Acute coronary syndrome

LECTURE IN INTERNAL MEDICINE FOR V COURSE STUDENTS

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US MLE TEST

A 58-year-old man with history of coronary artery disease, hypertension, and hyperlipidemia presents to an emergency department for evaluation of chest pain. He reports somewhat suddenly experiencing dull left-sided chest discomfort while at rest at home that was not relieved with taking nitroglycerin. His vital signs are: T 37.1, HR 94 beats per minute, BP 133/87, and O2 saturation 97% on ambient air. His ECG (shown, figure A) shows no ST-segment changes; serum troponin is not elevated. His chest pain subsequently resolves and he is admitted to the cardiac service for further management. Which of the following is by itself not an indication for early percutaneous coronary intervention?

1. Hemodynamic instability, 2. Positive cardiac biomarker, 3. ST-segment elevation on ECG, 4. Recurrent or refractory chest pain, 5. Heart failure

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

US MLE TEST

Correct Answer 2: This patient with known coronary artery disease (CAD) presents with angina at rest, thus the diagnosis is unstable angina, a variant of acute coronary syndrome (ACS). All of the above are strong indications for intervention except answer 2, positive cardiac biomarker, which alone is not enough to warrant this management.

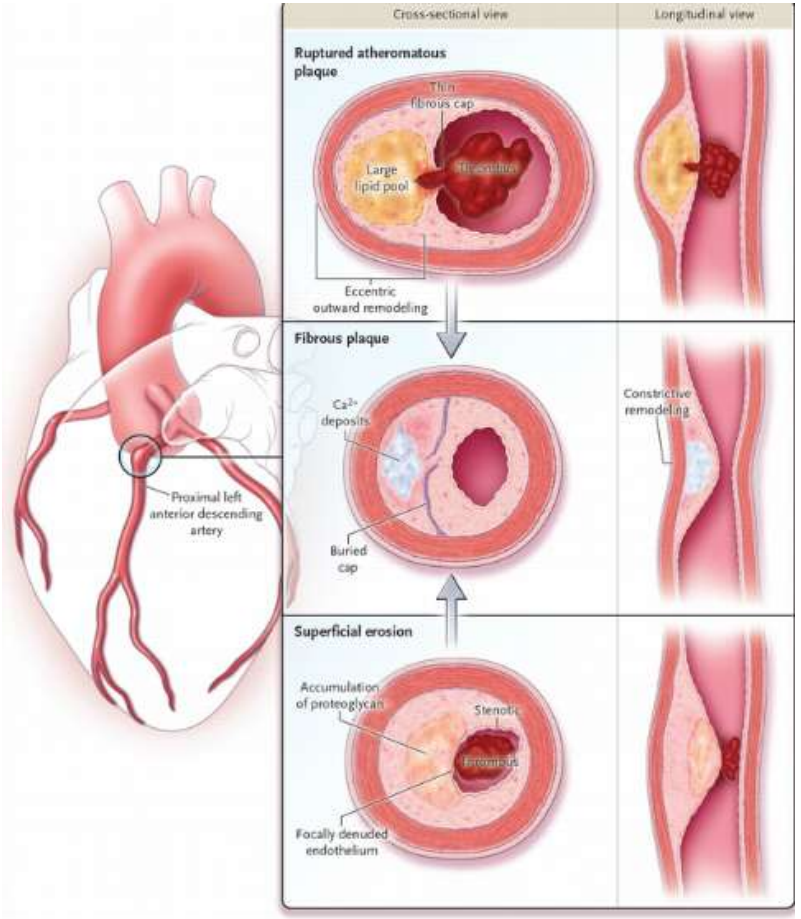
Incorrect Answers:

Answers 1, 3-5: These are all strong indications for early PCI in patients with UA/NSTEMI.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

Plan of the Lecture

- Definition
- Epidemiology
- Risk factors
- Etiology
- Mechanisms
- Classification
- Clinical investigation
- Diagnosis
- Treatment
- Lifestyle modifications and cardiac rehabilitation
- Prognosis
- Prophylaxis
- Abbreviations
- Diagnostic and treatment guidelines



nejm.org/doi/full/10.1056/NEJMra1216063

Definition 1

Acute coronary syndrome (ACS) has evolved as a useful operational term that refers to a spectrum of conditions compatible with acute myocardial ischemia and/or infarction (MI) that are usually due to an abrupt reduction in coronary blood flow ranging clinically from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non–ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina (UA).

emedicine.medscape.com/article/1910735-overview content.onlinejacc.org/article.aspx?articleid=1910086

Definition 2

Most patients with STEMI develop QMI, and a few develop NQMI. Most of those without ST-elevation develop NQMI and only a few may develop QMI

The ACS associated with high healthcare costs, frequent recurrences and hospitalizations, and high risks of sudden death and short-term mortality.

emedicine.medscape.com/article/1910735-overview content.onlinejacc.org/article.aspx?articleid=1910086

Definition
Unstable Angina 1

- UA is defined as myocardial ischemia at rest or minimal exertion in the absence of cardiomyocyte necrosis
- Among patients presenting with suspected NSTE-ACS, the introduction of high-sensitivity cardiac troponin measurements in place of standard troponin assays resulted in an increase in the detection of MI and a reciprocal decrease in the diagnosis of UA.

eurheartj.oxfordjournals.org/content/37/3/267

Definition
Unstable Angina 2

- Compared with NSTEMI patients, individuals with UA do not experience myocardial necrosis, have a substantially lower risk of death and appear to derive less benefit from intensified antiplatelet therapy as well as early invasive strategy.

eurheartj.oxfordjournals.org/content/37/3/267

Definition
Definitions of Myocardial Infarction 1

- Universal: MI defines cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischemia.

Definition
Definitions of Myocardial Infarction 2

- My: Acute myocardial infarction is a disease or a clinical syndrome accompanying other diseases, which is represented by acute coronarogenous aseptic inflammation of the part of a heart wall, and clinically correlates with stress reactions of body control systems and is determined by the extent, localization, nature, and stage of structural transformations in the infarction zone, as well as circulation changes, and their consequences.

eurheartj.oxfordjournals.org/content/37/3/267 im.medicine.karazin.ua//downloads/presentations/Yabluchanskiy_Infarction_Strategy.pdf

US MLE TEST

A 63-year-old diabetic woman with a history of GERD is presenting to the ER in the afternoon with new onset abdominal pain. Her symptoms began while she was walking her dog this morning when she began to feel a dull, 3/10 pain in her mid-upper abdomen. She attributed this to her meal which contained bacon and eggs that morning and said that it felt similar to her usual GERD symptoms but was also accompanied by fatigue and nausea which made her anxious. Her last bowel movement was yesterday morning and was "normal." Physical exam was remarkable for a nervous elderly woman appearing her stated age. Vitals were within normal limits. No abnormalities were noted on HEENT, heart or lung exam. Abdominal exam showed a soft, nontender, nondistended abdomen with no guarding or rebound. Which of the following studies should be ordered first?

1. Abdominal X-ray , 2. Electrocardiogram, 3. Abdominal CT with contrast, 4. Adbominal CT without contrast, 5. Chest X-ray.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

US MLE TEST

Correct Answer 2: This patient is at a high risk for having a myocardial infarction with an atypcial presentation. Although abdominal pathology should be considered, it is absolutely vital that an electrocardiogram be performed to rule out myocardial ischemia as a cause of her symptoms.
Incorrect Answers:
1, 3, 4: These imaging studies are useful for investigating abdominal pathology which this patient may be presenting with. However, an ECG must be used to rule out a cardiac emergency first. Although an abdominal emergency is possible (e.g. small bowel obstruction, mesenteric ischemia, and perforated diverticulitis), her somewhat mild symptoms make these less likely., 5: A chest X-ray is not especially useful for investigating her acute cardiac or abdominal events unless a perforated bowel is suspected which is unlikely given the lack of severe symptoms.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

Epidemiology

Acute Coronary Syndrome around the World 1

- Worldwide, coronary artery disease (CAD) is the single most frequent cause of death
- Over seven million people every year die from CAD, accounting for 12.8% of all deaths
- Every sixth man and every seventh woman in Europe will die from myocardial infarction.

