Acute coronary syndromes: how to make a correct diagnosis in the emergency room

Síndromes coronarianas agudas: como fazer um diagnóstico correto na sala de emergência

Antonio Eduardo Pereira Pesaro¹, Thiago Domingos Corrêa², Luciano Forlenza³, Jaime Freitas Bastos⁴, Marcos Knobel⁵, Elias Knobel⁶

ABSTRACT

The assessment of patients with chest pain or other symptoms suggestive of myocardial ischemia continues as one of the greatest challenges for physicians who work in emergency units. Although the doctor’s experience is relevant, the evaluation of chest pain patients is frequently made in a subjective and inefficient manner when specific diagnostic protocols are not applied. This leads to unnecessary hospitalizations and high hospital expenditures. Our objective is to furnish simple tools that can be easily applied in daily clinical practice as guidelines for managing patients with chest pain in emergency centers. We describe three steps for a correct diagnosis of patients with precordial pain, based on medical history, physical examination, and ancillary tests. These stages include the initial clinical analysis and the use of a specific diagnostic protocol, if necessary. Finally, we present the protocol model implemented at our emergency care center.

Keywords: Myocardial ischemia/diagnosis; Chest pain; Myocardial infarction/diagnosis; Emergency service, hospital; Emergency medical services

INTRODUCTION

The evaluation of patients with chest pain or other symptoms suggestive of myocardial ischemia remains one of the greatest challenges for physicians who work in emergency centers¹⁻². North American data show that approximately six million patients each year seek emergency units for this reason, corresponding to 5.5% of all patients seen in this sector³. Of the six million chest pain patients evaluated in emergency centers, four million are admitted for suspected acute coronary syndrome (ACS)⁴. Nevertheless, due to limitations of the medical history, physical examination, and electrocardiographic findings, many individuals with thoracic pain are hospitalized without an established diagnosis⁵. Furthermore, only 30% of cases have a subsequent confirmation of ACS, and this significantly increases health-related costs⁶.

On the other hand, among the patients evaluated for thoracic pain who are discharged from emergency units, 2% to 13% possess non-diagnosed acute myocardial infarcts⁷. In these patients, the lack of diagnostic accuracy entails a greater risk for adverse events and a mortality rate twice as high as in patients who are appropriately managed⁶⁻⁷.

Although the physician’s experience is relevant, the evaluation of chest pain patients is frequently made in a subjective and ineffective manner when specific diagnostic protocols are not followed. Consequently, high patient admission rates result in hospital costs of approximately 2.5 to 5 thousand dollars per patient, and up to 10 billion dollars a year because of unnecessary hospitalizations⁶⁻⁹.

¹ Assistant physician at the Intensive Care Unit of the Hospital Israelita Albert Einstein – HIAE, São Paulo (SP), Brazil.
² Resident at the Intensive Care Unit of the Hospital Israelita Albert Einstein – HIAE, São Paulo (SP), Brazil.
³ Physician at the Emergency Department of the Hospital Israelita Albert Einstein – HIAE, São Paulo (SP), Brazil.
⁴ Assistant physician at the Intensive Care Unit of the Hospital Israelita Albert Einstein – HIAE, São Paulo (SP), Brazil.
⁵ Coronary Unit coordinator and assistant physician of the Intensive Care Unit of Hospital Israelita Albert Einstein – HIAE, São Paulo (SP), Brazil.
⁶ Vice-President of Clinical Practice of the Sociedade Beneficente Israelita Albert Einstein and Emeritus Director of the Intensive Care Unit of Hospital Israelita Albert Einstein – HIAE, São Paulo (SP), Brazil.
Acute coronary syndromes: how to make a correct diagnosis in the emergency room

81

The application of systematic models of patient care in individuals with chest pain can reduce costs and improve the quality of medical care given\(^{10-12}\). In this context, Chest Pain Units (CPU) were designed to enable implantation of systematized patient care models for patients with thoracic pain or any other symptom suggestive of possible myocardial ischemia. These units use quick diagnostic protocols in order to reduce the time necessary for ACS detection (especially in situations of elevated ST segments, where reperfusion treatment should begin as soon as possible), decrease rates of non-diagnosed acute myocardial infarction (AMI), reduce the number of unnecessary hospital stays, and cut medical costs in general\(^{13,14}\). Additionally, the objective of programs associated with UDTs is the continuous education of the community, in terms of reducing the time lapse between the onset of the patient’s symptom and his seeking an emergency center\(^{9, 15-17}\).

