The Interdisciplinary Management of Acute Chest Pain

Raphael R. Bruno, Norbert Donner-Banzhoff, Wolfgang Söllner, Thomas Frieling, Christian Müller, Michael Christ

SUMMARY

Background: Acute chest pain of non-traumatic origin is a common reason for presentation to physician’s offices and emergency rooms. Coronary heart disease is the cause in up to 25% of cases. Because acute chest pain, depending on its etiology, may be associated with a high risk of death, rapid, goal-oriented management is mandatory.

Methods: This review is based on pertinent articles and guidelines retrieved by a selective search in PubMed.

Results: History-taking, physical examination, and a 12-lead electrocardiogram (ECG) are the first steps in the differential diagnostic process and generally allow the identification of features signifying a high risk of life-threatening illness. If the ECG reveals ST-segment elevation, cardiac catheterization is indicated. The time-dependent measurement of highly sensitive troponin values is a reliable test for the diagnosis or exclusion of acute myocardial infarction. A wide variety of other potential causes (e.g., vascular, musculoskeletal, gastroenterologic, or psychosomatic) must be identified from the history if they are to be treated appropriately. Elderly patients need special attention.

Conclusion: Acute chest pain is a major diagnostic challenge for the physician. Common errors are traceable to non-recognition of important causes and to an inadequate diagnostic work-up. Future studies should be designed to help optimize the interdisciplinary management of patients with chest pain.


Acute chest pain is one of the more common symptoms that bring patients to the emergency room (1). In this CME article, we present an illustrative case to motivate a discussion of the most important facts about the causes and diagnostic work-up of non-traumatic chest pain. The discussion will be presented from the points of view of various medical specialties, each with its own particular emphasis.

Learning objectives

Readers of this article should become able to
- name the most important components of the diagnostic work-up of chest pain, and
- critically discuss the life-threatening and non-life-threatening diseases and disorders that cause chest pain, along with their clinical significance.

Method

The authors selectively searched international databases (PubMed, Google Scholar) and the current guidelines of the European and German Cardiological Societies (2–4) and the German College of General Practitioners and Family Physicians (Deutsche Gesellschaft für Allgemeinmedizin und Familienmedizin, DEGAM) (5, e1).

Case illustration

F.M., a 49-year-old man, is taken to the hospital by the emergency medical service. He says that he suddenly felt an intense, dull pain behind the breastbone while driving. In a panic, bathed in sweat, he called the emergency medical service and was picked up.

The epidemiology and clinical relevance of chest pain

3–6% of all patients presenting to the emergency room have chest pain as their chief complaint.
The emergency physician: The epidemiology and clinical relevance of chest pain

3–6% of all patients presenting to the emergency room have chest pain as their chief complaint (6). Whenever this is the case, a rapid evaluation should be performed to determine whether any of the following life-threatening diseases may be present—the “big five” of acute chest pain:

- Acute myocardial infarction
- Pulmonary arterial embolism
- Acute aortic syndrome
- Tension pneumothorax
- Boerhaave syndrome.

20–25% of emergency patients with chest pain have an acute coronary syndrome (6, e2). Patients who are experiencing myocardial infarction with...
ST-segment elevation (STEMI) can be immediately identified from the 12-lead electrocardiogram (ECG) taken on arrival.

The acute coronary syndrome without ST-segment elevation (NSTE-ACS) is subdivided into non-ST-elevation myocardial infarction (NSTEMI) and unstable angina pectoris. Patients with NSTE-ACS are further evaluated with the aid of time-dependent changes in cardiac-specific biomarkers such as the highly sensitive cardiac troponin T (hs-cTnT) or troponin I (hs-cTnI) (Figure 1).

Delays in the diagnosis of an acute aortic syndrome or pulmonary arterial embolism are usually due to an atypical presentation. In Germany, acute pulmonary arterial embolism causes about 40,000 deaths each year, with a high early mortality (2). The aortic syndrome arises with an incidence of 2.0 to 3.5 cases per 100,000 person-years; many more cases probably go unrecognized, as this entity, too, is associated with high early mortality (7). Patients with an aortic syndrome who present to a physician immediately but have atypical (e.g., neurological) symptoms receive the correct diagnosis promptly in only 15% of cases (8). About 40% of patients with an aortic syndrome are first thought to be suffering from an acute coronary syndrome; such misdiagnoses confer an unfavorable prognosis (9). Without treatment, the death rate of patients with an acute aortic syndrome is about 1% per hour (e3, e4).

The general practitioner:
Important considerations for general practice
Patients who come to their general practitioner’s office with chest pain are much less likely to display life-threatening causes than those who present with chest pain to the emergency room. The common etiologies in the former situation are gastroenterological, musculoskeletal, respiratory, and psychosomatic (5, 10).

