend an adverse CV prognosis. Other key physical examination findings are: A cardiac examination during or immediately after an episode of myocardial ischemia may reveal a displaced ventricular impulse; third or fourth heart sounds; and a transient apical systolic murmur of mitral insufficiency due to reversible papillary muscle dysfunction. These findings are more prevalent in patients with extensive CAD and severe left ventricular LV systolic dysfunction and may likewise indicate an adverse prognosis. However, these findings are not specific for SIHD. Lastly, it is also recommended to obtain BMI, waist circumference and waist-to-hip ratio to assist in evaluation of Metabolic Syndrome and weight management for lifestyle modification. This baseline ECG will be useful for comparison in future situations. A normal resting ECG is seen in approximately half of patients with SIHD, even in patients with severe angina, and does not exclude the diagnosis of ischemia. In addition, a normal ECG suggests the presence of normal resting LV function and a more favorable long-term prognosis. The occurrence of ST-T wave abnormalities and Q waves may correlate with the severity of underlying disease in patients with known CAD and worsens prognosis. Abnormal Q waves are specific but insensitive indicators of previous myocardial infarction MI. Other conditions that can produce ST-T wave abnormalities include LV hypertrophy LVH and dilation; conduction disturbances; electrolyte abnormalities; neurogenic effects; and anti-arrhythmic drugs. The resting ECG may also show these mentioned abnormalities such as LVH; conduction disturbances such as left bundle branch block LBBB or left anterior fascicular block; arrhythmias such as atrial fibrillation AF or ventricular premature beats; and even pre-excitation. Although these abnormalities may have low sensitivity and specificity for CAD, these findings suggest a poor prognosis since they are often associated with multi-vessel disease and impairment of LV function. A resting lead ECG should also be recorded during or

immediately after an episode of chest pain to allow detection of ST-segment changes in the presence of ischemia. Such information may then lead to appropriate investigations and treatment decisions. In patients with vasospastic angina, an ECG during or immediately after an episode of chest pain can be diagnostic because the ST-segment shifts are usually reversible once coronary vasospasm is relieved with nitrates. Knowledge of lipid and glucose level sis important because of the well-recognized association between elevated levels and adverse CV outcomes. The lipid profile and glycemic status should be re-assessed periodically to detect development of dyslipidemia and diabetes, respectively, and to determine efficacy of treatment if initiated. There are no recommendations from foreign guidelines as to the frequency of repeat measurements, but expert consensus suggests annual measurement. However, patients with initial high levels of lipids or glucose should have more frequent measurements to determine efficacy of treatment. The CBC provides information related to possible cause of ischemia e. Thyroid hormone levels should also be measured when there is a clinical suspicion of a thyroid disorder. Routine measurement of baseline liver function tests early after beginning statin therapy is recommended as some patients develop elevations in liver enzymes with intensive statin therapy. The usefulness of the chest x-ray as a routine test in patients with SIHD is not well established even though a chest x-ray is frequently used in the assessment of patients with chest pain. The chest x-ray does not provide specific information for diagnosis or event risk stratification. However, it should be considered in the above conditions to be able to rule out these atypical causes of chest pain. Echocardiography is now recommended in the initial evaluation of all patients with symptoms suggestive of SIHD. In the first version of the PHA guidelines, echocardiography is recommended only in patients with clinically detected murmurs, history and ECG changes of prior MI, and signs or symptoms of HF. There are several reasons why echocardiography should be performed in all patients with a first presentation of suspected SIHD. First of all, disorders such as aortic stenosis or hypertrophic obstructive cardiomyopathy can be ruled out as alternative causes of angina. Secondly, regional wall motion abnormalities may be detected, which increase the likelihood of CAD. The segmental wall motion abnormalities seen with ischemia or infarction correspond closely with the coronary artery blood supply to the myocardium. Resting two-dimensional and Doppler transthoracic echocardiography also provide information on global ventricular function, an important prognostic parameter in patients with SIHD. The severity of diastolic dysfunction also correlates with the prognosis in recent published studies and a restrictive filling pattern has points to adverse consequences. Tissue Doppler imaging and strain rate measurements are additional methods for detecting heart failure with preserved LV ejection fraction in patients who complain of shortness of breath as angina equivalent. For all these reasons, echocardiography is now recommended as a necessary examination in the initial evaluation of all patients with suspected SIHD. Ambulatory ECG Holter monitoring has a specific role in detecting arrhythmias and suspected vasospastic angina ST segment elevation during acute ischemia and is recommended in these subgroups of patients. There is, however, no good evidence to support its routine use as a diagnostic or prognostic tool over that provided by the exercise ECG and as tress imaging study.