eurheartj.oxfordjournals.org/content/33/20/2569

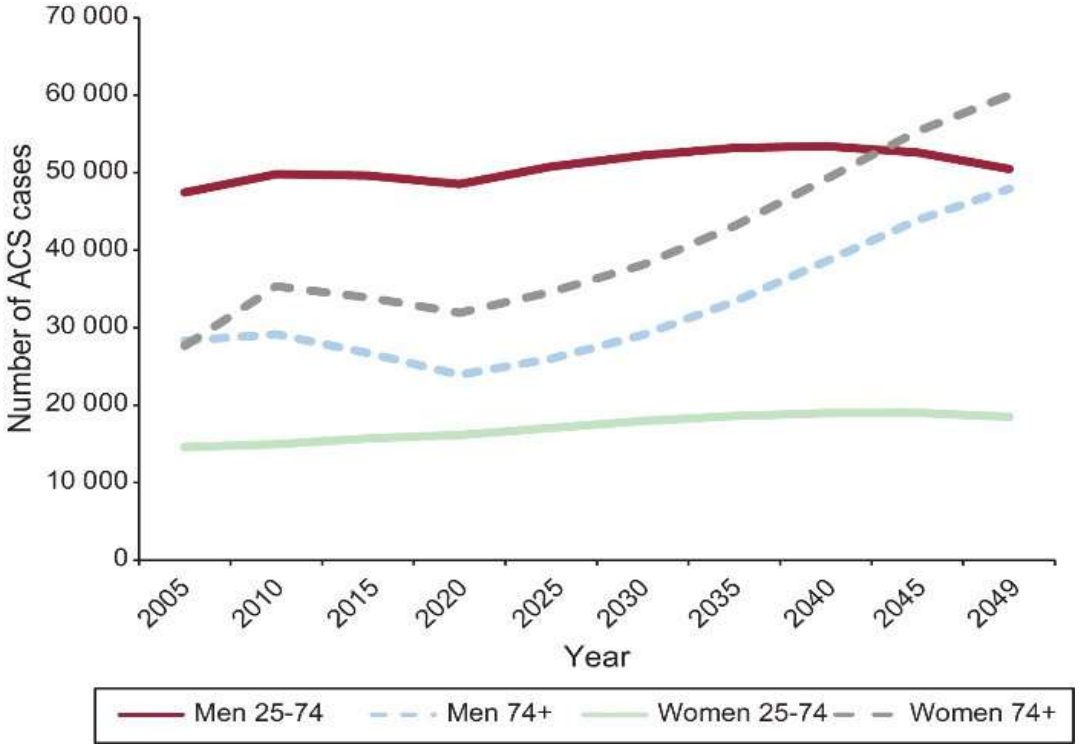
Epidemiology

Acute Coronary Syndrome around the World 2

- The incidence of hospital admissions for myocardial infarction varies among countries
- The most comprehensive STEMI registry is probably in Sweden, where the incidence is 66 STEMI/100 000/year.

eurheartj.oxfordjournals.org/content/33/20/2569

Epidemiology
(Acute Coronary Syndrome in the Spanish Population)

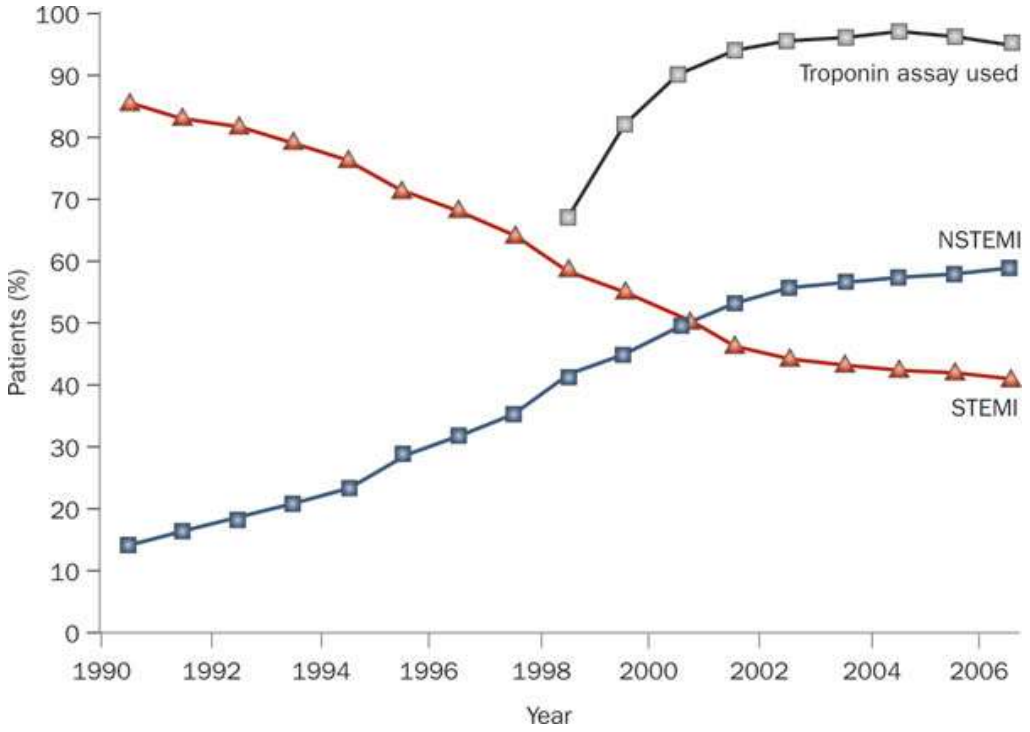


Number of acute coronary syndrome cases, trend from 2005 to 2049 by sex and age group in the Spanish population. ACS, acute coronary syndromes.

revespcardiol.org/en/epidemiology-of-acute-coronary-syndromes/articulo/90202863/

Epidemiology

(Troponin and Diagnosis of ST-elevated and non-ST-elevated Acute Myocardial Infarction)

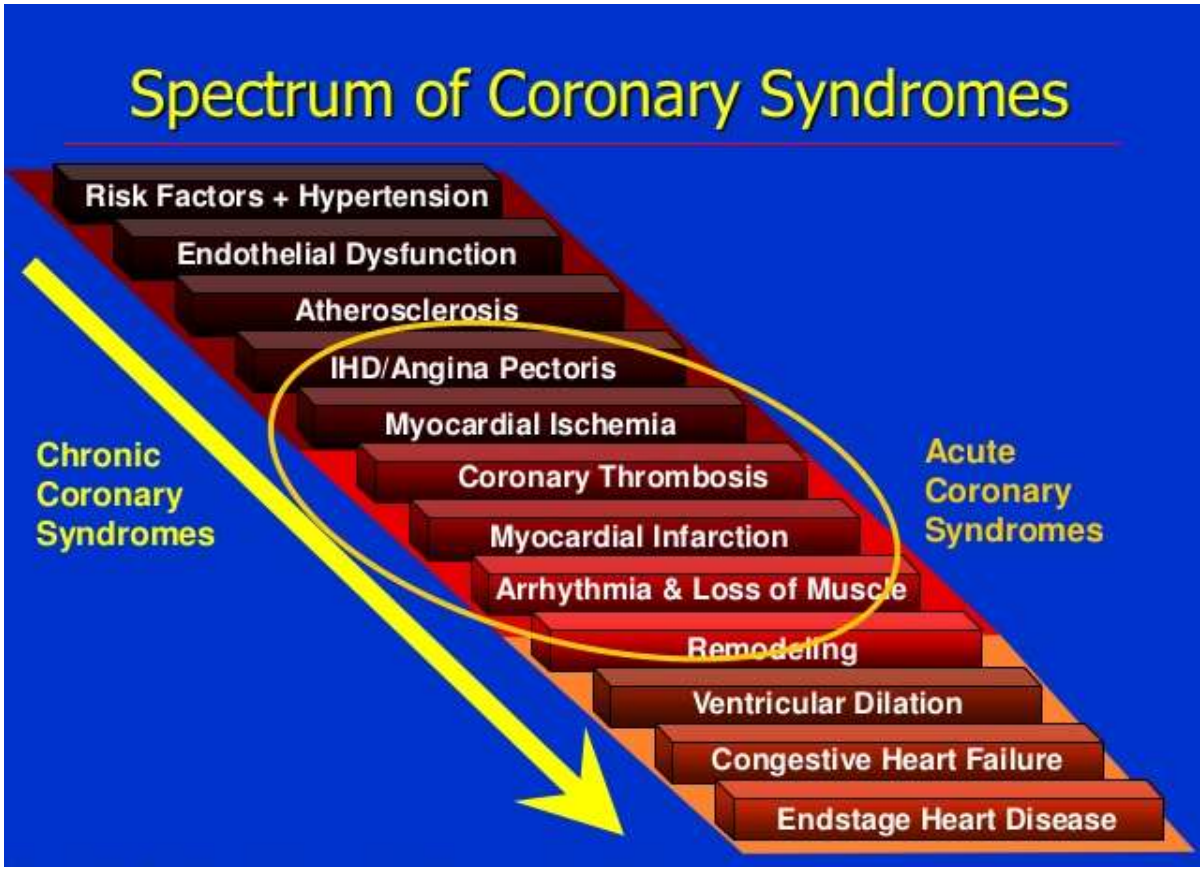


Quality of a diagnosis depends strictly from diagnostic procedures.

nature.com/nrcardio/journal/v8/n3/images/nrcardio.2010.199-f2.jpg

Risk Factors

The risk factors for an ACS are the same as those for other types of clinical atherosclerosis including heart disease.



mayoclinic.org/ image.slidesharecdn.com/asimnstemi-final-140204114442-phpapp01/95/acute-coronary-syndrome-nstemi-7-638.jpg?cb=1400815933

Etiology 1

- The hallmark of an ACS is the sudden imbalance between myocardial oxygen consumption (MVO_2) and demand, which is usually the result of coronary artery obstruction.