The Marburg Heart Score is a useful aid to differential diagnosis in the general practice setting (Box 1). The degree of danger is often unclear at first; the general practitioner’s task is to interpret the historical and clinical findings with the greatest possible precision. The most important consideration is the rapid hospitalization of patients with potentially life-threatening diseases.

The cardiologist:
Clinical evaluation
Acute myocardial infarction often presents with a complaint of squeezing (“visceral”) retrosternal pain or pressure, radiating into the shoulder, arm(s), or back (e5). Radiation into the left shoulder or arm is not very specific, while radiation into the right arm or both arms is highly specific (11). The distinction between typical and atypical symptoms is not very useful in the differential diagnosis; nor is improvement after the administration of nitroglycerine (12, 13, e6).

Pleuritic chest pain of pulmonary or other extra-cardiac origin (e.g., pulmonary arterial embolism, pneumothorax, pneumonia, pleuritis, pericarditis) is of a sharp quality and is more intense on coughing or deep inspiration.

The emergency physician:
Triage
Hemodynamically stable patients with chest pain are classified as urgent (e.g., Emergency Severity Index category 2, ESI 2), while patients who are unstable on either respiratory or hemodynamic grounds are classified as critical (ESI 1) (14, 15). In all such patients, the vital signs are measured immediately but have atypical (e.g., neurological) symptoms receive the correct diagnosis promptly in only 15% of cases (8). About 40% of patients with an aortic syndrome are first thought to be suffering from an acute coronary syndrome; such misdiagnoses confer an unfavorable prognosis (9). Without treatment, the death rate of patients with an acute aortic syndrome is about 1% per hour (e3, e4).

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**Considerations from general practice**
Patients who come to their general practitioner’s office with chest pain generally do not have a life-threatening illness. The common etiologies are gastroenterological, musculoskeletal, respiratory, and psychosomatic.

**Unstable patients**
In all patients who are considered critical, the vital signs are measured and a 12-lead ECG with right ventricular leads is recorded at once.
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Case illustration: Continuation I

The patient is of normal weight; he seems anxious. He continues to complain of pressing chest pain (numerical pain scale [NPS]: 7 out of 10) with accompanying autonomic manifestations (cold sweat, agitation). The vital signs are: respiratory rate, 25/min; heart rate, 83/min; blood pressure, 150/100 mm Hg; oxygen saturation on room air, 98%; tympanic temperature, 36.2°C. He is alert and oriented and is assigned ESI category 2. To treat the pain, he is given 4 mg of morphine IV.

The emergency physician:
Initial interpretation
The patient is hemodynamically stable. His elevated respiratory rate may be due to a severe cardiac, pulmonary, or vascular disease, but it could also reflect hyperventilation suggesting a psychosomatic reason. The physical examination reveals no evidence of a tension pneumothorax. The intensity of this patient’s pain (NRS ≥ 7) justifies the acute administration of an opiate; there is no cause for concern that this might impair the diagnostic work-up by “masking” the pain (e12, e13).

Case illustration: Continuation II

Within 5 minutes, a 12-lead ECG with right-ventricular leads is recorded.

The emergency physician:
Reading the ECG
The ECG findings (Figure 2) are consistent with a benign variant of early repolarization; a STEMI is highly unlikely (17). This prognostically benign normal variant is common in young men. An elevated ST takeoff from the QRS complex is typically followed by a concave ST-segment transition (17).

In all patients with acute chest pain, the ECG should be analyzed in a structured process (Table 1). The sum of the negative T waves, the number of limb leads with negative T waves, ST elevation in leads V1 or aVR, low voltage, or a newly arisen right bundle branch block are independent predictors of cardiogenic shock (18, e14). Hyperventilation can cause a wide variety of ECG changes and can also be associated with chest pain (e15); thus, an arterial blood-gas analysis should be performed as well.

The cardiologist:
Further ECG recordings
If the 12-lead ECG is normal, recording from right ventricular leads is recommended (3). The ECG on admission does not suffice to diagnose or rule out an acute myocardial infarction, as it is normal in up to 14% of patients with an occluded epicardial coronary vessel (19). Until the diagnosis has been definitively established, the ECG should be monitored continuously, and the 12-lead ECG should be repeated within three hours, or sooner if symptoms reappear (Figure 1) (3). In emergency diagnosis, a 12-lead ECG with right ventricular leads is recorded at once. Unstable patients (Box 2) also undergo a focused cardiac ultrasound examination (FoCUS, [e7]) for further differential diagnostic evaluation: this can detect regional wall-motion abnormalities in the heart. More than 35% of elderly patients with an acute myocardial infarction do not complain of chest pain (e8). For precisely this reason, elderly patients need special attention (16). The elderly often have other, accompanying illnesses and thus a higher risk of death. Should, therefore, be a low clinical threshold for invasive coronary work-up in elderly patients, taking their degree of frailty into consideration (e9, e10).