## Chapter 5 : Acute coronary syndrome, Myocardial Infarction, Unstable Angina - Cancer Therapy Advisor

*The focus of this chapter is the diagnosis and management of patients with Non ST Elevation Myocardial Infarction (NSTEMI) and unstable angina (UA), which are collectively referred to as NSTE-ACS (Non ST Elevation Acute Coronary Syndromes). This chapter deals with the pathophysiology, definition, criteria and management of patients with NSTEMI.*

In the absence of classic ECG changes, cardiac biomarker elevation can identify patients with myocardial necrosis. Differential diagnosis Conditions that can be confused with ACS might include pericarditis, aortic dissection, pneumonia, pneumothorax, acute pulmonary embolism, gastroesophageal reflux, esophageal spasm, and musculoskeletal injury, to name a few. Confirmatory tests Obtaining a lead ECG at presentation, serially, and with each episode of chest pain is vital. Given that traditional biomarkers tend to rise hours following infarction, and peak at about 24 hours, laboratory evaluation of biomarker elevation should continue for at least 9 hours after presentation. Specific Treatment All patients with presumed ACS should receive an aspirin, mg to mg, chewed for rapid buccal absorption. All patients should get supplemental oxygen. Provided patients do not have evidence of RV infarction and hence are not preload-sensitive , sublingual and intravenous nitrates nitroglycerin can be administered for symptom relief. Heparin - either unfractionated or low molecular weight - should be considered as an antithrombotic strategy for all ACS patients. The risk of needing surgical revascularization i. Clopidogrel should also be administered to all ACS patients who have an aspirin allergy. Statins, particularly high-dose 80mg atorvastatin, improve short- and long-term outcomes for patients with ACS. If percutaneous intervention cannot successfully restore blood flow to injured myocardium, surgical revascularization should be considered. Disease monitoring, follow-up and disposition Expected response to treatment Given the underlying pathophysiology contributing to the development of acute coronary syndromes - particularly those pathways involving enhanced platelet activation and aggregation, along with thrombosis - the use of antiplatelet and antithrombotic therapies should decrease atherothrombotic sequelae. As a result, coronary reperfusion and myocardial oxygen delivery should be enhanced. This, along with appropriate revascularization strategies i. PCI or fibrinolysis should decrease myocardial necrosis and alleviate the symptoms associated with ACS. Preservation of myocardial contractile function is paramount, and left ventricular ejection fraction should be monitored with short-term and long-term echocardiographic evaluation. In addition, vasodilators should improve coronary perfusion and reduce ischemic symptoms when given to patients with ACS. Beta blockers decrease the sympathetic stimulation that contributes to myocardial oxygen supply-demand mismatch. These agents may improve symptoms, but also have been shown to improve both short- and longer-term mortality. In the absence of acute ST-segment elevations on the presenting ECG, the clinician often has time for further evaluation and consideration of alternative diagnoses. Normal cardiac biomarkers are certainly reassuring, and rule out the presence of true myocardial infarction. If an invasive management approach is pursued i. Follow-up All patients should follow up with a cardiologist after hospital discharge. Careful evaluation and management of cardiovascular risk factors i. Beta blockers, statins, and ACE inhibitors particularly for patients with systolic dysfunction should be carefully titrated. The appropriate dosage and duration of antiplatelet therapies should be considered in the outpatient arena - therapies that depend a lot on the type of ACS presentation, the type of PCI performed i. In patients with left ventricular contractile dysfunction, follow-up imaging with echocardiography is usually appropriate to monitor for progression or improvement. Pathophysiology The majority of acute coronary syndromes result from disruption of a vulnerable atherosclerotic plaque within an epicardial coronary artery. Exposure of constituents of these lipid-rich plaques, along with the exposed coronary endothelium, to circulating blood promotes a rapid and robust thrombotic process that primarily involves platelet activation and aggregation. In other cases, there may be transient complete occlusion that improves via the effects of endogenous fibrinolysis. The pathophysiology is similar in patients with UA; the difference is that in NSTEMI, myocardial necrosis occurs and cardiac biomarkers are elevated, while in UA true necrosis and infarction does not occur. In true myocardial infarction, irreversible cell injury begins to occur 20 or more minutes following cessation of myocardial perfusion. The benefit of reperfusion strategies