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Etiology 2

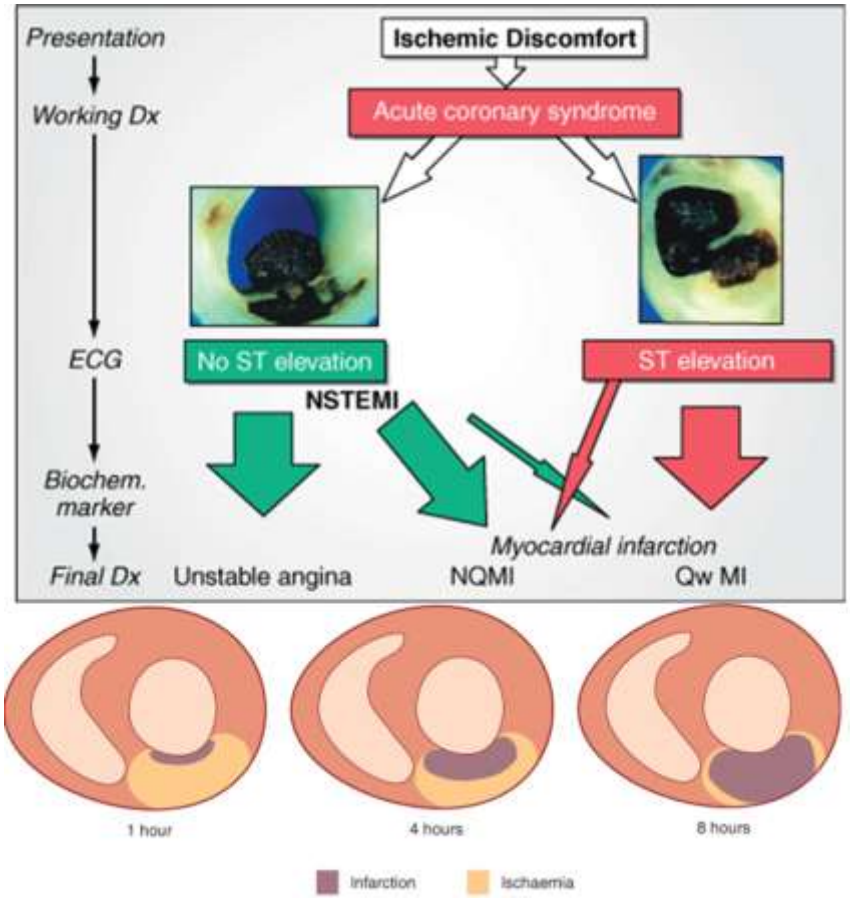
- The imbalance may also be caused by other excessive myocardial oxygen demand in the setting of a stable flow-limiting lesion; acute coronary insufficiency due to vasospastic angina, coronary embolism, coronary arteritis; noncoronary causes (hypotension, severe anemia, hypertension, tachycardia, hypertrophic cardiomyopathy, severe aortic stenosis); nonischemic myocardial injury (myocarditis, cardiac contusion, cardiotoxic drugs); multifactorial causes (stress cardiomyopathy, pulmonary embolism, severe heart failure, sepsis).

content.onlinejacc.org/article.aspx?articleid=1910086

Mechanisms

In Reversible and Irreversible Ischemia Terms 1

- UA develops, until local coronary circulation disturbances cross the time of reversible ischemia.



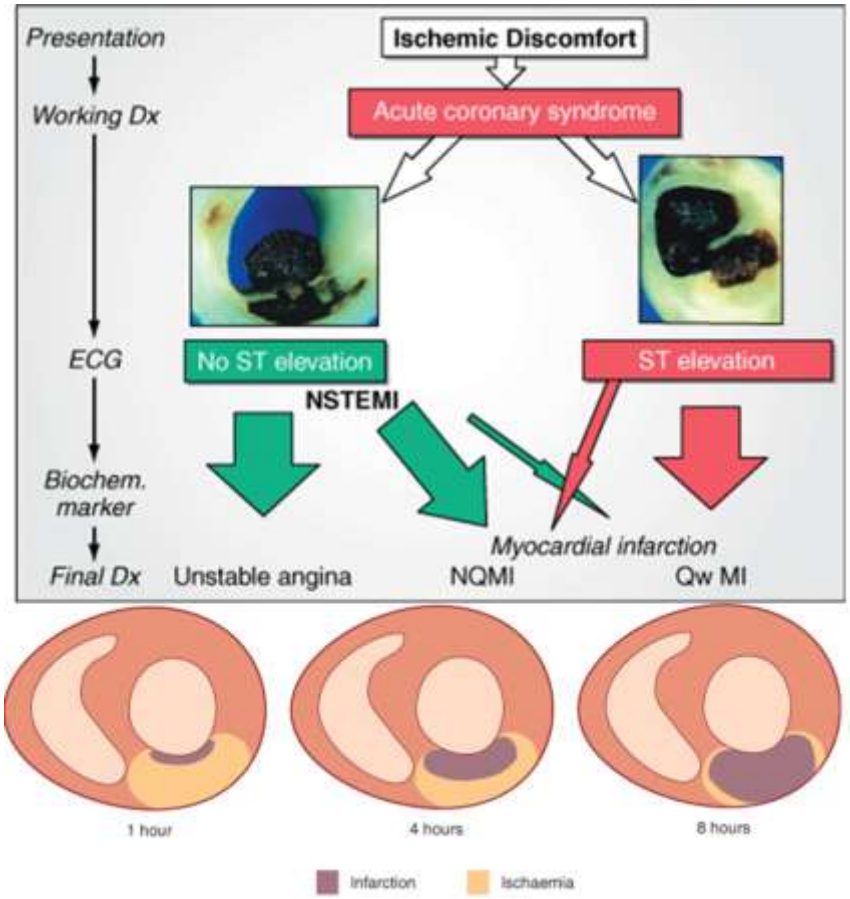
Dx - abbreviation for diagnosis.

medicinembbs.blogspot.com/2011/06/what-is-difference-between-nstemi-and.html

Mechanisms

In Reversible and Irreversible Ischemia Terms 2

- MI develops, when local coronary circulation disturbances cross the time threshold and make the ischemia irreversible.



Dx - abbreviation for diagnosis.

medicinembbs.blogspot.com/2011/06/what-is-difference-between-nstemi-and.html

Mechanisms
Key Moments

- Mechanisms of an ACS were selected by evolution and consist at systemic and local levels
- These mechanisms are aimed at providing most favourable of possible the ACS courses
- Their disturbances build complications of the ACS.

Mechanisms

Systemic Level: 1

- The response of body systems to an ACS is realized through stress and manifests itself as brain mediated sympathetic activation and increased functional activity of a hypothalamo-pituitary an adrenal systems with the change of functions of all target organs.

Mechanisms
Systemic Level: 2

- Course and outcome of the ACS, all other conditions being equal, an adequate organization of stress (eustress) becomes of primary importance
- For STEMI leukocyte reactions are important for the further development of the process:
  - These reactions are triggered by the ejection of leukocytes from the depot to the systemic blood flow

Mechanisms
Systemic Level: 3

- Since the depot mainly contains neutrophils, leukocytosis appears as the shift in cell count, neutrophils are activated and migrate to the infarction zone by positive chemotaxis
- Infarction zone products getting in the blood flow play the role of attractants for them.

