



CODEN [USA]: IAJPBB

ISSN: 2349-7750

INDO AMERICAN JOURNAL OF  
**PHARMACEUTICAL SCIENCES**

<http://doi.org/10.5281/zenodo.1495518>

Available online at: <http://www.iajps.com>

Review Article

**DIAGNOSIS OF SUSPECTED CARDIAC ISCHEMIA IN  
EMERGENCY DEPARTMENT**

<sup>1</sup>Saad mostafa Al-Harbi, <sup>2</sup>Mashaal Abdulelah Al-zaylaee, <sup>2</sup>Jumana Khalid Alansari, <sup>2</sup>Saja Omar Bakhshwain, <sup>2</sup>Ghady Khaled al-sadiq, <sup>2</sup>Deana Ahmed Faraj, <sup>2</sup>Arwa Emad Ghorab, <sup>2</sup>Wedad Ahmed Bahri, <sup>2</sup>Elham Saeed Bagaryn, <sup>3</sup>Khawater Nasser almotairi, <sup>3</sup>Alanood Sami Alfaris

<sup>1</sup> October University

<sup>2</sup> Batterjee Medical College

<sup>3</sup> Ibsina Medical College

**Abstract:**

*In this review we discuss the fundamental medical devices of past, health examination, and electrocardiography are presently extensively acknowledged to allow very early identification of low-risk patients who have less than 5% possibility of ACS or those who at high risk to develop MI. We searched PubMed, Embase and web of science databases for all published studies that evaluated the accuracy of diagnose ischemic heart attack at the emergency department, through 2018. ACI is a life-threatening condition whose identification can have major economic and therapeutic significance as far as threatening dysrhythmias and avoiding or restricting myocardial infarction dimension. The identification of ACI remains to challenge the skill of even knowledgeable medical professionals, yet doctors proceed (appropriately) to admit the overwhelming majority of patients with ACI; while doing so, they admit many patients without acute ischemia, overstating the probability of ischemia in low-risk patients because of magnified problem for this medical diagnosis for prognostic and therapeutic factors. The goal of the first evaluation of a patient who offers to an outpatient setting with possible ACS has changed from diagnosis to risk stratification. In many cases, the technique is comparable for patients being reviewed in the office and the ED and ought to include a history, physical checkup, and ECG.*

**Corresponding author:**

**Mashaal Abdulelah Al-zaylaee,**  
Batterjee Medical College

QR code



Please cite this article in press Mashaal Abdulelah Al-zaylaee et al., *Diagnosis of Suspected Cardiac Ischemia in Emergency Department.*, Indo Am. J. P. Sci, 2018; 05(11).

**INTRODUCTION:**

Myocardial ischemic injury results from severe impairment of the coronary blood supply generally produced by thrombosis or other acute alterations of coronary atherosclerotic plaques. Chest pain and signs and symptoms consonant with myocardial ischemia are one of the most common factors for emergency department (ED) examination, accounting for about 8% to 10% of the 119 million ED visits annually [1]. Chest pain is among minority illness processes in which patients might initially appear to be well but actually have an underlying severe problem. Inadvertent discharge of patients with acute coronary syndrome (ACS) has actually been related to a short-term mortality of 2%, as well as significant danger of liability [1]. Determining patients with chest ache that are at risk of negative events is essential not only to ED physicians but also to all physicians that evaluate such patients. In one insurance policy industry - based research, the physician group most likely to be sued for missed myocardial infarction (MI) was family practitioners (32%), followed by basic internists (22%) and ED physicians (15%) [2].

Myocardial infarction can be misdiagnosed for a variety of factors. Misconception of results on electrocardiography (ECG) occurs in 23% to 40% of misdiagnosed MIs [3]. Younger age, physician lack of experience, and irregular presentations are extra typical in these patients [3]. An insurance policy declares - based research found that in 28% of circumstances no diagnostic study, not even ECG, was ordered [2]. For that reason, standardizing the evaluation process is crucial for recognizing patients who initially appear to be low danger but who really have ACS.

In this review we discuss the fundamental medical devices of past, health examination, and electrocardiography are presently extensively acknowledged to allow very early identification of low-risk patients who have less than 5% possibility of ACS or those who at high risk to develop MI.