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special attention should be paid to the “neglected” aVR lead: an elevation in this lead may indicate acute occlusion of the left coronary artery (“main stem ECG”) or severe 3-vessel coronary artery disease (20, e16). Ischemic changes in the ECG (Table 2) are predictive of major adverse cardiac events (21).

The general practitioner: ECG and general practice

General practitioners usually see patients in a very early stage of myocardial infarction, in which ST elevation often cannot be seen and cardiac biomarker levels are normal. The history and physical findings are crucially important. A single ECG recording and bio-marker measurement cannot rule out an acute coronary syndrome.

The acute coronary syndrome

STEMI, NSTEMI, and unstable angina pectoris together constitute the acute coronary syndrome; each of these entities represents a different degree of severity of the same underlying disease of the coronary vessels.
bio-marker measurement cannot rule out an acute coronary syndrome.

The cardiologist:
Further remarks on the acute coronary syndrome
The three entities STEMI, NSTEMI, and unstable angina pectoris together constitute the acute coronary syndrome, each of them represents a different degree of severity of the same underlying disease of the coronary vessels (3). NSTEMI should not merely be considered a less dangerous variant of STEMI, although it is true that the 30-day mortality of NSTEMI is lower, its long-term prognosis is comparably unfavorable (22, e17). Coronary heart disease is a chronic inflammatory disease. An NSTEMI is an alarm signal for recurrent infarction and death. The correct diagnosis of an acute coronary syndrome without ST elevation is essential, because excellent treatment options are available for this condition.

The emergency physician:
Differential-diagnostic considerations
The symptoms and signs of the patient described would lead the physician in the emergency room to suspect an acute coronary syndrome without ST elevation, while not yet ruling out an acute aortic syndrome or a pulmonary arterial embolism. The Wells Score enables risk stratification for thromboembolic syndromes. 3 points are awarded if there is evidence of a deep leg-vein thrombosis, and a further 3 points if other diagnoses appear unlikely.

1.5 points are awarded for each of the following conditions, if present: heart rate above 100/min, immobility for more than three days, any surgery undergone in the past four weeks, and any prior history of pulmonary arterial embolism or deep pelvic vein thrombosis. Finally, 1 point is awarded for hemoptysis and 1 point for neoplasia (e21).

A pulmonary arterial embolism can be ruled out if the pre-test probability is low (e.g., Wells Score < 2 points: 1.3% probability of pulmonary arterial embolism [e21]) and the highly sensitive D-dimer assay yields subthreshold values. High-risk features should also be considered in the differential-diagnostic evaluation of an acute aortic syndrome (eBox 1). If there are grounds to suspect an acute aortic syndrome, the degree of elevation of the D-dimer values can serve as a guide to the potential indication for the appropriate imaging studies.

The emergency physician:
Physical examination
The physical examination is conducted at the same time that venous blood is drawn for laboratory testing (complete blood count, sodium, potassium, creatinine, blood urea nitrogen, highly sensitive cardiac troponin T [hs-cTnT]) and arterial blood is taken for blood gas analysis. The physical examination serves to identify high-risk features for pulmonary arterial embolism or an acute aortic syndrome (eBox 1).

Case illustration: Continuation III
The physical examination reveals no abnormality of the heart, lungs, or abdomen, and the peripheral pulses are symmetrically palpable. The patient

TABLE 1

<table>
<thead>
<tr>
<th>Condition</th>
<th>Typical ECG changes</th>
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<tbody>
<tr>
<td>Myocardial infarction</td>
<td>ST elevation (measured at the J point) in leads V2–V3</td>
</tr>
<tr>
<td></td>
<td>≥ 0.25 mV for men up to age 40</td>
</tr>
<tr>
<td></td>
<td>≥ 0.2 mV for men over age 40</td>
</tr>
<tr>
<td></td>
<td>≥ 0.15 mV for women and/or ST elevation (measured at the J point) in other leads</td>
</tr>
<tr>
<td></td>
<td>≥ 0.1 mV in the absence of left-heart hypertrophy or left bundle branch block</td>
</tr>
<tr>
<td></td>
<td>– ST depression in V1–V3, mainly in combination with terminal T negativity. This may</td>
</tr>
<tr>
<td></td>
<td>be associated with concomitant ST elevation by ≥ 0.25 mV in leads V4–V6</td>
</tr>
<tr>
<td></td>
<td>– ST elevation in aVR by ≥ 0.05 mV with depression in the chest wall leads,</td>
</tr>
<tr>
<td></td>
<td>indicating main stem or proximal RIVA stenosis</td>
</tr>
<tr>
<td></td>
<td>– V7–V9 ≥ 0.05 mV (0.1 mV for men under age 40)</td>
</tr>
<tr>
<td></td>
<td>– newly arisen T negativity</td>
</tr>
<tr>
<td></td>
<td>– newly arisen left bundle branch block</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Newly arisen right bundle branch block, axis deviation, SIQII type, inappropriate</td>
</tr>
<tr>
<td></td>
<td>axis type, T negativity over the anterior wall, P pulmonale</td>
</tr>
<tr>
<td>Aortic syndrome</td>
<td>Usually nonspecific changes, normal in 30%, ST elevations in 5% (RCA &gt; LCA)</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>Precordial R loss and T inversion, low QRS amplitude, rightward axis deviation; a</td>
</tr>
<tr>
<td></td>
<td>left pneumothorax can produce ECG changes resembling those of cardiac ischemia</td>
</tr>
</tbody>
</table>