dramatically diminishes with time and, in the absence of ongoing symptoms or shock, percutaneous intervention is unlikely to provide significant improvements in contractile function, morbidity, or mortality. The total process of ventricular remodeling, fibrosis, and scarring takes several weeks to complete. Epidemiology There are a number of risk factors for ACS. These include advanced age, diabetes mellitus, hypertension, hyperlipidemia, tobacco abuse, family history, and male sex. Prognosis Patients who present to the hospital with an ACS have substantial morbidity and mortality. If contractile dysfunction results from an ACS presentation, morbidity and mortality are significantly increased. Patients should be monitored for potential mechanical complications of their ACS. These include congestive heart failure, cardiogenic shock, acute mitral valve insufficiency due to papillary muscle ischemia, infarction, or rupture , ventricular free wall rupture, ventricular septal rupture, left ventricular aneurysm or pseudoaneurysm, and cardiac tamponade. In addition, patients with ACS are at risk for the development of both atrial and ventricular arrhythmias. Episodes of ventricular arrhythmias that develop within the first 48 hours of presentation are more commonly related to transient electrical instability and are usually not chronic problems. Conduction disease can also occur following ACS presentation, particularly in patients with acute myocardial infarction. Often conduction blocks are transient and related to acute ischemia; however, advanced conduction disease e. Special considerations for nursing and allied health professionals. No sponsor or advertiser has participated in, approved or paid for the content provided by Decision Support in Medicine LLC.

## Chapter 6 : Diagnosis and Management of Acute Coronary Syndrome: An Evidence-Based Update

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The diagnosis and classification of ACS is based on a thorough review of clinical features, including electrocardiogram ECG findings and biochemical markers of myocardial necrosis. In this article, we review the topic of ACS with particular emphasis on initial management and use of the newer medications. Specific coronary interventions performed by the cardiologist eg, stents or balloon angioplasty are beyond the scope of this review. Previous Section Next Section Scope of the Problem Coronary heart disease CHD is responsible for more than half of all cardiovascular events in individuals less than 75 years of age. The prevalence of CHD is estimated to be 6. During the past several years, the rates of hospitalization for MI and mortality associated with CHD have decreased. A diagnosis of MI was responsible for approximately , deaths in the US in , and ACS was associated with an estimated , hospital discharges in  Diagnosis Clinical Presentation A diagnosis of ACS should be considered in all patients presenting with ischemic symptoms. Clinical signs and symptoms of ischemia include various combinations of chest pain, upper extremity, mandibular or epigastric discomfort, dyspnea, diaphoresis, nausea, fatigue, or syncope. The pain and discomfort associated with an ACS event may occur with exertion or at rest and is often diffuse rather than localized. Atypical symptoms of ACS may occur in certain patient populations such as women, the elderly, diabetics, or postoperatively. In these situations, ACS may be associated with palpitations, cardiac arrest, or with an asymptomatic clinical presentation. For patients who do not have these factors, consideration should be given to an alternative disease process. Other clinical conditions, such as pericarditis, dissecting aortic aneurysm, and mitral valve prolapse represent nonischemic, cardiac causes of myocardial injury and thus do not fall within the definition of ACS. In addition, there are several noncardiac conditions that may manifest with similar symptoms of ACS, including musculoskeletal pain, esophageal discomfort, pulmonary embolism, or anxiety. Elevated cardiac biomarkers in and of themselves do not indicate the underlying mechanism of injury and do not differentiate between ischemic or nonischemic causes. An elevation in troponin concentration is based on specific assays and is defined as a value exceeding the 99th percentile of a normal reference population. At this level, sensitive cardiac troponin I assays have a positive likelihood ratio LR of 11â€”14 and a negative LR of 0. Troponin levels should be measured on first assessment, within 6 hours of the onset of pain, and in the 6â€”12 hour time frame after onset of pain, due to the delayed increase in circulating levels of cardiac biomarkers strength of recommendation A. In addition, it is important to understand that elevations in troponin may be seen for up to 2 weeks after the onset of myocardial necrosis. If troponin concentrations are unavailable, then CKMB should be measured. A meticulous evaluation of ECG changes can assist in estimating time of the event, amount of myocardium at risk, patient prognosis, and appropriate therapeutic strategies. In addition, measurement of B-type natriuretic peptide may be considered to assist in predicting risk of morbidity and mortality in patients with suspected ACS. Early risk stratification can assist in determining whether a patient should be managed with either an early invasive strategy or an initial conservative strategy and can help determine the pharmacologic therapies that are recommended Figure 1.