**METHODOLOGY:**

We searched PubMed, Embase and web of science databases for all published studies that evaluated the accuracy of diagnose ischemic heart attack at the emergency department, through 2018. We limited our search to only English studies with human subjects. Furthermore, we searched the references lists of each included studies for more data.

**DISCUSSION:**

- **CLINICAL PRESENTATION**

**Chest pain**

Chest pain or chest discomfort is one of the most common and intricate symptoms for which patients seek emergency medical care. Published records recommend that up to 7% of visitations to the ED involve ailments relating to chest pain [4]. The complaint of breast discomfort incorporates a wide range of problems, which vary from unimportant to high danger in terms of risk to the patient's life and consist of, however, are not restricted to, acute cardiac ischemia (AMI and UAP), thromboembolic disease (pulmonary embolism), aortic dissection, pneumothorax, pneumonia, myocarditis, and pericarditis. Chest pain may be perceived as pain or as feelings such as tightness, pressure, or indigestion, or as discomfort most obvious for its radiation to a surrounding part of the body. Senior patients or patients with diabetes mellitus may have changed capacity to particularly localize pain. Individuals and cultural groups differ in their expression of pain and capacity to connect with wellness professionals, to ensure that presentation might range from just irritating to cataclysmic for problems that seem almost equal when unbiased requirements are matched. The degree of pain does not always associate with the seriousness of disease, making recognition of potentially deadly conditions extremely difficult in specific patients. Because of the significant nature of lots of conditions offering with chest pain and the capacity for significant reduction in morbidity and death with very early medical diagnosis and treatment, scientific policies have actually been developed to guide clinicians with their initial analysis of chest pain, stressing prompt triage, assessment, and initiation of treatment [5]. These clinical regulations are not assessed in this article other than those that apply to ACI.

Normally, the chest pain of acute ischemia has a deep visceral character, avoiding the patient from centering the pain to a particular area of the chest. It is typically described as a pressure-like hefty weight on the chest, a tightness, a tightness regarding the throat, and/or an aching feeling, not influenced by respiration, setting, or movement, that begins gradually, reaches its maximum strength over a duration of 2 to 3 minutes, and lasts for minutes or longer instead of seconds.

**Anginal pain equivalents**

Dyspnea, present in roughly one third of patients with infarction in some series [6], is one of the most essential anginal pain matching. In their multicenter ED trial, Pope et al [7] identified that 16% of patients with supposed ACI offered with a chief complaint of shortness of breath and had an 11% incidence of ACI

at final diagnosis (6% AMI, 5% UAP); in 8%, this was the only complaint, with a 10% incidence of ACI (5% AMI, 5% UAP). A last diagnosis of ACI was not extra constant in patients with a presenting signs and symptom of lack of breath (56% ACI versus 56% non-ACI;  $P = 1/4 = 0.5$ ). As a chief complaint, shortness of breath was extra typically connected with a final medical diagnosis of non-ACI (18% non-ACI versus 7% ACI;  $P = 1/4 = 0.001$ ), potentially mirroring a high occurrence of patients with lung illness in the study population. Due to the fact that 4% to 14% of AMI patients [6]. and 5% of UAP patients present only with abrupt difficulty taking a breath [7], ACI must be taken into consideration as a reason for inexplicable lack of breath.

Both diaphoresis and vomiting, when connected with breast pain, enhance the probability of infarction [6]. Diaphoresis takes place in 20% to 50% of patients with AMI [9]. One study showed that the visibility of nausea without vomiting did not discriminate, however vomiting was considerably much more frequent in patients that "ruled in" [9]. Pope et al [7] identified nausea in 28% of patients with suspected ACI: patients with nausea as a presenting signs and symptom had a 26% incidence of ACI at last medical diagnosis (10% AMI, 16% UAP); patients with nausea or vomiting as their chief complaint (2%) had a 15% occurrence of ACI (11% AMI, 4% UAP); and less than 1% of patients had queasiness or vomiting as their only signs and symptom.

So-called "soft" scientific features, such as exhaustion, weakness, despair, wooziness, and "clouding of the mind," are surprisingly common, taking place in 11% to 40% of patients with AMI [9].