(2, 17, 18, 20, 4, e16, e18, e19, e20)

RCA/LCA, right/left coronary artery; RIVA, ramus interventricularis anterior (anterior interventricular branch).

NSTEMI as an alarm signal
Coronary heart disease is a chronic inflammatory disease. An NSTEMI is an alarm signal for recurrent infarction and death.

Good treatment options
The correct diagnosis of an acute coronary syndrome without ST elevation is essential, because excellent treatment options are available for this condition.
reports no known pre-existing illnesses or allergies. He is a non-smoker and participates in fairly demanding endurance sports several times a week. His mother died a few weeks ago, in her late 70’s, of an aortic dissection. His father sustained an NSTEMI in his early 50’s.

The cardiologist:
Risk factors
An NSTEMI is still possible despite the absence of typical cardiac risk factors, as a first-degree relative is known to have had an acute myocardial infarction before age 60. The regular performance of demanding exercise without angina pectoris never rules out an acute coronary syndrome (23); conversely, exercise-induced angina pectoris is not necessarily associated with coronary heart disease (24). An acute myocardial infarction must, therefore, be included in the differential diagnosis.

A positive family history of an acute aortic syndrome is a high-risk feature (e11, e18). The D-dimers should be measured in such a case, so that an acute aortic syndrome can be “non-invasively” ruled out in case the values are low, in view of the suggestive clinical findings and the atypical symptoms for an acute coronary syndrome (25, e22). The diagnostic tests of choice, if the clinical suspicion is high, are transesophageal echocardiography and angiographic computerized tomography (angio-CT) with contrast medium; these two tests are of comparable sensitivity.

Case illustration: Continuation IV
Focused echocardiography (FoCUS), performed while the history is being taken, reveals a normal left ventricular ejection fraction, normal endocardial wall motion of the left ventricle, no pericardial effusion, no evidence of right ventricular strain, and no evidence of a widened ascending aorta or a dissection membrane.

The emergency physician:
Diagnostic considerations
Focused echocardiography is used in the emergency setting to expand the diagnostic possibilities; with it, important questions can be answered in a matter of minutes (26).

If the patient has a pneumothorax, lung sliding is absent, and a “lung point” or “stratosphere sign” is seen in the ultrasonographic image (eFigure 1, eFigure 2). Ultrasound is better than x-rays for the detection of pneumothorax, especially ventral pneumothorax (e23). Moreover, it more readily reveals pleural effusions, pulmonary edema (e24), infiltrates (27), and peripheral pulmonary arterial embolism (eTable).

In the emergency situation, the physician should also consider a possible psychogenic origin of the patient’s symptoms. The recent, dramatic death of his mother may have precipitated a panic attack. The blood gas analysis yields evidence of hyperventilation (pH 7.53, pO2 92 mmHg, pCO2 23 mmHg).

The gastroenterologist:
Esophageal rupture
The gastroenterologist considers the possibility of life-threatening gastroenterological conditions as the cause of the patient’s pain. Perforation of the esophagus (usually in its thoracic segment) arises iatrogenically, in the setting of endoscopic procedures, in 50–75% of cases (28). Boerhaave syndrome, i.e., esophageal rupture caused by vomiting, is present in 10–15% of patients with esophageal perforations (e25); it follows upon an unusually intense emesis (e26).

In the present case, esophageal rupture seems very unlikely, as there is no history of vomiting or of a recent endoscopic procedure.