Recent results from institutions that use CPU have showed reduced rates of non-diagnosed AMI (0%-0.4%) in comparison with rates obtained in centers that do not have a specific diagnostic strategy for treating chest pain (2%-10%)\(^{6-9}\). According to data published in our region, depending on the degree of technical ineptness of physicians and the absence of a protocolled diagnostic strategy, the assessment of patients with an initial non-diagnostic electrocardiogram (ECG) correlates with rates of non-diagnosed AMI as high as 20%\(^{5}\). Patients with atypical myocardial ischemia presentations or with minimal electrocardiographic modifications are frequently discharged from hospitals due to the absence of a correct diagnosis. This type of mistake significantly raises mortality in ACS patients who do not receive adequate treatment\(^{6-8}\).

Despite a high prevalence of thoracic pain in emergency centers, there is still a deficiency in standardization of medical care for these patients, which can generate difficulties in diagnosing acute coronary syndromes and delay treatment.

### OBJECTIVE

Our objective is to provide simple tools that are easy to use in daily clinical practice, in order to guide management of patients with chest pain in emergency centers.

We describe three steps for correct diagnosis and stratification of patients with precordial pain. These three stages are an attempt to diagnose ACS based on the medical history, physical examination, and ancillary tests. These phases include the initial clinical analysis, and, when necessary, the use of a specific diagnostic protocol.

Thus, at the end of the three steps we intend to arrive at one of two possibilities:

- the patient does not have ACS and will be referred to ambulatory follow-up or to a physician of another medical specialty;
- the patient does have ACS and should be treated immediately.

### First step: what type of pain is reported?

The first step in managing a patient with precordial pain is to know how to characterize the pain by means of a short medical history that still is one of the most important points in the assessment. Pain characteristics suggestive of angina and the presence of associated symptoms such as sudoreis, vomiting, or dyspnea, are very useful in making the correct diagnosis. At the same time, the medical history allows the elaboration of possible differential diagnoses of non-cardiac etiologies\(^{14,18,19}\).

The Coronary Artery Surgery Study (CASS) presented a classification focused on type of pain in patients with suspected coronary artery disease (CAD) which is useful in estimating the probability of ACS\(^{19}\). In our study, it was possible to classify precordial pain in four distinct groups: definitively anginal, probably anginal, probably not anginal, or definitively not anginal\(^{20}\) (chart 1). As to the triggering factor, despite the fact that ACS usually manifests with rest pain, several patients presented with recent onset or recently worsened angina brought on by light effort over the previous weeks (chart 2). Exertion as a triggering factor is an important component of pain that is probably or definitely anginal.

#### Chart 1. Assessment of type of chest pain

<table>
<thead>
<tr>
<th>Type of pain</th>
<th>Characteristics of pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definitely anginal</td>
<td>Retrosternal or precordial pain or discomfort, generally precipitated by physical exertion, which may radiate to the shoulder, mandible, or internal aspect of the arm (both), with duration of a few minutes and alleviated by rest or nitrate in less than 10 minutes</td>
</tr>
<tr>
<td>Probably anginal</td>
<td>Most, but not all, characteristics of anginal pain</td>
</tr>
<tr>
<td>Probably non-anginal</td>
<td>Few characteristics of definitely anginal pain (“atypical pain”, symptoms of “anginal equivalent”)</td>
</tr>
<tr>
<td>Definitely non-anginal</td>
<td>No characteristics of anginal pain, strongly indicative of a non-cardiac diagnosis</td>
</tr>
</tbody>
</table>