#### • MEDICAL HISTORY

In addition to today clinical characteristics, the existence of a CAD threat element traditionally has been taken into consideration diagnostically helpful in the ED setting. Pope et al's [7] ED series showed a relation amongst patients with a background of diabetes mellitus (31% ACI versus 18% non-ACI;  $P = 1/4 = 0.001$ ), myocardial infarction (45% ACI versus 20% non-ACI;  $P = 1/4 = 0.001$ ), or angina pectoris (63% ACI versus 29% non-ACI;  $P = 1/4 = 0.001$ ) and a final medical diagnosis of ACI; nevertheless, these results require cautious analysis. From the Framingham Study, it is popular that the risk for establishing ischemic heart disease is enhanced over years by the following aspects: male gender, progressing age, a smoking habit, hypertension, hypercholesterolemia, glucose intolerance, ECG problems, a type A character, an inactive way of living, and a family history of very early CAD [6]. Clinicians usually

assess these factors when offering preventative care, due to the fact that they predict the occurrence of future coronary illness. Coronary danger points were developed to provide an estimate of danger over years, nonetheless. Thus, the Framingham Study showed that high blood pressure enhances the danger of ischemic heart disease twofold over 4 years [8], nevertheless just a really small portion of this threat puts on the few hours of the ED patient's acute disease. A patient's record of coronary danger elements is additionally based on prejudices and mistakes. This background is probably much less dependable than the techniques used to assign threat in longitudinal research studies.

#### • PHYSICAL EXAMINATION

The physical checkup is usually not really useful in identifying ACI when compared to the worth of historic data and ECG findings, apart from when it points to an alternate procedure. Medical professionals should not be lulled right into a sense of security by breast pain that is partly or fully duplicated by palpation, nevertheless, because 11% of these patients may have AMI or UAP [9]. Pope et al [7] discovered the pulse rate to be lower in patients with a last medical diagnosis of ACI versus those with a final diagnosis of non-ACI ( $P = 1/4 = 0.02$ ), however this difference was not considered clinically significant. Pulse rate monitoring in isolation appeared to be generally not valuable in ACI identification. First, the patient's pulse rate could be slowed down by the presence of b-blockers as part of a previous treatment routine or by coincident vagal stimulation from ACI (ie, reflex bradycardia and vasodepressor results related to inferoposterior wall ACI) or diagnostic/therapeutic procedures in the ED (eg, phlebotomy, intravenous access). Second, the patient's pulse might be raised by adrenergic extra from the anxiety of a visit to the ED, in addition to the adrenergic excess (eg, tachycardia and enhanced peripheral vascular resistance) connected with feasible ongoing ACI.

#### • ELECTROCARDIOGRAPHY

Whether a patient provides to an office or an ED, the first ECG is the easiest, most basic, most important device for very early danger stratification. Present referrals show that it ought to be conducted within 10 minutes of ED demonstration and might best be considered one of the "crucial indicators" for patients with chest discomfort [10]. All offices should have this capability in addition to a mechanism to give quick analysis. The existence of ST-segment altitude ought to trigger consideration for instant reperfusion treatment. ST-segment anxiety is connected with a significant increase in threat of MI and ischemic

problems [11]. As little as 0.5 mm of ST-segment depression predicts raised threat; the higher the level of anxiety, the higher the possibility of MI and mortality [12]. Although T-wave inversion is usually thought about consistent with ischemia, danger is lower than with ST-segment anxiety [11]. The presence of significant Q waves follows previous MI; nevertheless, when examined independently of other findings, it is much less anticipating of adverse cardiac events than ST-segment depression or elevation. In the absence of ischemic signs, atrial fibrillation is connected with a reduced rate of MI and does not mandate assessment for myocardial necrosis in the lack of additional risky features [13]. However, at discussion initial ECG results in many patients do not show ischemia; in these situations, risk of MI and cardiac issues is reduced, such that in the lack of various other high-risk results, examination can happen in settings aside from an intensive care unit, such as an ED or CPU [14].