### TABLE 2

<table>
<thead>
<tr>
<th>Initial ECG findings in patients with acute chest pain</th>
<th>Probability of at least one major adverse cardiac event within 30 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>– Normal</td>
<td>3.2%</td>
</tr>
<tr>
<td>– Nonspecific abnormalities</td>
<td>9.4%</td>
</tr>
<tr>
<td>– Abnormalities that are not characteristic of acute cardiac ischemia</td>
<td>10.2%</td>
</tr>
<tr>
<td>– Newly arisen abnormalities that are typical of acute cardiac ischemia, or evidence of a prior myocardial infarction</td>
<td>36.6%</td>
</tr>
<tr>
<td>– ST elevation in acute myocardial infarction</td>
<td>72.7%</td>
</tr>
</tbody>
</table>

*Major adverse cardiac events: death, acute myocardial infarction, or revascularization procedure. From (21).
The specialist in psychosomatic medicine: 
The recent death of the patient’s mother from aortic dissection arouses the suspicion of a possible non-organic cause of the patient’s chest pain (a somatoform functional disorder, an anxiety or panic disorder, or depression) (e27–e29). The diagnostic evaluation for such disturbances should be carried out in parallel with the somatic evaluation. One-quarter to one-half of such patients present repeatedly to the emergency room with the same or similar complaints (29, e30).

● Somatoform autonomic functional disorders of the cardiovascular system are stress-processing disorders associated with more or less well-localized functional somatic complaints (somaticization tendency), and with further nonspecific autonomic symptoms (e31). Patients with somatoform disorders present a therapeutic challenge to emergency teams (30, e32, e33).

● Panic disorders are characterized by episodes of chest pain of sudden onset, usually accompanied by tachycardia, dizziness, a feeling of illness, nausea, shortness of breath, fear (including fear of death), and emotional tension (e34). Patients with panic disorders often suffer from depression as well. Patients who have both an underlying cardiac disease and an anxiety disorder or depression are more likely to suffer from anxious self-observation (31) (eBox 2).

The general practitioner: 
What to do next
In the non-emergency setting of the general practitioner’s office, psychosocial and somatic hypotheses should be considered in parallel.

The traditional sequential workup (first rule out somatic disease, then evaluate for a possible psychosomatic disorder) is obsolete and may aggravate the patient’s somatization. Instead, the patient should be asked about his or her own thoughts about the possible cause of the problem, the degree of subjective impairment should be assessed, and possible psychosocial factors should be judged. Somatic and psychosocial causes and factors are not mutually exclusive. Inadequate communication in the emergency care setting often leads to unnecessary dramatization, good communication is the precondition for a well-targeted diagnostic approach.

The cardiologist: 
What to do next
The gold standard for the identification of cardiac ischemia is the sequential measurement of highly sensitive cardiac troponin (hs-cTnT or hs-cTnI). Creatine kinase (CK) measurement is of low diagnostic precision and is therefore not recommended (3). The negative predictive value of an initially negative highly sensitive troponin value (hs-cTn) is above 95% (4). An acute myocardial infarction can be

Panic disorder as a differential diagnosis
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Identifying cardiac ischemia
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definitively diagnosed (“ruled in”) on the basis of a time-dependent change in circulating hs-cTnT levels that exceeds the defined cutoff values (4, e34). Recent studies have shown that diagnostic decisions can be made with fairly high certainty as early as 1 hour after the patient’s arrival in the emergency room (Figure 3) (4, e34). Invasive diagnostic evaluation should be performed within two hours if a high-risk constellation is present (persistent angina pectoris, acute heart failure, hemodynamic instability, life-threatening ventricular arrhythmia) (3) (Figure 1). If the GRACE Risk Score exceeds 140 (e35–37), or if a primary risk criterion is present (relevant rise or fall of hs-cTnT, dynamic changes of the ST segment or T wave [symptomatic or clinically silent]), an invasive diagnostic evaluation should ensue within 24 hours, i.e., at the next (early) elective opportunity (Figure 1, Box 3).

**Case presentation: Continuation V**

Aside from the abovementioned abnormalities of the arterial blood gases, there were no abnormal laboratory findings. The hs-cTnT value was <3 ng/L; the D-dimer concentration was 0.2 mg/L.

**The cardiologist:**

**Exclusion of an acute myocardial infarction**

The hs-cTn value should be determined in all patients with acute chest pain of suspected cardiac origin.

The lack of a time-dependent change in the hs-cTn level is an important diagnostic criterion (Δhs-cTn < 20% in three hours, [32]). A single hs-cTnT concentration that is below the level of detection (5 ng/L for hs-cTnT) at the time of admission, as in the case presented, has a high negative predictive value for the exclusion (ruled out) of a myocardial infarction (Figure 3). A false-negative diagnosis rate below 1% is considered acceptable in the current British guidelines and elsewhere (32, 33, e33–36).