Adapted from the CASS study\(^{14-18}\)

#### Chart 2. Forms of presentation of unstable angina\(^{20}\)

<table>
<thead>
<tr>
<th>Definition</th>
<th>Characteristics</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Progressive angina</td>
<td>Prior angina that worsened by at least one functional class (CCS*) to at least functional class III in the previous 4 weeks</td>
<td></td>
</tr>
<tr>
<td>Recent onset angina</td>
<td>Angina that began at class III in the previous 4 weeks</td>
<td></td>
</tr>
<tr>
<td>Rest angina</td>
<td>Prolonged anginal pain, generally lasting longer than 20 minutes</td>
<td></td>
</tr>
</tbody>
</table>

CCS*: Cardiovascular Canadian Society. CCS I: Angina under less than habitual exertion. CCS II: Angina under habitual exertion. CCS III: Angina under more than habitual exertion. CCS IV: Resting angina.

Nevertheless, in spite of the indisputable role of pain type evaluation, this method has limitations. In the Multicenter Chest Pain Study, acute ischemia was diagnosed in 22% of the patients with “stabbing” chest
pain, 13% of patients with pleuritic pain, and 7% of patients whose pain could be entirely reproduced upon palpation\(^{(19)}\). Additionally, patients could not have chest pain as the presentation of ACS, but rather epigastralgia, nausea, vomiting, sudoresis, or dyspnea (ischemic equivalents)\(^{(9)}\).

**Second step: how probable is [the presence of] an ACS at the moment?**

The primary challenge is to establish the probability of the clinical picture presented by the patient being an ACS. Merging information from the clinical evaluation of pain described in Step 1, personal antecedents, physical examination, and ECG, it is possible to estimate the probability of myocardial ischemia and therefore make treatment decisions\(^{(21)}\) (chart 3).

<table>
<thead>
<tr>
<th>High probability</th>
<th>Medium probability</th>
<th>Low probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any one of the points below:</td>
<td>Absence of high probability points and at least one of the following points:</td>
<td>Absence of high and medium probability points and:</td>
</tr>
<tr>
<td>• Definitely anginal pain</td>
<td>• Pain probably not anginal in diabetic patients</td>
<td>• Pain definitely not anginal</td>
</tr>
<tr>
<td>• Probably anginal pain (especially in advanced age patients)</td>
<td>• 2 risk factors except DM*</td>
<td>• Pain probably not anginal and 1** risk factor (except DM)</td>
</tr>
<tr>
<td>• Hemodynamic and ECG changes during precordial pain</td>
<td>• Extra-cardiac vascular disease**</td>
<td>• ECG normal or with non-specific changes</td>
</tr>
<tr>
<td>• Non-dynamic ST depression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>of 0.5 to 1 mm or inversion of T wave &gt; 1 mm</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Risk factors for CAD: systemic arterial hypertension, dyslipidemia, smoking, diabetes mellitus (DM), and advanced age

** Extra-coronary manifestation of atherosclerosis: cerebrovascular disease, aortopathy, renovascular hypertension, and chronic arterial obstruction in limbs

*** Individually assess patients who might be of intermediate probability if there is only one risk factor, but this factor is in advanced age patients

ECG is the best initial diagnostic test as it is quick, low cost, and universally available. Among patients with chest pain and an elevated ST segment, the incidence of AMI is 80%-90%\(^{(22)}\). For those with a depressed ST segment or a presumably new inverted T wave, the incidence of infarction or unstable angina is around 20%\(^{(22,23)}\). For patients with a normal ECG, the AMI risk is about 4% among those with a history of CAD and 2% in those with no history of CAD\(^{(24)}\). In other words, a non-specific or normal ECG does not exclude an ACS. However, serial tracings can increase the sensitivity of the ECG in a diagnostic assessment\(^{(22)}\).