## Chapter 7 : Unstable angina: MedlinePlus Medical Encyclopedia

*NICE's guidelines on unstable angina and NSTEMI: early management (CG94), myocardial infarction: cardiac rehabilitation and prevention of further cardiovascular disease (CG) and myocardial infarction with ST-segment elevation: acute management (CG) are being combined and updated.*

Symptoms Symptoms of angina may include: Chest pain that you may also feel in the shoulder, arm, jaw, neck, back, or other area Discomfort that feels like tightness, squeezing, crushing, burning, choking, or aching Discomfort that occurs at rest and does not easily go away when you take medicine Shortness of breath Sweating With stable angina, the chest pain or other symptoms only occur with a certain amount of activity or stress. The pain does not occur more often or get worse over time. Unstable angina is chest pain that is sudden and often gets worse over a short period of time. You may be developing unstable angina if the chest pain: Starts to feel different, is more severe, comes more often, or occurs with less activity or while you are at rest Lasts longer than 15 to 20 minutes Occurs without cause for example, while you are asleep or sitting quietly Does not respond well to a medicine called nitroglycerin especially if this medicine worked to relieve chest pain in the past Occurs with a drop in blood pressure or shortness of breath Unstable angina is a warning sign that a heart attack may happen soon and needs to be treated right away. See your health care provider if you have any type of chest pain. Exams and Tests The provider will do a physical exam and check your blood pressure. The provider may hear abnormal sounds, such as a heart murmur or irregular heartbeat, when listening to your chest with a stethoscope. Tests for angina include: Blood tests to show if you have heart tissue damage or are at a high risk for heart attack, including troponin I and T, creatine phosphokinase CPK , and myoglobin. Stress tests, such as exercise tolerance test stress test or treadmill test , nuclear stress test , or stress echocardiogram. This test involves taking pictures of the heart arteries using x-rays and dye. It is the most direct test to diagnose heart artery narrowing and find clots. Treatment You may need to check into the hospital to get some rest, have more tests, and prevent complications. Blood thinners antiplatelet drugs are used to treat and prevent unstable angina. You will receive these drugs as soon as possible if you can take them safely. Medicines include aspirin and the prescription drug clopidogrel or something similar ticagrelor, prasugrel. These medicines may be able to reduce the chance of a heart attack or the severity of a heart attack that occurs. During an unstable angina event: You may get heparin or another blood thinner and nitroglycerin under the tongue or through an IV. Other treatments may include medicines to control blood pressure, anxiety, abnormal heart rhythms , and cholesterol such as a statin drug. A procedure called angioplasty and stenting can often be done to open a blocked or narrowed artery. Angioplasty is a procedure to open narrowed or blocked blood vessels that supply blood to the heart. A coronary artery stent is a small, metal mesh tube that opens up expands inside a coronary artery. A stent is often placed after angioplasty. It helps prevent the artery from closing up again. A drug-eluting stent has medicine in it that helps prevent the artery from closing over time. Heart bypass surgery may be done for some people. The decision to have this surgery depends on: Which arteries are blocked How many arteries are involved Which parts of the coronary arteries are narrowed How severe the narrowings are Outlook Prognosis Unstable angina is a sign of more severe heart disease. How well you do depends on many different things, including: How many and which arteries in your heart are blocked, and how severe the blockage is If you have ever had a heart attack How well your heart muscle is able to pump blood out to your body Abnormal heart rhythms and heart attacks can cause sudden death. Possible Complications Unstable angina may lead to: Abnormal heart rhythms arrhythmias Heart failure When to Contact a Medical Professional Seek medical attention if you have new, unexplained chest pain or pressure. If you have had angina before, call your provider. Call if your angina pain: Is not better 5 minutes after you take nitroglycerin your provider may tell you to take 3 total doses Does not go away after 3 doses of nitroglycerin Is getting worse Returns after the nitroglycerin helped at first Call your provider if: You are having angina symptoms more often You are having angina when you are sitting rest angina You are feeling tired more often You are feeling faint or lightheaded, or you pass out Your heart is beating very slowly less than 60 beats a minute or very fast more than beats a minute , or it is not steady You are having trouble taking