Troponin measurement in general medical practices with the aid of so-called point-of-care tests (POCT) must be viewed with caution: some of the assays are of low diagnostic sensitivity (34).

Elevated hs-cTn values are common in elderly patients and presumably reflect the combined effects of the atherosclerotic load and impaired renal clearance (Box 3).

**The emergency physician:**

**Further considerations regarding laboratory testing**

A number of studies have shown that the hs-cTn value in pulmonary arterial embolism can be used for valid risk assessment. Moreover, the measurement of natriuretic peptides (BNP, NT-proBNP) is recommended for risk stratification in pulmonary arterial embolism (35, 36).

The two differential diagnoses that were mentioned initially—pulmonary arterial embolism and an acute aortic syndrome—are highly unlikely, considering their low pre-test probability and the subthreshold D-dimer level. The indiscriminate

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**BOX 3**

**The differential diagnosis of elevated troponin levels**

- Chronic or acute renal dysfunction
- Severe acute or chronic congestive heart failure
- Hypertensive crisis
- Tachy- or bradycardia
- Pulmonary arterial embolism, severe pulmonary hypertension
- Inflammation, e.g., myocarditis
- Acute neurologic disease, e.g., stroke or subarachnoid hemorrhage
- Aortic dissection, aortic valve disease, or hypertrophic cardiomyopathy
- Cardiac contusion, cardiac ablation, pacemaker, cardioversion, or endomyocardial biopsy
- Hypothyroidism
- Apical ballooning syndrome (takotsubo cardiomyopathy)
- Infiltrative diseases, e.g., amyloidosis, hemochromatosis, sarcoidosis, or scleroderma
- Cardiotoxic agents, e.g., Adriamycin, 5-fluorouracil, herceptin, snake venom
- Burns of more than 30% of the body surface
- Rhabdomyolysis
- Critical illness, primarily respiratory failure or sepsis

*modified from (31)

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**Risk stratification in pulmonary arterial embolism**

The measurement of natriuretic peptides (BNP, NT-proBNP) is recommended for risk stratification in pulmonary arterial embolism.

**Age-adapted D-dimer assessment**

The interpretation of D-dimer levels in patients over age 50 should be age-adapted (cutoff value = age × 10 µg/L).
measurement of D-dimer levels tends to confuse, rather than clarify, the diagnostic picture, and it generates high costs. D-dimers should only be measured as part of a targeted diagnostic evaluation in which validated instruments for risk stratification are used, such as the Wells or GENEVA Scores (e38, e39).

The interpretation of D-dimer levels in patients over age 50 should be age-adapted (cutoff value = age × 10 µg/L) (37). In the outpatient setting, the Wells score can be used in combination with a bedside D-dimer test and selective compression ultrasonography to rule out a deep leg-vein thrombosis (e40).

D-dimer levels can be used to rule out an aortic aneurysm (25, e22), the plasma level is correlated with the extent of dissection (e41). Tomographic imaging is recommended if there is a high clinical suspicion of pulmonary arterial embolism or an aortic aneurysm (2).

Case illustration: Continuation VI

A second troponin measurement three hours after admission reveals no pathological rise in the hs-cTnT level. The patient is now asymptomatic.

The gastroenterologist:

Gastroenterological causes of chest pain

About 60% of cases of chest pain of gastroenterological origin are accounted for by gastroesophageal reflux disease (GERD), and a further 20% by disorders of esophageal motility, including esophageal spasm, hypertensive peristalsis (“nutcracker esophagus”), and achalasia (e42).

Gastroesophageal reflux and esophageal motility disorders can cause ST segment abnormalities. In about 30% of cases of chest pain of gastrointestinal origin, esophageal changes other than the ones just mentioned are present (eBox 3). Particularly in elderly patients, esophageal lesions caused by the swallowing of tablets must be considered (specifically: antibiotics, antiviral drugs, potassium chloride, nonsteroidal anti-inflammatory drugs, and bisphosphonate) (e42).

Acute pancreatitis may present with diffuse symptoms, including dyspnea and chest pain. ST-segment elevations and inversions are present in up to 50% of cases, particularly in the posterior wall (38).

Case presentation: Continuation VII

After the various life-threatening diseases in the differential diagnosis have been ruled out, and after consultation with a specialist from the liaison psychosomatic service, the patient is discharged into ambulatory care. He receives outpatient psychotherapy and his symptoms are greatly improved.

The emergency physician and the specialist in psychosomatic medicine: Telling the patient the diagnosis

When the patient’s problem has been determined to be of psychosomatic origin, thorough patient education has a decisive impact on the further course. Statements such as “We didn’t find anything wrong” merely increase the chance of chronicisation of symptoms and of “doctor hopping” (e43, e44). In contrast, patients are reassured by being told matter-of-factly that unresolved stress often leads to physical disturbances such as chest pain, and that these disturbances are not life-threatening (e45). Another option is further ambulatory treatment by a general practitioner who has supplementary, specialized training in psychosomatic medicine. The key to the correct diagnosis and treatment of patients with non-cardiac chest pain is smoothly functioning interdisciplinary collaboration (Box 2).