Mechanisms

Systemic Level: 4

- The activation of neutrophils appears as hyperenzymemia, higher contents of eicosanoids, leukotrienes in particular, protein carbohydrate complexes, and other biologically active agents
- Stress is changing as the process develops and leukocytosis declines with the leukogram changes
- Granulocyte counts decrease, and their functional activity is suppressed, while agranulocyte counts and their activity increase.

Mechanisms
Systemic Level: 5

- The structural change of the leukogram is the result of controlling effects of infarction zone products getting in the blood flow: neutrophil decay products from the infarction zone are repellents for neutrophils and attractants for agranulocytes.

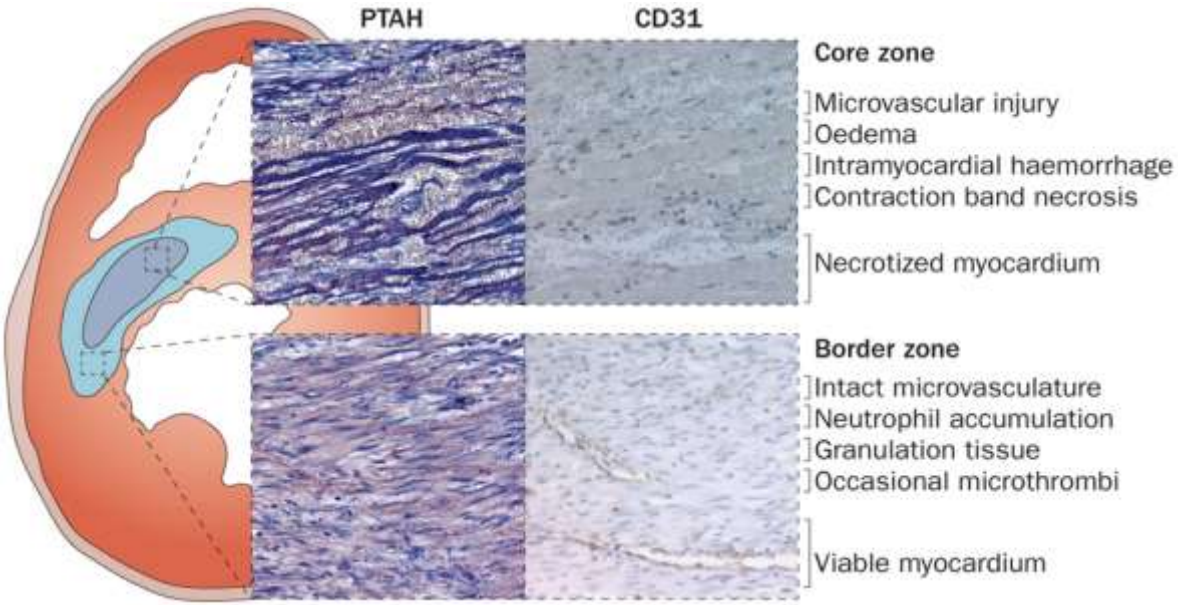
Mechanisms

Systemic Level: 6

- As result an enzyme level in blood falls, while the proteins and protein-carbohydrate complexes content grows.
- These are the systemic manifestations of an inflammation process in the infarction zone
- With the termination of an acute myocardial infarction phase, regulation problems disappear and not a trace remains of the stress.

Mechanisms
Local Level 1

The local level is the heart.

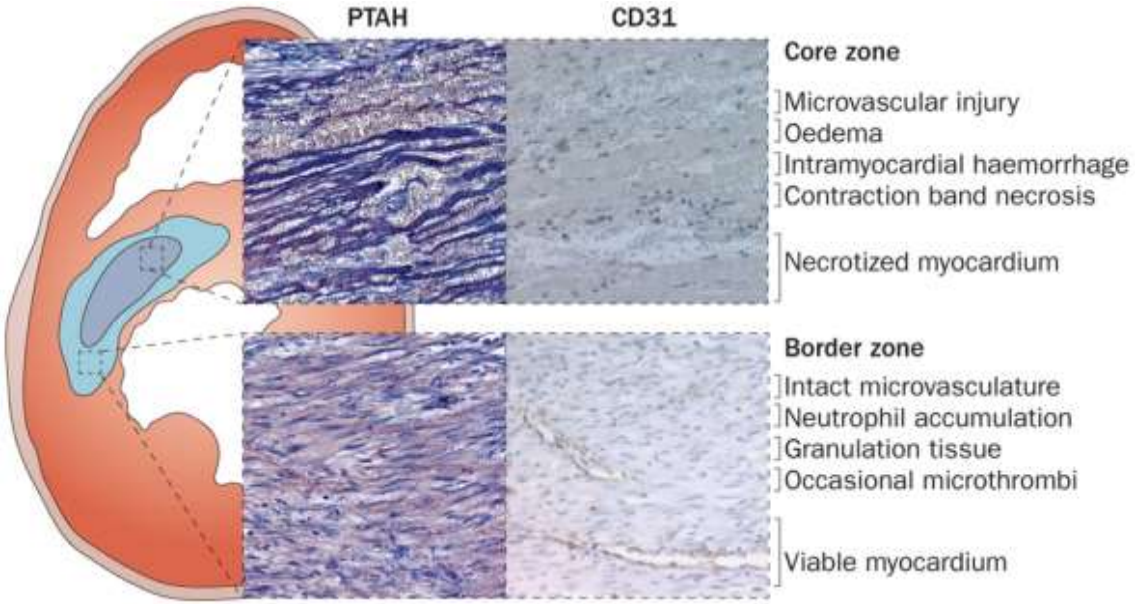


The top panels show the infarction zone and the bottom panels show the border (periinfarction) zone.

im.medicine.karazin.ua//downloads/presentations/Yabluchanskiy_Infarction_Strategy.pdf amjmedsci.org/

Mechanisms
Local Level 2

The components of a pathologic process are not only changes in the infarction zone and recovery of heart shape and size but also adaptive changes of heart biomechanics.



The top panels show the infarction zone and the bottom panels show the border (periinfarction) zone

im.medicine.karazin.ua//downloads/presentations/Yabluchanskiy_Infarction_Strategy.pdf amjmedsci.org/

Mechanisms

Infarction Zone: 1

- All that happens is inflammation: special, aseptic, alterative but still inflammation
- The first one is ischemia, reversible myocardial changes (NSTEMI)
- The transfer to irreversible changes (STEMI) marks the onset of necrosis, and with the transfer from ischemic changes in the infarction zone to the necrotic ones, the inflammation starts in accordance with its traditional scheme.

Mechanisms

Infarction Zone: 2

- The necrotized myocardium undergoes destruction, and decay products are removed through the peri-infarction zone
- The necrotized myocardium is specifically destroyed by neutrophil getting by chemotaxis from blood into the infarction zone and produsing cathepsins
- Their migration rate is rather high, about 2-4 mm per hour and even the largest possible infarction is infiltrated by neutrophils in 6 hours at the most.

Mechanisms

Infarction Zone: 3

- At the same time, fibroblast precursors enter the infarction zone and the recovery process begins
- It is impossible to separate necrotic and reparative processes, to look at them as the individual ones
- They are synchronized and interconnected not only at the level of the infarction zone itself but also at the systemic blood level.

Mechanisms

Infarction Zone: 4

- The result of a natural inflammation course in the infarction zone is the formation of a full-fledged scar in the place of necrosis
- Maturation of a granulation tissue results in its consolidation followed by a decrease in infarction zone sizes
- Depending on conditions, they can decrease by 25% or more.

Mechanisms
Infarction Zone: 5

- I should remind those who want to strongly intervene in the infarction zone that the phenomena occurring there (inflammation, compensatory and adaptive responses) are protective reactions originated as the result of evolution
- We may intervene in these processes but carefully.

Mechanisms
Periinfarction Zone 1

- Systemic mechanisms and the infarction zone are interconnected through the periinfarction zone, first of all, through its microcirculatory bed.
- Wastes from the infarction zone are removed through it, and the products necessary for reparative processes enter there the same way.

Mechanisms
Periinfarction Zone 1

- The larger the infarction–periinfarction zone interface, the better the mutual effect of the infarction zone and systemic control
- The peri-infarction zone cannot be smaller than that required for uncomplicated healing of the infarction.

Mechanisms
Heart Shape and Size 1

- In the case of infarction, a part of a myocardium becomes disabled and its functions should be compensated
- Hypertrophy of an intact myocardium develops
- Healing of the infarction zone accompanied with the consolidation of scar leads to a decrease in its size.