Low-probability patients do not need a supplementary myocardial ischemia evaluation, but differential diagnoses for the clinical presentation (pulmonary thromboembolism, dissection of the aorta, osteomuscular disease, gastrointestinal diseases, etc.) should be investigated. High-probability patients should be considered as having an ACS and therefore, treatment should be promptly initiated\(^{(21)}\).

Patients with a medium probability of having an ACS should be submitted to a specific chest pain evaluation protocol, described in the third step, since they require symptom monitoring and additional subsidiary tests to aid in the diagnosis and treatment decision. In this group it is also vital to consider differential diagnoses such as acute dissection of the aorta, pulmonary embolism, and gastrointestinal diseases\(^{(19,21)}\).

**Third step: does the patient need to be included in the diagnostic protocol of the chest pain unit?**

As was discussed above, Chest Pain Units use quick or accelerated diagnostic protocols in order to diminish the rates of non-diagnosed AMI, reduce unnecessary hospitalizations, and cut medical costs\(^{(9,15-17)}\).

One possibility for the CPU protocol consists in maintaining the patient with a moderate probability of ACS under observation for up to 12 hours after the onset of pain. During this period, the patient will be clinically watched and submitted to sequential clinical evaluations. An acute change in the physical examination, such as the presence of signs of heart failure (dyspnea, pulmonary congestion, hypotension, presence of the third heart sound or new mitral insufficiency) is strongly suggestive of ACS\(^{(5)}\).

Electrocardiograms should be performed in a sequential manner (i.e., 3, 6, 9, and 12 hours after onset of pain and at each new chest pain episode). The presence of dynamic ischemic changes from admission ECG, on serial ECGs, or in monitoring of the ST segment (particularly ST segment depression), identify a patient with ACS\(^{(5)}\).

Myocardial necrosis markers constitute the fundamental point for diagnostic and prognostic assessments of patients with a probable ACS. These may be divided into early and late markers (chart 4) and should also be collected in a sequential manner after the start of pain\(^{(5,25,26)}\).

**Characteristics of myocardial necrosis markers**

<table>
<thead>
<tr>
<th>CK-MB</th>
<th>Troponin</th>
<th>Myoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevation (hours)</td>
<td>4-6</td>
<td>3-6</td>
</tr>
<tr>
<td>Peak (hours)</td>
<td>12-20</td>
<td>10-24</td>
</tr>
<tr>
<td>Normalization (days)</td>
<td>2-3</td>
<td>10-15</td>
</tr>
</tbody>
</table>

Early necrosis markers are represented by the subtypes of CK-MB (MB1 and MB2) and by myoglobin. Plasma levels of CK-MB subtypes start to climb within 2 hours and peak at 12 hours after symptoms begin, normalizing in approximately 24 hours. They confer reliability to the diagnosis of myocardial necrosis after the first 6 hours of symptom onset (95.7% sensitivity and 93.9% specificity). Blood myoglobin increases in 2-3 hours from symptom onset and peaks in 8 hours, with a return to normal levels in 12 hours. It has minimal specificity for heart muscle,
since it is also released by skeletal muscles. However, if negative in 6 hours, it has a high negative predictive value for acute infarct of the myocardium (75%-97%)\(^5,25,26\).

Conversely, late necrosis markers are represented by CK-MB (activity/mass) and by troponins I and T. They are called late markers since they only allow diagnostic accuracy (sensitivity and specificity over 93%) after 10-12 hours from onset of symptoms. Their sensitivity for early diagnosis (in 6 hours) is only 60%, as it is not possible to exclude the diagnosis of myocardial necrosis before 10-12 hours. There are several different protocols for sample collection intervals depending on the algorithm used by the CPU for a secondary stratification of the pain. The most common utilize sample collections 0-3-6-9 and 12 hours after admission or only at 6 and 12 hours after pain starts\(^5,25,26\).