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What to do if a pulmonary arterial embolism is suspected

Tomographic imaging is recommended if there is a high clinical suspicion of pulmonary arterial embolism or an aortic aneurysm.

Psychosomatic causes

When the patient’s problem has been determined to be of psychosomatic origin, thorough patient education has a decisive impact on the further course.
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- “The Diagnosis and Treatment of Optic Neuritis” (issue 37/2015) until 6 December 2015,
Please answer the following questions to participate in our certified Continuing Medical Education program. Only one answer is possible per question. Please select the most appropriate answer.

**Question 1**
A patient presents with acute chest pain. Which of the following leads you to suspect an anxiety disorder as the cause?
- a) The patient tells you that organic heart disease is not the cause.
- b) The symptoms are independent of exertion.
- c) The 12-lead ECG is normal.
- d) There is a prior history of mental illness.
- e) The patient is under 40 years old.

**Question 2**
According to the literature, what percentage of patients presenting to the emergency room with chest pain have an acute coronary syndrome?
- a) 1–5%
- b) 15–25%
- c) 30–40%
- d) 45–55%
- e) 60–70%

**Question 3**
Which of the following establishes the diagnosis of pneumothorax?
- a) Thoracic ultrasound, with demonstration of the lung point
- b) Pulmonary artery angiography, with corresponding artefacts
- c) Ultrasound, with visible lung sliding
- d) Echocardiography, with D-shaping
- e) Lack of demonstration of the stratosphere sign

**Question 4**
What ECG finding is typical of myocardial infarction?
- a) ST depression in V1–V3, mainly in the setting of simultaneous terminal T-wave inversion
- b) Low QRS amplitude
- c) New right bundle branch block with axis deviation
- d) Preterminal inverted T-waves in leads V5 and V6
- e) Precordial R loss and T inversion

**Question 5**
What is Boerhaave syndrome?
- a) ECG abnormalities that are not characteristic of acute ischemia
- b) Stress-processing disturbances associated with localized functional somatic symptoms
- c) Spontaneous esophageal perforation after massive vomiting
- d) A special type of acute aortic syndrome
- e) Regional myocardial wall-motion abnormalities

**Question 6**
In a 50-year-old man with acute chest pain, laboratory testing on emergency admission reveals an hs-cTnT concentration of 458 ng/L. What is the likely diagnosis?
- a) Pulmonary embolism
- b) Somatoform functional disorder
- c) Persistent angina pectoris
- d) Acute heart failure
- e) Acute myocardial infarction

**Question 7**
What diagnosis can be excluded by the D-dimer concentration in a patient with acute chest pain?
- a) Acute heart failure
- b) Aortic aneurysm
- c) Acute myocardial infarction
- d) Tension pneumothorax
- e) Pericarditis

**Question 8**
In what syndrome can the Well score be used for risk stratification?
- a) Left heart syndrome
- b) Wolff-Parkinson-White syndrome
- c) Mallory-Weiss syndrome
- d) Thrombembolic syndrome
- e) Chronic hypertension syndrome

**Question 9**
What must be considered in the differential diagnosis of elevated hs-cTnT or hs-cTnl levels?
- a) Bronchial asthma
- b) Uncomplicated urinary tract infection
- c) Obstructive airway disease
- d) Mucoviscidosis
- e) Pulmonary arterial embolism

**Question 10**
In persons over age 50, the cutoff D-dimer level for the exclusion of pulmonary arterial embolism should be age-adapted. What is the appropriate formula for clinical use?
- a) Age × 10 µg/L
- b) Age × 20 µg/L
- c) Age × 30 µg/L
- d) Age × 40 µg/L
- e) Age × 50 µg/L
Supplementary material to:

The Interdisciplinary Management of Acute Chest Pain
by Raphael R. Bruno, Norbert Donner-Banzhoff, Wolfgang Söllner, Thomas Frieling, Christian Müller, and Michael Christ

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e.TABLE

Focused echocardiography and thoracic ultrasound in the emergency room (e7)

<table>
<thead>
<tr>
<th>Right-ventricular strain</th>
<th>Focused echocardiography</th>
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</thead>
<tbody>
<tr>
<td>RV dilatation</td>
<td>Pericardial effusion / tamponade</td>
</tr>
<tr>
<td>RV increased pressure</td>
<td>Regional wall movement abnormalities</td>
</tr>
<tr>
<td>Impaired RV function</td>
<td>Left-heart failure</td>
</tr>
<tr>
<td></td>
<td>Aortic dissection</td>
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</tbody>
</table>