Mechanisms
Heart Shape and Size 2

- The heart shape is remodeled in such a way that its anatomic proportions are restored to favor its pumping functions
- In the best case, the traces of infarction are difficult to reveal, even after thorough investigations.

Mechanisms

Complications: Frequency and Immediate Cause 1

- Acute myocardial infarction is a complex and vulnerable process
- In the most optimistic estimations, its complications are observed somewhere in a quarter of cases, if it is diagnosed and treated correctly.

Mechanisms

Complications: Frequency and Immediate Cause 2

- The immediate cause of complications is inadequate stress or distress
- Have little patience, and we would see how distress is realized as complications.

Mechanisms

Complications: Standard Mechanism 1

- The cause of complications is (hyper reactive, hypo reactive or intermittent) distress
- The mechanism of complications, irrespective of a distress type, is always the same, viz. desynchronization of necrotic and reparative processes.

Mechanisms
Complications: Standard Mechanism 2

- Desynchronization of necrosis and reparation leads to the loss of heart wall strength in the infarction zone with cardiac aneurysm or heart rupture outcomes.

Mechanisms

Complications: Hyperreactive Distress 1

- The result of hyperreactive distress is in intensive and rapid migration of polynuclears to the infarction zone with acceleration of necrosis and destruction of infarcted myocardium and lag behind of reparative processes
- There is the desynchronization.

Mechanisms
Complications: Hyperreactive Distress 2

- Fast destruction of myocardium in the infarction zone has one more consequence, viz. high concentration gradients of all products formed in it along the boundary with the peri-infarct zone with the electronic instability of a heart and as a result, arrhythmia.

Mechanisms

Complications: Hyporeactive Distress 1

- In this the case everything develops slowly.
- Systemic reactions are sluggish or are absent at all
- Thus, there are problems with the infiltration of polynuclears to the infarction zone and the necrotic phase is slow, which leads to the delay of reparative processes and their slow development.

Mechanisms

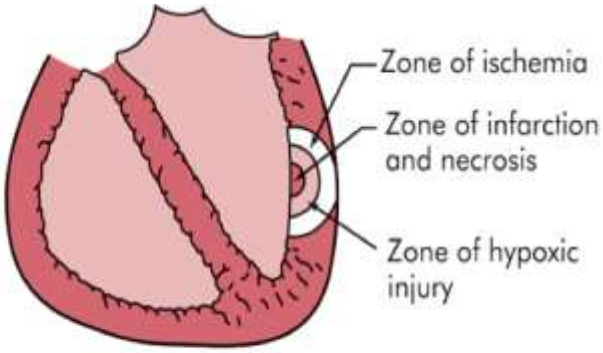
Complications: Hyporeactive Distress 2

- There is the desynchronization again
- Hypo reactive distress with a sluggish disease pattern determines large sizes of aneurysm and as a rule, heart rupture occurs in the thinnest area of aneurysm.

Mechanisms

Complications: Intermittent Distress

Intermittent distress means superposition of the above mechanisms by the formula 'out of the frying-pan into the fire‘ and everything becomes much more dangerous.



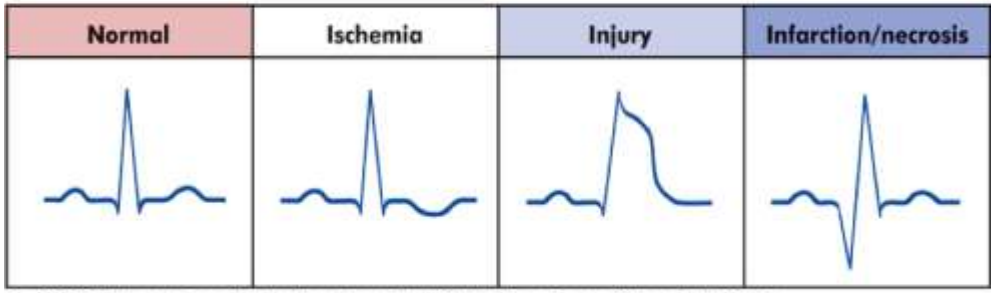


Fig. 23-25. Electrocardiographic Alterations Associated With the Three Zones of Myocardial Infarction

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US MLE TEST

A 70-year-old man presents to the emergency room complaining of left-sided crushing substernal chest pain that began suddenly while he was walking his dog. He denies any past medical history, has not seen a physician recently, and has smoked one pack per day for the past 50 years. Vital signs are: BP 85/50 mmHg, HR 50 bpm, RR 22, T 99.1 deg F. Physical exam shows an obese, nervous man with jugular venous distension and clear lung fields. An electrocardiogram shows ST elevations in II, III, and aVF. Which one of the following medications should not be administered to this patient?

1. Aspirin, 2. Atorvastatin, 3. Nitroglycerin,
4. Clopidogrel, 5. Heparin

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

US MLE TEST

Correct Answer 3: This patient is likely having a right ventricular infarct and therefore should not be given nitrates. Right ventricular myocardial infarctions (RVMI) are usually caused by an occlusion of the proximal right coronary artery. The diagnosis of RVMI can be strongly suspected when hypotension, raised jugular venous pressure (distended neck veins), and clear lung fields are present in a patient whose 12-lead electrocardiogram has findings of an inferior wall infarction as well as ST-elevation in lead V4R. These patients' cardiac output is highly dependent on pre-load, so any drug that limits venous return (nitrates/diuretics), slows heart rate (beta blockers), or decreases contractility (calcium channel blockers) are relatively contraindicated.
Incorrect Answers:
1, 2, 4, & 5: The other drug choices are appropriate for use in right and left ventricular infarcts and confer a mortality benefit.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

Classification

International Classification of Diseases (ICD) 1

Chapter IX
Diseases of the circulatory system
(I00-I99)

I20-I25 Ischemic heart diseases

I20.0 Unstable angina: Angina (crescendo, de novo effort, worsening effort), Intermediate coronary syndrome, Preinfarction syndrome

I21 ST elevation (STEMI) and non-ST elevation (NSTEMI) myocardial infarction

Classification

International Classification of Diseases (ICD) 2

Chapter IX
Diseases of the circulatory system
(I00-I99)

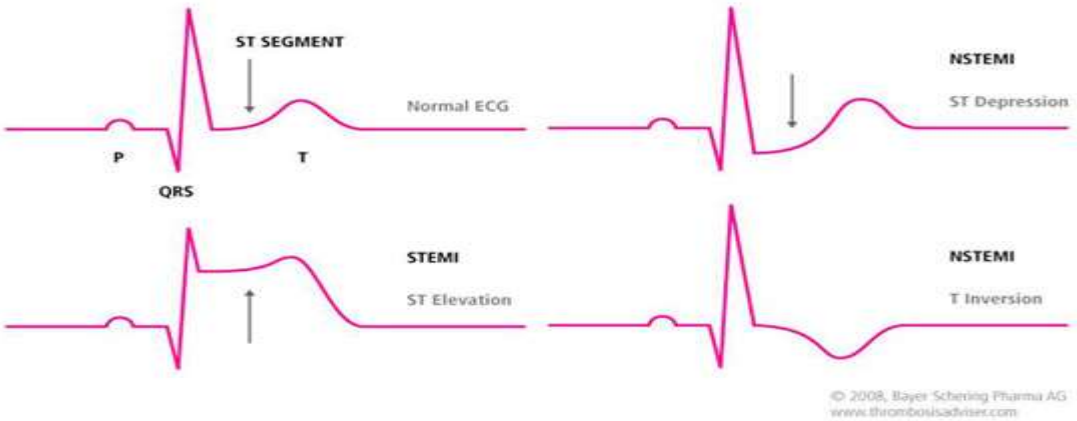
I20-I25 Ischemic heart diseases

123 Certain current complications following ST elevation (STEMI) and non-ST elevation (NSTEMI) myocardial infarction (within the 28 day period)

124 Other acute ischemic heart diseases

Classification

Acute Coronary Syndromes Types



- STEMI - occlusive thrombus - ST elevation (and Q waves) - Cardiac Enzyme elevation present
- NSTEMI - non-occlusive thrombus - NO ST/Q - Cardiac Enzyme elevation present
- UA - non-occlusive thrombus - NO ST/Q - Cardiac Enzyme elevation absent.

medicinembbs.blogspot.com/2011/06/what-is-difference-between-nstemi-and.html hrombosisadviser.com/en/acs/a-leading-cause-of-mortality