Regardless of its importance, ECG has a variety of limitations, consisting of a reasonably reduced diagnostic level of sensitivity for ACS, especially for unstable angina; ischemic modifications are apparent at the time of presentation in just 20% to 30% of patients who have an acute MI [14]. On the other hand, 5% to 10% of patients with MI have typical searchings for on ECG at presentation [14]. The sensitivity of ECG is affected by the structural place of the perpetrator vessel and is less likely to be diagnostic in patients with left circumflex lesions [13].

Continuous examination has looked for to recognize methods to enhance the analysis sensitivity of initial ECG. Serial analysis (every 15-30 minutes) ought to be executed routinely in patients with continuous symptoms or ECG findings that are symptomatic yet not diagnostic of ischemia [10]. With constant ST-segment tracking, a different device, 12-lead ECG, is executed at prespecified time periods, and an alarm system sounds when considerable ST-segment changes occur [15]. Nonetheless, the yield is reduced in low-risk patient populaces.

Various techniques that have actually been assessed consist of addition of posterior leads and multilead

ECG devices. Results have been mixed for routine use posterior leads; yield is likely greater when used to separate the patient with anterior ST-segment clinical depression that has ischemia alone from one that has acute posterior MI. Making use of body mapping and multilead ECG equipments can raise level of sensitivity for determining patients that have posterior MI or left bundle branch block MI [16]. However, the cost-effectiveness of regular use is unclear.

#### • BIOMARKERS

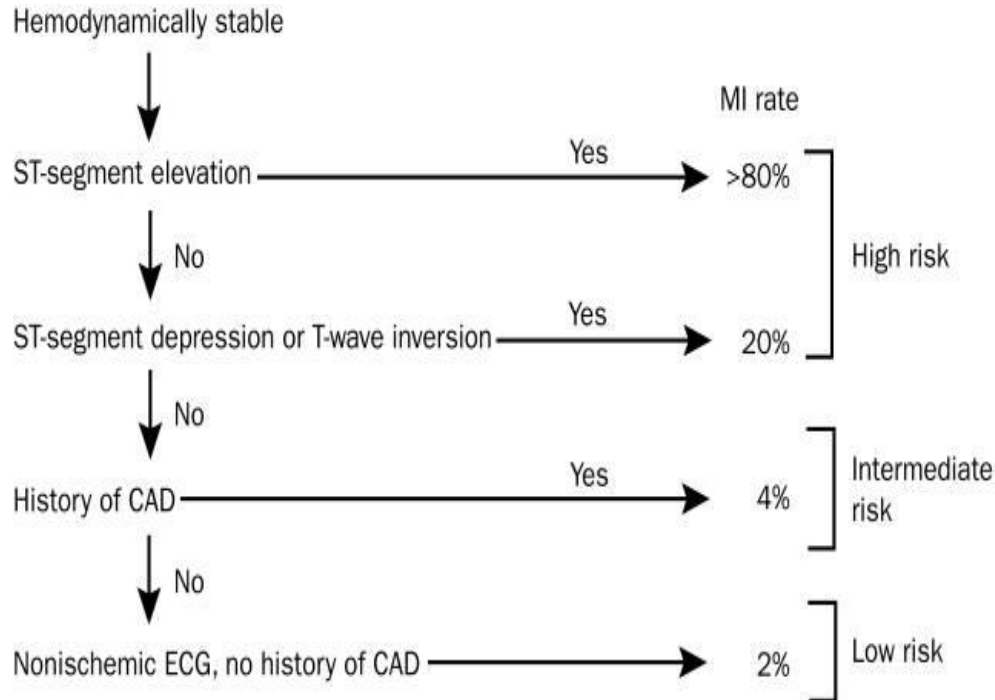
Current recommendations suggest that all patients with suspected ACS must undergo serial cardiac biomarker testing [17]. If baseline records are unfavorable, further sampling must be acquired 6 to 8 hours later relying on syndrome onset. Creatine kinase and creatine kinase MB were the standard signals for determining patients with MI; however, because of their less than ideal level of sensitivity and specificity, present referrals show troponin as the preferred biomarker. Troponin is thought about the criterion common cardiac biomarker for detecting MI since troponin I and troponin T are not found in the blood of healthy persons. Troponin has lots of qualities of an optimum diagnostic signal: it has premium level of sensitivity and uniqueness compared with other markers and assists identify patients with increased short- and long-term danger of cardiac events [10]. Altitudes recognize patients who benefit precisely from aggressive therapy, such as antithrombotic and antiplatelet pharmacotherapy, as well as very early coronary intervention [18]. For that reason, it is suggested that every marker technique include either troponin I or troponin T. Because many assays for troponin I exist, knowledge of a certain assay's features and recommendation varieties is essential for interpreting the outcomes [19]. An essential factor to consider is that a variety of other nonatherothrombotic problems can cause myocardial damages (Table 1) [20]. Distinct troponin elevations associated primarily to ACS from non--ACS-related illness is essential but can be difficult. A serial fluctuation in troponin is most likely to be associated with an ACS etiology [17].