your heart medicines You have any other unusual symptoms If you think you are having a heart attack, get medical treatment right away. Prevention Some studies have shown that making a few lifestyle changes can prevent blockages from getting worse and may actually improve them. Lifestyle changes can also help prevent some angina attacks. Your provider may tell you to: Lose weight if you are overweight Stop smoking Exercise regularly Drink alcohol in moderation only Eat a healthy diet that is high in vegetables, fruits, whole grains, fish, and lean meats Your provider will also recommend that you keep other health conditions such as high blood pressure, diabetes, and high cholesterol levels under control. If you have one or more risk factors for heart disease, talk to your provider about taking aspirin or other medicines to help prevent a heart attack. Aspirin therapy 75 to mg a day or drugs such as clopidogrel, ticagrelor or prasugrel may help prevent heart attacks in some people. Aspirin and other blood thinning therapies are recommended if the benefit is likely to outweigh the risk of side effects. Alternative Names Accelerating angina; New-onset angina; Angina - unstable; Progressive angina; CAD - unstable angina; Coronary artery disease - unstable angina; Heart disease - unstable angina; Chest pain - unstable angina Patient Instructions.

## Chapter 8 : Unstable angina: assessment and management | Medicine Today

*Each year, more than 1 million patients are admitted to U.S. hospitals because of unstable angina and non-ST-segment elevation myocardial infarction (UA/NSTEMI).*