Classification

Myocardial Infarction

- Anatomic perspective: transmural, nontransmural (subendocardial, subepicardial)
- Localization: anterior, lateral, posterior, septal, inferior, right ventricle
- Clinical periodization: acutest (UA), acute (predominance of necrotic processes), subacute (predominance of reparative processes), postinfarction (consolidation of postinfarction scar)
- Course: noncomplicated, complicated (hyperreactive, hyporeactive).

metalyse.com/myocardial_infarction/types.html

Clinical Investigation
Symptoms 1

- The cardinal symptom is chest pain, experienced as tightness around the chest and radiating to the left arm and the left angle of the jaw
- This may be associated with diaphoresis (sweating), nausea and vomiting, as well as shortness of breath.

en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

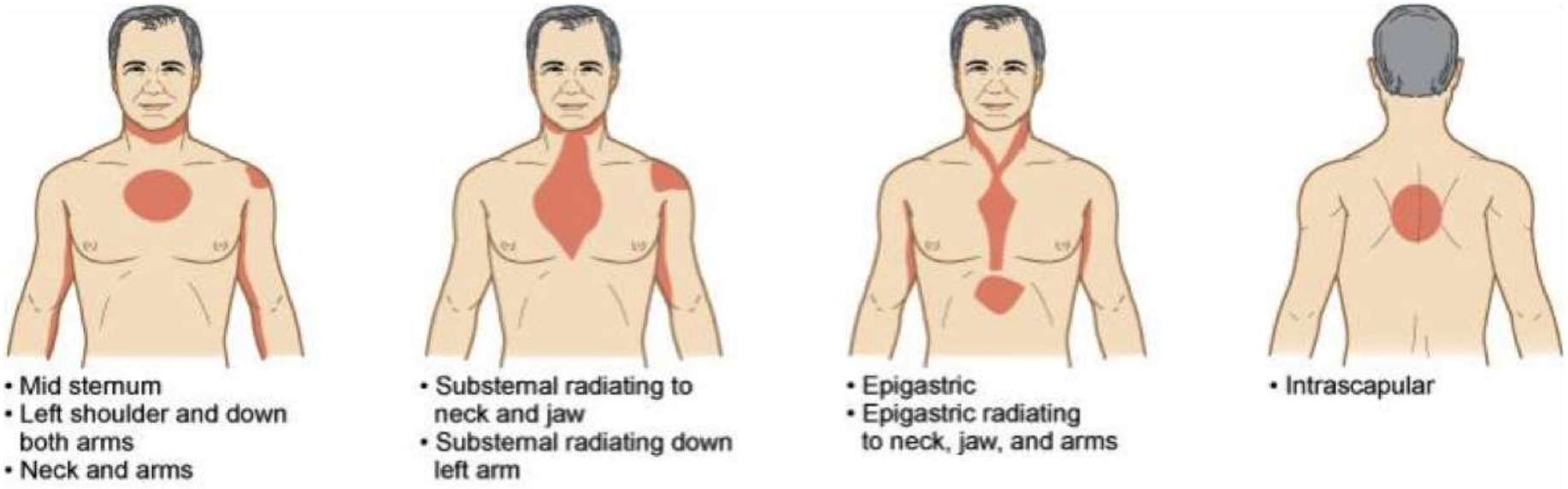
Clinical Investigation
Symptoms 2

- In many cases, the sensation is "atypical", with pain experienced in different ways or even being completely absent (which is more likely in female patients and those with diabetes)
- Some may report arrhythmia, anxiety or a sense of impending doom (angor animi) and a feeling of being acutely ill
- The description of the chest discomfort as a pressure has little utility in aiding a diagnosis as it is not specific for ACS.

en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

Clinical Investigation

Common Locations and Patterns of Chest Pain 1



- The characteristics of discomfort-related to chest pain may be divided into four categories: location, character, duration and relationship to exertion and other exacerbating or relieving factors.

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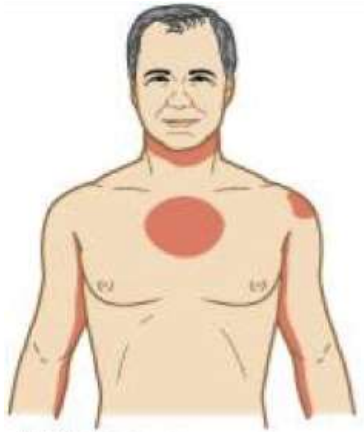
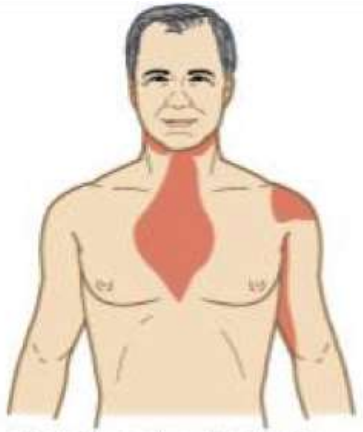
Clinical Investigation

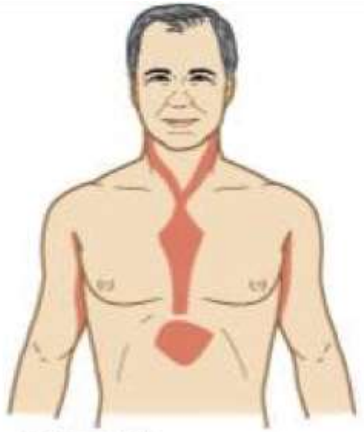
Common Locations and Patterns of Chest Pain 2



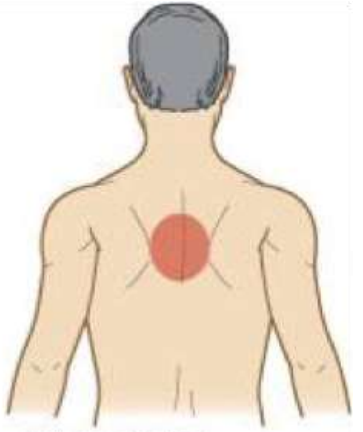
- Mid sternum
- Left shoulder and down both arms
- Neck and arms



- Substernal radiating to neck and jaw
- Substernal radiating down left arm



- Epigastric
- Epigastric radiating to neck, jaw, and arms



- Intrascapular

- The discomfort caused by myocardial ischemia is usually located in the chest, near the sternum, but may be felt anywhere from the epigastrium to the lower jaw or teeth, between the shoulder blades or in either arm to the wrist and fingers.

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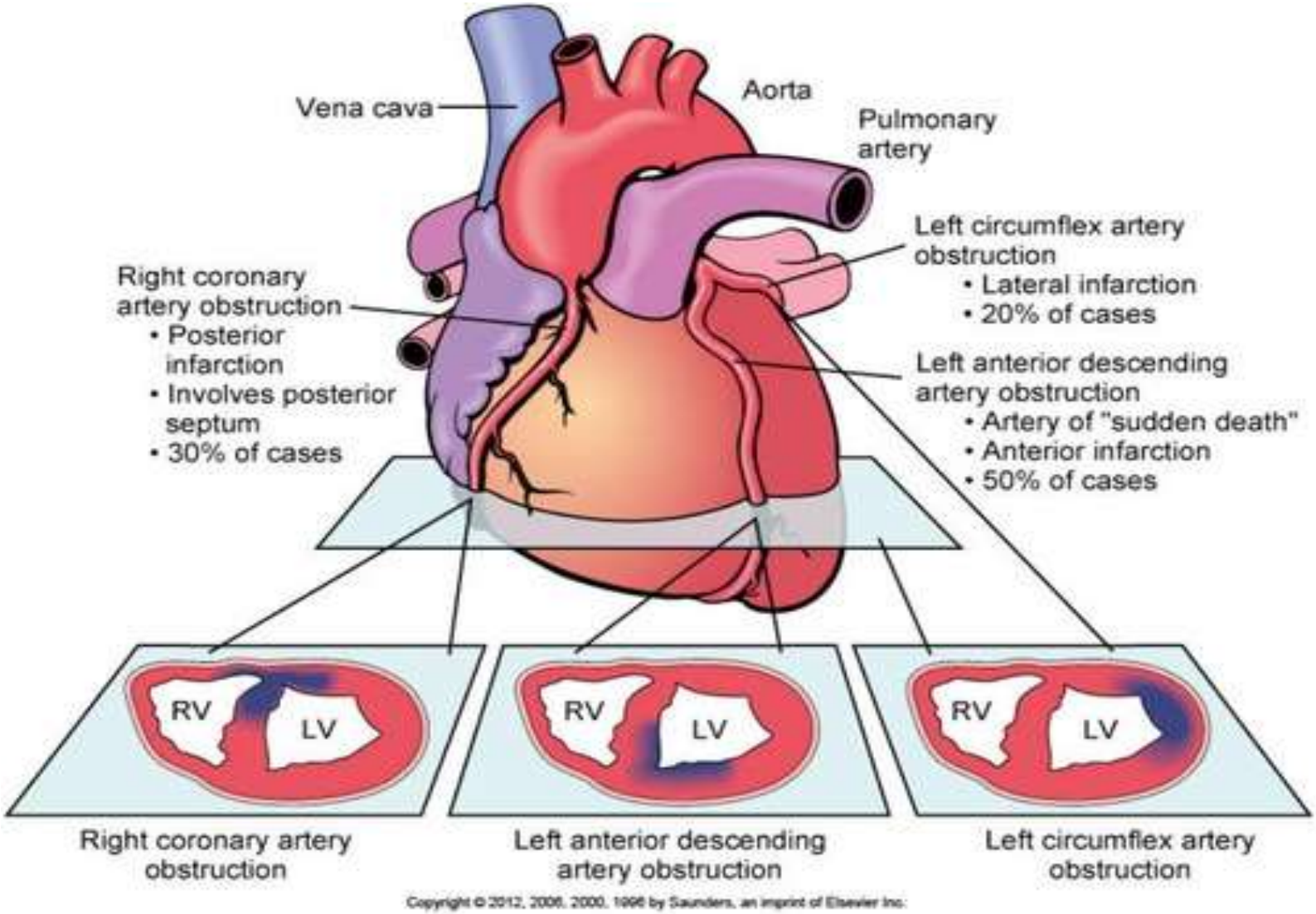
Clinical Investigation