**Table 1.** Differential Diagnosis of Increased Troponin Level in Patients without Acute Coronary Syndrome or Heart Failure [20].

<b>Acute disease</b>	<b>Chronic disease</b>
○ Cardiac and vascular	○ ESRD
• Acute aortic dissection	○ Cardiac infiltrative disorders
• Cerebrovascular accident	• Amyloidosis
– Ischemic stroke	• Sarcoidosis
– Intracerebral hemorrhage	• Hemochromatosis
– Subarachnoid hemorrhage	• Scleroderma
• Medical ICU patients	○ Hypertension
• Gastrointestinal bleeding	○ Diabetes
○ Respiratory	○ Hypothyroidism
• Acute PE	
• ARDS	Iatrogenic disease
○ Cardiac inflammation	○ Invasive procedures
• Endocarditis	• Htx
• Myocarditis	• Congenital defect repair
• Pericarditis	• RFCA
○ Muscular damage	• Lung resection
○ Infectious	• ERCP
• Sepsis	○ Noninvasive procedures
• Viral illness	• Cardioversion
○ Other acute causes of cTn increase	• Lithotripsy
• Kawasaki disease	○ Pharmacologic sources
• Apical ballooning syndrome	• Chemotherapy
• Thrombotic thrombocytopenic purpura	• Other medications
• Rhabdomyolysis	
• Birth complications in infants	Myocardial injury
– Extreme low birth weight	○ Blunt chest injury
– Preterm delivery	○ Endurance athletes
• Acute complications of inherited disorders	○ Envenomation
– Neurofibromatosis	• Snake
– Duchenne muscular dystrophy	• Jellyfish
– Klippel-Feil syndrome	• Spider
• Environmental exposure	• Centipede
– Carbon monoxide	• Scorpion
– Hydrogen sulfide	
– Colchicine	

**• CLINICAL RISK SCORES**

A suggested technique to risk stratification for patients with prospective ACS is the application of scoring systems based on the past and first clinical presentation. The most basic system depends on 1 set of cardiac markers, ECG results, and history of CAD (Figure 1). If findings and history are typical, the patient can be taken into consideration reduced risk, with a chance of MI of less than 5% to 6% [21]. Some of the very first verified chest discomfort formulas were obtained by Goldman and colleagues [22]. These algorithms are based primarily on ECG findings and chest discomfort characteristics. The Goldman formula works in forecasting the necessity for critical care unit admission, advancement of cardiovascular problems, and results; it can consequently be made use of to facilitate decisions relating to disposition, such as whether to admit to a cardiac care or observation device [22]. Due to the fact that the risk of MI in lower-risk patients remains higher than 1%, it cannot recognize patients that can be swiftly released from the ED without further evaluation.



**Figure 1.** Risk of myocardial infarction based on presenting characteristics. CAD = coronary artery disease; ECG = electrocardiography; MI = myocardial infarction [21].

**• IMMEDIATE EXERCISE TEST**

Immediate exercise examinations in the ED for reduced risk patients seem viable, affordable, secure and accurate for determining those who can be discharged for additional outpatient evaluation, however is possibly not as extensively made use of as it might [23]. The anxiety of mistakenly checking a patient with ACS is likely huge, nevertheless may be exaggerated in the appropriately chosen populace [23]. Contraindications to exercise screening in this scenario consist of ischaemic ECG changes, aortic stenosis, noticeable unpredictable angina, uncompensated heart failure, dysrhythmias and uncontrolled hypertension.