This chapter deals with the pathophysiology, definition, criteria and management of patients with NSTEMI and unstable angina. STEMI, on the other hand, is caused by a complete coronary artery occlusion, which results in complete stop of blood flow and thus more extensive myocardial ischemia referred to as transmural ischemia. As in patients with STEMI, those with NSTEMI and unstable angina are at considerable risk of developing life-threatening ventricular arrhythmias ventricular tachycardia , ventricular fibrillation and subsequently cardiac arrest. Although ventricular arrhythmias may occur any time after coronary artery occlusion, the vast majority occur within the first hour s. This underlines the importance of prompt diagnosis and intervention. NSTEMI is by definition an acute myocardial infarction, whereas unstable angina is not an infarction. Unstable angina is only diagnosed if there are no evidence of myocardial infarction necrosis. However, unstable angina is considered an acute coronary syndrome because it is an imminent precursor to myocardial infarction. The size, location and duration of the occlusion is of prime importance but additional factors may also influence the infarction process, which is normally completed between 2 and 12 hours after symptom onset. The continuous loss of myocardium and the electrical instability calls for prompt diagnosis and treatment. Therefore, most communities have developed a regional system of care which includes the dispatch center, ambulance, emergency department, catheterization laboratory and cardiology ward. Most patients undergo coronary angiography within 48 hours or earlier if the patient is at high risk of death or other complications. Low-risk patients may be evaluated after 48â€”72 hours. Management is discussed in detail below. An acute coronary syndrome is caused by an abrupt reduction in coronary blood flow. The reduction in coronary blood flow is due to atherothrombosis, which occurs when an atherosclerotic lesion disrupts. Atherotrombosis obstructs coronary blood flow and causes ischemia in the myocardium supplied by the artery. Figure 1 shows the process from atherothrombosis to classification of acute coronary syndromes. Disruption of atherosclerotic lesions result in atherothrombosis which causes abrupt reduction in coronary blood flow. As discussed previously, ischemia results in ECG changes. In fact, the type of ischemia will determine which type of ECG changes that occur. Hence, acute coronary syndromes can be classified according to the ECG. The classification is based solely on the presence of ST segment elevations. For the sake of clarity: Cases who do not display elevated troponins are classified as unstable angina UA. Elevated troponins with a pattern consistent with myocardial necrosis; refer to Diagnosis of Acute Myocardial Infarction is evidence for myocardial infarction i. Figure 2 presents the natural history and classification of acute coronary syndromes. This is explained by increased use of revascularization percutaneous coronary intervention or fibrinolysis , advances in anticoagulants and antiplatelet agents, as well as aggressive primary preventive strategies using statins, blood pressure lowering drugs and antidiabetic drugs. Reduced smoking rates have certainly also contributed to the observed trends. In it was possible to detect myocardial infarctions times smaller than what was possible in  It is expected that the proportion classified as unstable angina will continue to decline as troponin assays become more sensitive. Despite the advances in management and detection of NSTEMI and unstable angina, these conditions cause considerable mortality and morbiditiy worldwide. NSTEMI and unstable angina are caused by partial incomplete occlusions The severity of acute coronary syndromes depends mainly on the location, size and duration of the occlusion. Considering the location, proximal occlusions are more severe than distal occlusions, simply because a proximal occlusion will affect more artery branches and therefore more myocardium. With respect to the size of the occlusion, it is obvious that a total complete occlusion will be more devastating than a partial incomplete occlusion. Refer to Figures 1, 2 and 3. The ischemia will affect the subendocardium which has poorest prerequisites in case of ischemia. The subendocardium is too far away from the blood in the ventricular cavity and the oxygen level in the coronary artery is reduced because oxygen has been extracted during the passage through the myocardium. Figure 4 summarizes the complications. Importantly, leads which display ST depressions do not necessarily reflect the