Distribution of ACS



quizlet.com/15191588/study-2-flash-cards/

Clinical Investigation
History 1

- The history should include any current symptoms and a complete inventory of comorbid conditions
- An inventory of cardiac risk factors, and a complete family history are essential components
- The history should also include information about the character and location of discomfort, radiation of discomfort, associated symptoms, and precipitating, exacerbating, or alleviating factors.

clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/coronary-artery-disease/

Clinical Investigation
History 2

- The importance of the family history should not be underestimated
- A detailed assessment, particularly of first-degree relatives for the presence of coronary artery disease and age of diagnosis is imperative when evaluating a patient's risk factor profile.

clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/coronary-artery-disease/

Clinical Investigation
Physical Examination 1

- The results of the physical examination of a patient may be entirely normal
- The presence of multiple risk factors or atherosclerosis symptoms increases the likelihood that a chest pain is related to myocardial ischemia
- Evaluation should include measurements of blood pressure, the ankle-brachial index, cardiac and carotid arteries auscultation for bruits.

clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/coronary-artery-disease/

Clinical Investigation
Physical Examination 2

- Examination of the chest wall, neck, and shoulders for deformities and tenderness may be helpful in diagnosing musculoskeletal chest discomfort
- The physical examination may help identify comorbid conditions that impact therapeutic risk and decision making.

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Clinical Investigation

Myocardial Infarction versus Stable Angina Comparison Chart 1

Symptoms	Myocardial Infarction	Stable Angina
Occurren-ce of pain	Occurs at any time	Occurs due to physical or emotional stress
Modality of pain	With damage to the heart and usually described as severe, steady, and crushing	With no damage to the heart

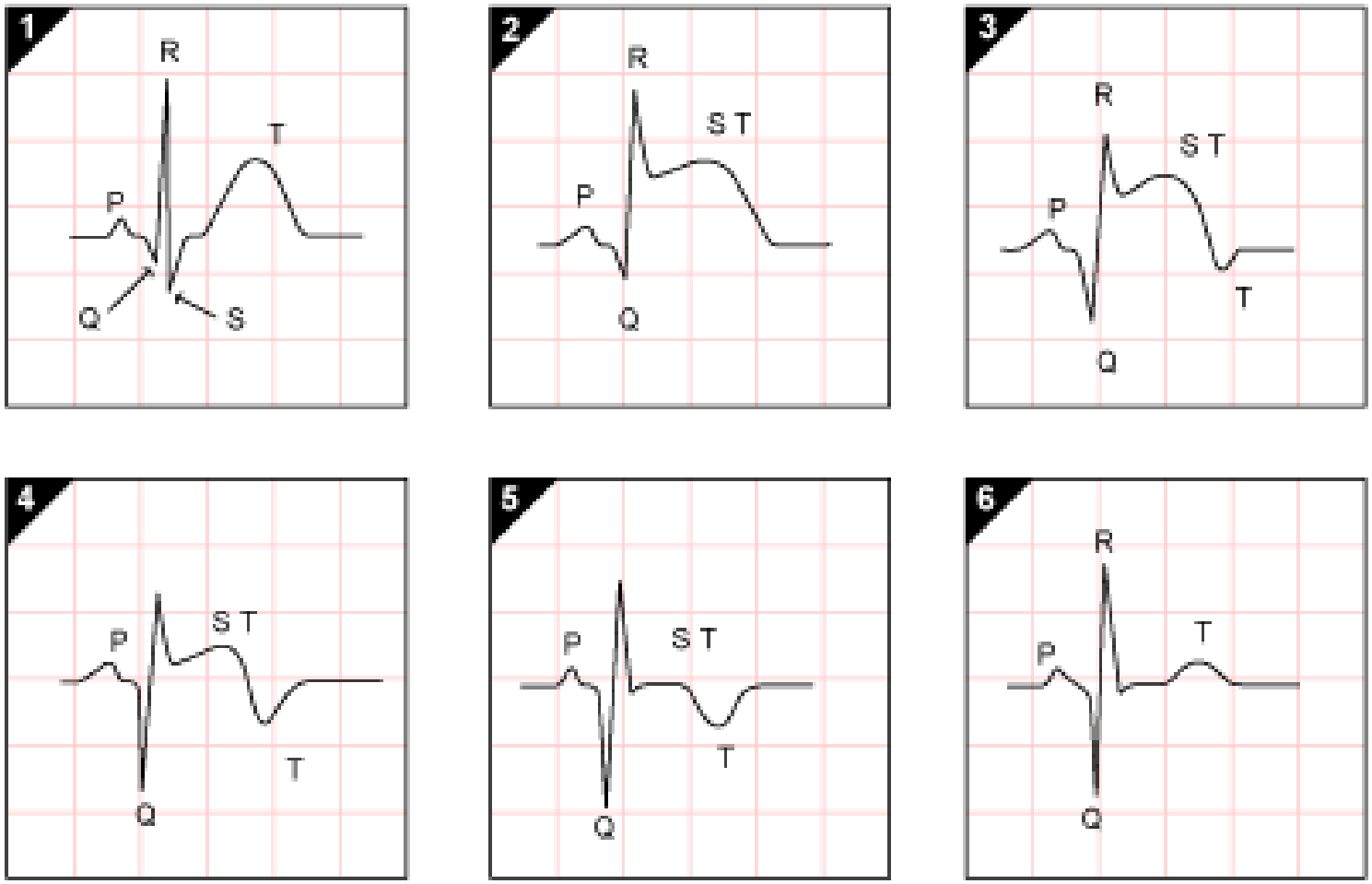
Clinical Investigation

Myocardial Infarction versus Stable Angina Comparison Chart 2

Symptoms	Myocardial Infarction	Stable Angina
Outcome	May be fatal	Usually not fatal
Relieving factors of pain	Symptoms persists after 15 min. and not relieved by rest or nitro	Symptoms relieved by rest or nitro within 10-15 min.
Duration of pain	Usually lasts for more than 15 min.	Usually for less than 15 min.; discomfort is transient, lasting 3-5 min.