**• DIFFERENTIAL DIAGNOSIS**

When patients present to the ED with chest pain, distinguishing ischemic from nonischemic reasons is difficult and regularly the significant emphasis of the examination. Since morbidity is high if a cardiac etiology is not identified early, the overlap of symptoms necessitates a preliminary analysis method that presumes symptoms are cardiac-related unless other causes are undoubtedly evident. Nevertheless, a high awareness of the many other root causes of chest discomfort is needed (Table 2) to direct the therapy of patients with even more usual, much less major disorders and to ensure that life-threatening noncardiac etiologies are not overlooked. Although the ED visit need to be concentrated on determining patients with lethal illness, further examination on discharge from the ED often is called for to identify the etiology of the signs and symptoms, specifically if they persist.

**Table 2.**Potential Noncardiac Causes of Chest Pain [24],[25].

<b>Pulmonary</b> Pulmonary embolism Pneumothorax Pneumonia Pleuritis
<b>Gastrointestinal</b> Gastritis Esophageal disease Reflux Spasm Esophagitis Gallbladder disease Pancreatitis
<b>Musculoskeletal</b> Costochondritis Fibrositis Rib fracture Herpes zoster
<b>Pyschogenetic</b> Anxiety disorder Panic disorder Hyperventilation Somatoform disorder

**CONCLUSION:**

ACI is a life-threatening condition whose identification can have major economic and therapeutic significance as far as threatening dysrhythmias and avoiding or restricting myocardial infarction dimension. The identification of ACI remains to challenge the skill of even knowledgeable medical professionals, yet doctors proceed (appropriately) to admit the overwhelming majority of patients with ACI; while doing so, they admit many patients without acute ischemia, overstating the probability of ischemia in low-risk patients because of magnified problem for this medical diagnosis for prognostic and therapeutic factors.

The goal of the first evaluation of a patient who offers to an outpatient setting with possible ACS has changed from diagnosis to risk stratification. In many cases, the technique is comparable for patients being reviewed in the office and the ED and ought to include a history, physical checkup, and ECG. Group 1 patients must be examined for immediate reperfusion treatment. Group 2 patients must be admitted to the clinic and, in the lack of contraindications, should get antiplatelet and antithrombotic therapy. Patients with ongoing signs and symptoms, persistent ECG adjustments, or hemodynamic instability needs to be reviewed for emergent coronary angiography. More treatment of classification 4 patients is based on the alternative medical diagnosis. Group 3, the low-risk chest

discomfort cohort, is a crucial one due to the fact that it accounts for the majority of patients undertaking ED analysis. Although no single variable (history, physical exam, ECG searching's for) can identify a patient at such reduced danger that additional analysis is unnecessary, the mix of several medical criteria can be utilized to far better establish the first examination procedure.

**REFERENCES:**

1. Pope JH, Aufderheide TP, Ruthazer R, et al. Missed diagnoses of acute cardiac ischemia in the emergency department. *N Engl J Med.* 2000;342(16):1163-1170.
2. Physician Insurers Association of America Acute Myocardial Infarction Study Rockville, MD: Physician Insurers Association of America; 1996:1.
3. Lee TH, Rouan GW, Weisberg MC, et al. Clinical characteristics and natural history of patients with acute myocardial infarction sent home from the emergency room. *Am J Cardiol.* 1987;60(4):219-224.
4. Callahan M. Current practice of emergency medicine. Philadelphia: BC Decker; 1991.
5. American College of Emergency Physicians. Clinical policy for the initial approach to adults presenting with a chief complaint of chest pain with no history of trauma. *Ann Emerg Med* 1995;25:274-99.
6. Kinlen L. Incidence and presentation of