**Focused echocardiography**

- **Pericardial effusion / tamponade**: Effusion – hemodynamically significant? Right atrial collapse? “Swinging heart” (i.e., in the effusion)?
- **Regional wall movement abnormalities**: Hypo- or akinesia, apical ballooning
- **Left-heart failure**: Visual estimation of pump function (the “eyeball method”)
- **Aortic dissection**: Aortic valve insufficiency, dissection membrane, pericardial tamponade, low ejection fraction, dilatation of the ascending aorta (> 40 mm)

**Thoracic ultrasound**

- **Pneumothorax**: Absent lung gliding, stratosphere sign, lung point
- **Pulmonary edema**: ≥ 3 B lines (comet tail) per field in 2 of 4 areas on both sides of the chest
- **Pleural effusion**: Pleural effusion – is drainage necessary? Anechoic? With echo-dense structures within?
- **Pulmonary arterial embolism**: Subpleural consolidations near the chest wall
- **Pneumonia**: Broncho-aerogram (in peripheral pneumonia)
eBOX 1

Suspected acute aortic syndrome: aid to clinical decision-making (from e18)

1. Determine whether the nature of the pain implies high risk
   - Site: chest, back, or abdomen, and
   - sudden onset, high intensity, and
   - quality: tearing, stabbing, cutting

2. Identify risk factors
   - Marfan syndrome / Loeys-Dietz syndrome / connective tissue diseases
   - Positive family history of aortic aneurysm
   - Previously diagnosed aortic valve disease
   - Prior aortic surgery or invasive procedure
   - Previously diagnosed thoracic aortic aneurysm

3. Identify physical findings that imply high risk
   - Check for evidence of hypoperfusion:
     - weak pulses?
     - blood pressure difference between the two arms (>20 mmHg)?
     - focal neurologic deficit arising at the same time as the pain?
   - Auscultate over the aortic valve to detect a heart murmur (new or previously unknown, onset at the same time as the pain?)
   - Evaluate for signs of hypotension / shock / syncope
   - Hypertensive crisis at time of onset?

4. Count the positive risk factors and evaluate the risk
   - Not in the high-risk group: < 2 high-risk features
   - In the high-risk group: ≥ 2 high-risk features

5. Pursue further diagnostic evaluation according to the assessed risk
   - Not in the high-risk group: further evaluation for other causes of chest pain
   - High-risk group: imaging of the aorta urgently indicated
Evidence from the history that supports a psychosomatic cause of chest pain (modified from the DEGAM chest pain guidelines [5])

- High likelihood of a somatoform functional disorder affecting the cardiovascular system:
  - repeated visits to the doctor with nonspecific autonomic symptoms, such as palpitations, tachycardia, or unusual sensations in the chest such as burning or aching
  - AND intense, distressing worry about heart disease, without reassurance from negative test results or doctors’ explanations
  - AND no adequate somatic explanation of symptoms

- High likelihood of an anxiety disorder:
  - anxiety attacks or panic attacks or sudden attacks of physical symptoms of unclear origin, such as tachycardia, dizziness or shortness or breath, at any time in the past 6 months
  - nervousness, anxiousness, or tension, and inability to stop worrying or to gain control of worries

- High likelihood of a depressive disorder if the answer is “yes” to either of these questions:
  - “Have you often felt dejected, depressed, or hopeless in the past month?”
  - “Have you often in the past month had little interest in, or pleasure from, things you used to enjoy?”

Gastrointestinal causes of chest pain (e13)

- Gastroesophageal reflux (GERD), erosive (ERD) or non-erosive (NERD)
- Barrett’s esophagus
- Esophageal motility disorders (diffuse esophageal spasm, hypercontractile esophagus, achalasia)
- Hypersensitive esophagus
- Schatzki’s ring, webs
- Eosinophilic esophagitis
- Mallory-Weiss syndrome, Boerhaave syndrome
- Drug-induced esophageal ulcer
- Infection (viral, thrush, esophagitis)
- Gastroduodenal ulcer
- Pancreatitis, biliary colic, cholangitis
**eFigure 1:** Pneumothorax: B- and M-mode ultrasonographic images of the right lung, lateral upper quadrant: stratosphere sign, characterized by horizontal artefacts below the pleural line.

**eFigure 2:** Proof of pneumothorax: B- and M mode ultrasonographic images of the right lung, lateral upper quadrant: the stratosphere sign (with absent lung gliding) in expiration alternating with the seashore sign (with preserved lung gliding) in inspiration, at the point where the pneumothorax begins.