After the thoracic pain protocol is applied, only two options are possible:

- the protocol is negative: the patient evolved without pain, there is no significant change in physical examination, serial ECGs show no new modifications, and myocardial necrosis markers are negative;
- the protocol is positive: the patient experienced at least one of the following changes: new anginal pain, physical examination suggestive of acute heart failure, any ECG with new modifications (new bundle branch block, ST/T segment changes) or an elevation in myocardial necrosis markers. In this case, the initially doubtful clinical presentation is now confirmed as ACS, and patient treatment should begin immediately.

A negative protocol does not exclude the possibility of ACS, but it excludes AMI\(^5,20,27\). Even when the situation is a case of ACS (remote possibility), it does not yet represent a high risk of progressing to infarction or death. After considering differential diagnoses for the reported complaint (pulmonary thromboembolism, dissection of the aorta, osteomuscular diseases, gastrointestinal diseases, etc), two options are still possible: discharge the patient from the hospital, but instruct him to return for an ambulatory visit in the following days with the objective of scheduling a non-invasive test for ischemia, or carry out the test in a hospital environment at the end of the chest pain protocol\(^5,20,27\). The objective of this non-invasive stratification is to detect the presence of functional myocardial ischemia and to evaluate the prognosis for this group of patients. The most frequently used tests are the ergometric stress test, stress echocardiogram, and myocardial perfusion scintigraphy with physical or pharmacological stress (dipiridamol, adenosine, or dobutamine)\(^5,20,27\).

Patients with non-invasive tests suggestive of ischemia should be treated as having ACS. Patients with normal non-invasive tests can be safely discharged\(^5,20,27\).

**CONCLUSION**

The acute coronary syndromes comprise an ample spectrum of clinical presentations. An appropriate management of patients who present to emergency care units with chest pain has a significant impact on morbidity, mortality, and reduction of hospital costs related to these treatments. Recently, a chest pain protocol was implemented at our center. The use of serial ECGs, myocardial necrosis markers, and resting myocardial scintigraphy has been particularly useful in patients with suspected ACS who had atypical pain or a non-diagnostic ECG (chart 5 and figure 1). Results of the impact of this protocol will be published soon.

---

**Figure 1.** Chest pain protocol of the Hospital Israelita Albert Einstein: quick assessment to exclude myocardial ischemia – General flowchart
Chart 5. Chest pain cases: definition of initial strategy – Hospital Israelita Albert Einstein

<table>
<thead>
<tr>
<th>Clinical manifestation</th>
<th>Initial ECG</th>
<th>Increased CV risk?</th>
<th>Initial diagnostic hypothesis</th>
<th>Initial strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical pain</td>
<td>Supra ST</td>
<td>Indifferent</td>
<td>AMI with supra ST</td>
<td>AMI protocol with supra</td>
</tr>
<tr>
<td></td>
<td>Ischemic</td>
<td></td>
<td>ACS with supra ST</td>
<td>ACS flowchart with supra</td>
</tr>
<tr>
<td></td>
<td>Normal or non-diagnostic</td>
<td>Yes</td>
<td>Probable ACS with supra ST</td>
<td>ACS flowchart with supra or chest pain protocol</td>
</tr>
<tr>
<td>Atypical pain or possible ischemic equivalent</td>
<td>Supra ST</td>
<td>Indifferent</td>
<td>AMI with supra ST</td>
<td>AMI protocol with supra</td>
</tr>
<tr>
<td></td>
<td>Ischemic</td>
<td></td>
<td>ACS with supra ST</td>
<td>ACS flowchart with supra</td>
</tr>
<tr>
<td></td>
<td>Normal or non-diagnostic</td>
<td>Yes</td>
<td>Possible ACS with supra ST</td>
<td>Chest pain protocol</td>
</tr>
<tr>
<td></td>
<td></td>
<td>No</td>
<td>Non-ischemic pain</td>
<td>Differential diagnosis of non-ischemic Chest pain</td>
</tr>
</tbody>
</table>


Erratum

Erratum for: Luchessi AD, Curi R, Costa Neto CM.
“e-amino group” and “a amino group” for “epsilon-amino group”.

einstein. 2007; 5(1):80-84