- myocardial infarction in an English community. *Br Heart J* 1973;35:616–22.
7. Pope J, Ruthazer R, Beshansky J, et al. Clinical features of emergency department patients presenting with symptoms of acute cardiac ischemia: a multicenter study. *J Thromb Thrombolysis* 1998;6:63–4.
  8. Marglois J, Kannal W, Feinlieb M, et al. Clinical features and acute course of atypical myocardial infarction—silent and symptomatic. *Am J Cardiol* 1973;32:1–6.
  9. Levene D. Chest pain—prophet of doom or nagging necrosis? *Acta Med Scand* 1981; 644(Suppl):11–3.
  10. Anderson JL, Adams CD, Antman EM, et al. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non-ST-Elevation Myocardial Infarction) [published correction appears in *J Am Coll Cardiol*. 2008;51(9):974] *J Am Coll Cardiol*. 2007;50(7):e1-e157
  11. Savonitto S, Ardissino D, Granger CB, et al. Prognostic value of the admission electrocardiogram in acute coronary syndromes. *JAMA* 1999;281(8):707-713.
  12. Kaul P, Newby LK, Fu Y, et al. Troponin T and quantitative ST-segment depression offer complementary prognostic information in the risk stratification of acute coronary syndrome patients. *J Am Coll Cardiol*. 2003;41(3):371-380.
  13. Zimetbaum PJ, Josephson ME, McDonald MJ, et al. Incidence and predictors of myocardial infarction among patients with atrial fibrillation. *J Am Coll Cardiol*. 2000;36(4):1223-1227
  14. Gibler WB, Sayre MR, Levy RC, et al. Serial 12-lead electrocardiographic monitoring in patients presenting to the emergency department with chest pain. *J ElectroCardiol*. 1993;26(suppl):238-243.
  15. Hoekstra JW, O'Neill BJ, Pride YB, et al. Acute detection of ST-elevation myocardial infarction missed on standard 12-lead ECG with a novel 80-lead real-time digital body surface map: primary results from the multicenter OCCULT MI trial. *Ann Emerg Med*. 2009;54(6):779-788.e1.
  16. Gibler WB, Sayre MR, Levy RC, et al. Serial 12-lead electrocardiographic monitoring in patients presenting to the emergency department with chest pain. *J ElectroCardiol*. 1993;26(suppl):238-243
  17. Morrow DA, Cannon CP, Jesse RL, et al. National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines: Clinical characteristics and utilization of biochemical markers in acute coronary syndromes. *Circulation* 2007;115(13):e356-e375
  18. Morrow DA, Cannon CP, Rifai N, et al. TACTICS-TIMI 18 Investigators Ability of minor elevations of troponins I and T to predict benefit from an early invasive strategy in patients with unstable angina and non-ST elevation myocardial infarction: results from a randomized trial. *JAMA* 2001;286(19):2405-2412.
  19. Apple FS, Jesse RL, Newby LK, et al. National Academy of Clinical Biochemistry and IFCC Committee for Standardization of Markers of Cardiac Damage Laboratory Medicine Practice Guidelines: analytical issues for biochemical markers of acute coronary syndromes. *Circulation* 2007;115(13):e352-e355.
  20. Kelley WE, Januzzi JL, Christenson RH. Increases of cardiac troponin in conditions other than acute coronary syndrome and heart failure. *Clin Chem*. 2009;55(12):2098-2112 Epub 2009 Oct 8
  21. Lee TH, Goldman L. Evaluation of the patient with acute chest pain. *N Engl J Med*. 2000;342(16):1187-1195.
  22. Goldman L, Cook EF, Johnson PA. Prediction of the need for intensive care in patients who come to emergency departments with acute chest pain. *N Engl J Med*. 1996;334(23):1498-1504.
  23. Amsterdam EA, Kirk JD, Diercks DB, et al. Immediate exercise testing to evaluate low-risk patients presenting to the emergency department with chest pain. *J Am Coll Cardiol* 2002;40:251–256.
  24. Salisbury AC, Olalla-Gomez C, Rihal CS, et al. Frequency and predictors of urgent coronary angiography in patients with acute pericarditis. *Mayo Clin Proc*. 2009;84(1):11-15
  25. Cannon CP, Lee TH. Approach to the patient with chest pain. In: Libby P, Bonow RO, Mann DL, Zipes DP, editors., eds. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine* 8th ed. Philadelphia, PA: Saunders; 2008:1195-1205