ischemic area. This implies that ST depressions in leads V3â€"V4 are not necessarily due to anterior wall ischemia. Ischemic ST depressions are characterized by a horizontal or downsloping ST segment. North American and European guidelines assert that the ST segment must be either downsloping or horizontal, otherwise ischemia is unlikely to be the cause Figure 5. Characteristics of ischemic ST segment depressions. These leads must have evident R-waves, or R-waves larger than S-waves. However, in some instances the subendocardial injury may be extensive in patients with NSTEMI, which may result in pathological Q-waves. Note that unstable angina does not result in any QRS changes because the condition does not lead to myocardial necrosis infarction. It is unusual, however, to display a normal ECG throughout the course. The patient should preferably be transported by the emergency medical services EMS. Studies have demonstrated several benefits of utilizing the EMS, such as reduced delay to evidence-based therapies and reduced delay to be seen by ED physician. Moreover, studies also demonstrate that patients who use the EMS have higher mortality and morbidity, as compared with the overall population with acute coronary syndromes. This is explained by the fact that patients who use the EMS have more comorbidities, higher prevalence of cardiovascular disease and are generally older. The EMS should immediately assess vital functions and address hemodynamic, electrical and respiratory instability. If the patient is hemodynamically stable, a brief history focus on coronary risk factors and current symptoms and risk stratification should be performed. Evidence-based therapies can be started already in the ambulance. Oxygen, morphine, nitrates and aspirin are safe and effective to administer en route to hospital. A defibrillator must always be ready and venous access should be established. Vital parameters include heart rate, heart rhythm, blood pressure, respiratory rate, oxygen saturation and consciousness. Prehospital personnel have proven to be highly capable of recognizing ischemic ECG changes and they may transmit ECGs electronically to the hospital for further evaluation. Although prehospital troponin analysis is available, it is generally discouraged since it does not improve survival. Emergency department ED The same clinical parameters must be assessed in the emergency department ED. Assessment of complications e. New lead ECGs are performed and serial recordings are acquired if appropriate. Guidelines state that a lead ECG should be evaluated within 10 minutes after the patients arrival in the ED. Continuous monitoring with lead ECG ST segment monitoring increases detection of ischemia, but such equipment is frequently unavailable. Cardiac troponin I or T levels are obtained at presentation and 3 to 6 hours after symptom onset. Rising or falling values, with at least one value above the upper normal limit is evidence for acute myocardial infarction. Note that normal troponins do not rule out myocardial infarction until 6 hours after the latest episode of symptoms it may require 6 hours for troponins to increase following myocardial necrosis. Patients without objective evidence of myocardial ischemia i. Guidelines recommend that patients without objective evidence of ischemia should undergo exercise stress testing once their condition has stabilized. Stress myocardial perfusion imaging, stress echocardiography or CTCA computerized tomography of coronary arteries are more expensive alternatives but offers greater sensitivity and specificity. Management in the emergency department ED Anti-ischemic and anti-thrombotic agents should be given without delay if the suspicion of NSTEMI or unstable angina is strong, provided that there are no contraindications. Among the potentially life-threatening differential diagnoses, aortic dissection is the most important one since it is a contraindication to several agents used in acute coronary syndromes. Management must, however, be individualized with respect to coronary angiography PCI. The majority of patients should undergo angiography within 24 hours, but high risk patients should be evaluated with angiography earlier. Guidelines recommend the use of validated risk models to estimate the risk of acute myocardial infarction, days and 1-year mortality. The higher the estimated risk, the earlier should angiography be performed. As always in patients with acute coronary syndromes, non-steroidal anti-inflammatory drugs NSAID should be withheld during the acute phase. NSAIDs except from aspirin increases mortality in patients with acute coronary syndromes. There is no evidence that oxygen confers any benefit. Oxygen is also appropirate in patients with pulmonary edema, heart failure and mechanical complications free wall rupture, ventricular septum defect, mitral prolapse of NSTEMI or unstable angina. Caution is required in patients with hypotension. This increases the workload on the heart and therefore aggravates the ischemia. Adequate doses of analgesics are necessary to prevent the potentially harmful effects of the sympathetic nervous system.

Analgesics also alleviate pain and relieves anxiety. Morphine is the drug of choice. The required dose of morphine depends on age, body mass index BMI and hemodynamic status. An initial dose of 2 to 5 mg IV is recommended. Injections may be repeated every 5 minutes until 30 mg have been administered. Morphine may cause bradycardia which can be managed with atropine 0. If a total of 30 mg morphine is insufficient to relieve the pain, one should suspect aortic dissection. Note that nitrates and beta blockers also exert analgesic effects explained below. It is crucial that the use of morphine does not limit the use of beta blockers, since they potentiate each others negative hemodynamic effect and only beta blockers reduce mortality. Nitrates cause vasodilatation by relaxing smooth muscle in arteries and veins. This reduces the workload on the myocardium and thus the oxygen demand. Nitrates therefore relieves ischemic symptoms chest pain and pulmonary edema. A dose of 0. Nitroglycerin infusion should be considered if the effect is inadequate severe angina or if there are signs of heart failure. Nitrates should not be administered in 1 patients with hypotension, 2 suspicion of right ventricular infarction, 3 sever aortic stenosis, 4 hypertrophic obstructive cardiomyopathy or 5 pulmonary embolism.

## Chapter 9 : Angina Pectoris (Stable Angina) Nursing Care Management: Study Guide

*Unstable angina or sometimes referred to as acute coronary syndrome causes unexpected chest pain, and usually occurs while resting. The most common cause is reduced blood flow to the heart muscle because the coronary arteries are narrowed by fatty buildups (atherosclerosis) which can rupture causing.*