Clinical Investigation

Sequence of Changes in evolving noncomplicated MI



nottingham.ac.uk/nursing/practice/resources/cardiology/acs/changes.php

Clinical Investigation
(ECG' Localizing of Myocardial Injury)

I **LATERAL**	aVR	V1 **SEPTAL**	V4 **ANTERIOR**
II **INFERIOR**	aVL **LATERAL**	V2 **SEPTAL**	V5 **LATERAL**
III **INFERIOR**	aVF **INFERIOR**	V3 **ANTERIOR**	V6 **LATERAL**

ems.sluhn.org/images/stemi_alert_opt.png

Clinical Investigation

Complications 1

- Complications may occur immediately following the heart attack, or may need time to develop
- Acute complications may include heart failure; aneurysm of the left ventricle myocardium; ventricular septal rupture or free wall rupture; mitral regurgitation, in particular if the infarction causes dysfunction of the papillary muscle; Dressler's syndrome; and abnormal heart rhythms, such as ventricular fibrillation, ventricular tachycardia, atrial fibrillation, and heart block.

en.wikipedia.org/wiki/Myocardial_infarction

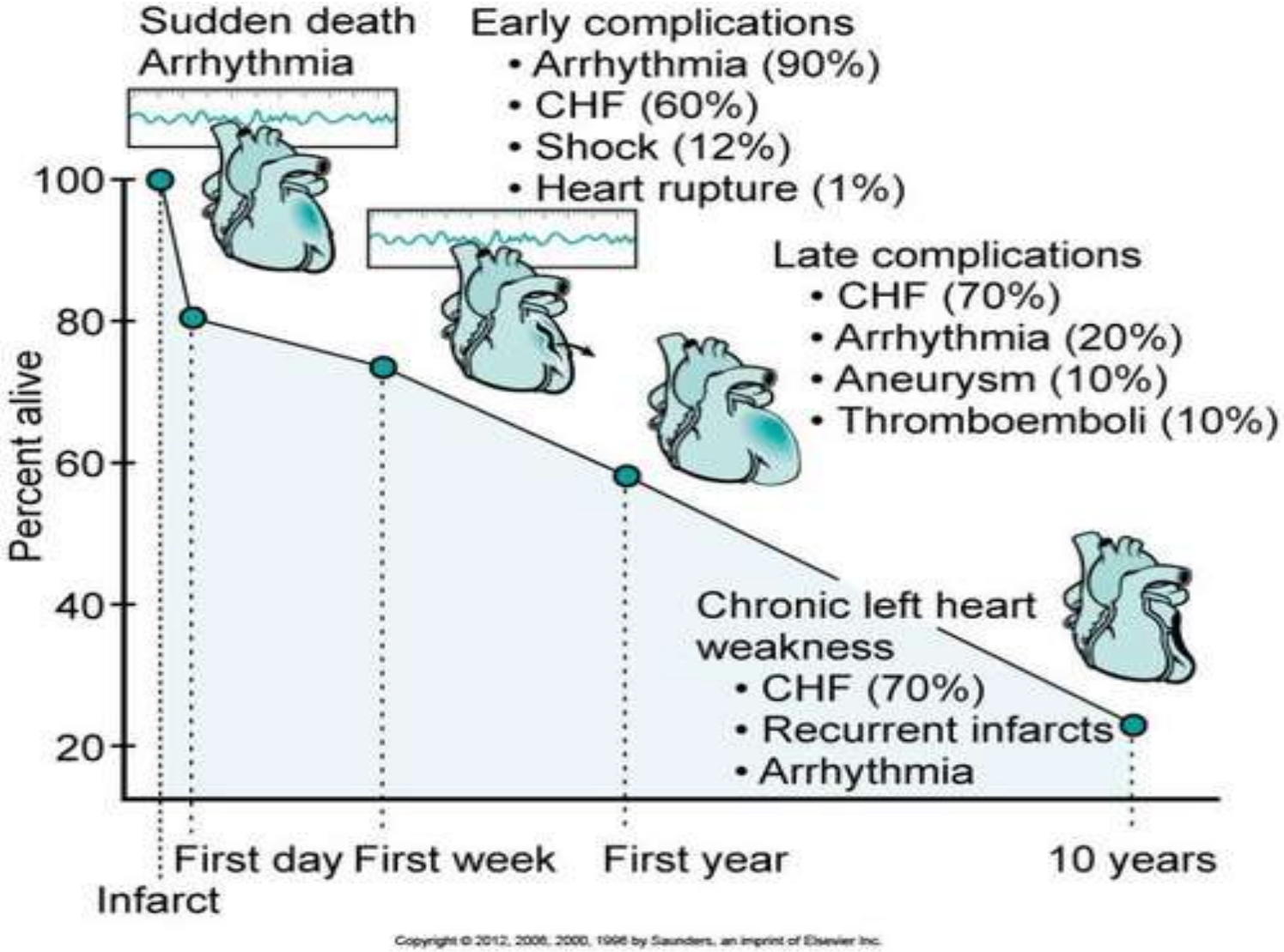
Clinical Investigation
Complications 2

- Longer-term complications include heart failure, atrial fibrillation, and an increased risk of a second MI.

en.wikipedia.org/wiki/Myocardial_infarction

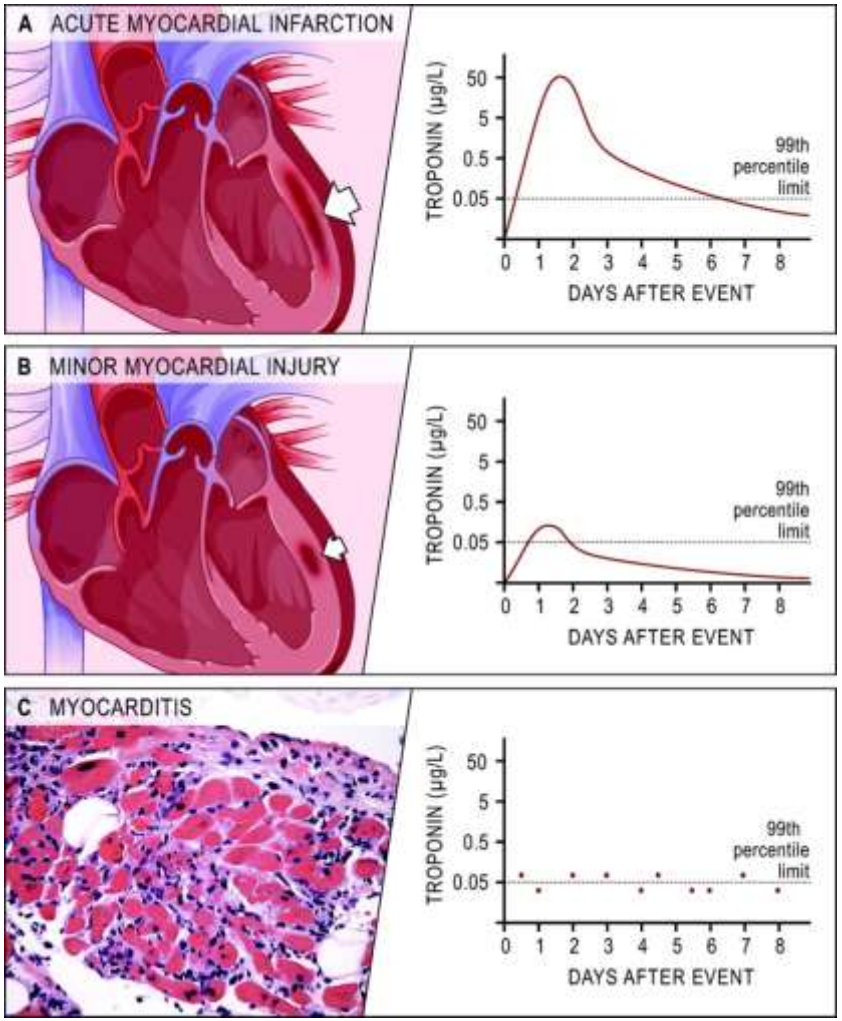
Clinical Investigation

Outcome of ACS



Diagnosis

- Blood tests: cardiac biomarkers; WBC; cholesterol and C-reactive protein levels
- Electrocardiogram (ECG)
- B-mode, Doppler and intravascular ultrasound
- Vascular catheterization and angiogram
- Other imaging tests.



en.wikipedia.org/wiki/Coronary_artery_disease#Diagnosis afghanheart.files.wordpress.com/2013/04/troponins.jpg

US MLE TEST

A 56-year-old male with a history of hypertension and type II diabetes presents to the emergency department with a complaint of crushing chest pain. The pain began 30 minutes ago while mowing his lawn. He denies any radiation of the pain into his arms or neck. An EKG is obtained as is shown in Figure A. Which of the following is the strongest predictor of this patient's long-term prognosis from his presenting condition?

1. Patient age, 2. Medical comorbidities, 3. Maximum cardiac troponin T value measured, 4. Time to initiation of aspirin therapy, 5. Time to restoration of coronary blood flow.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

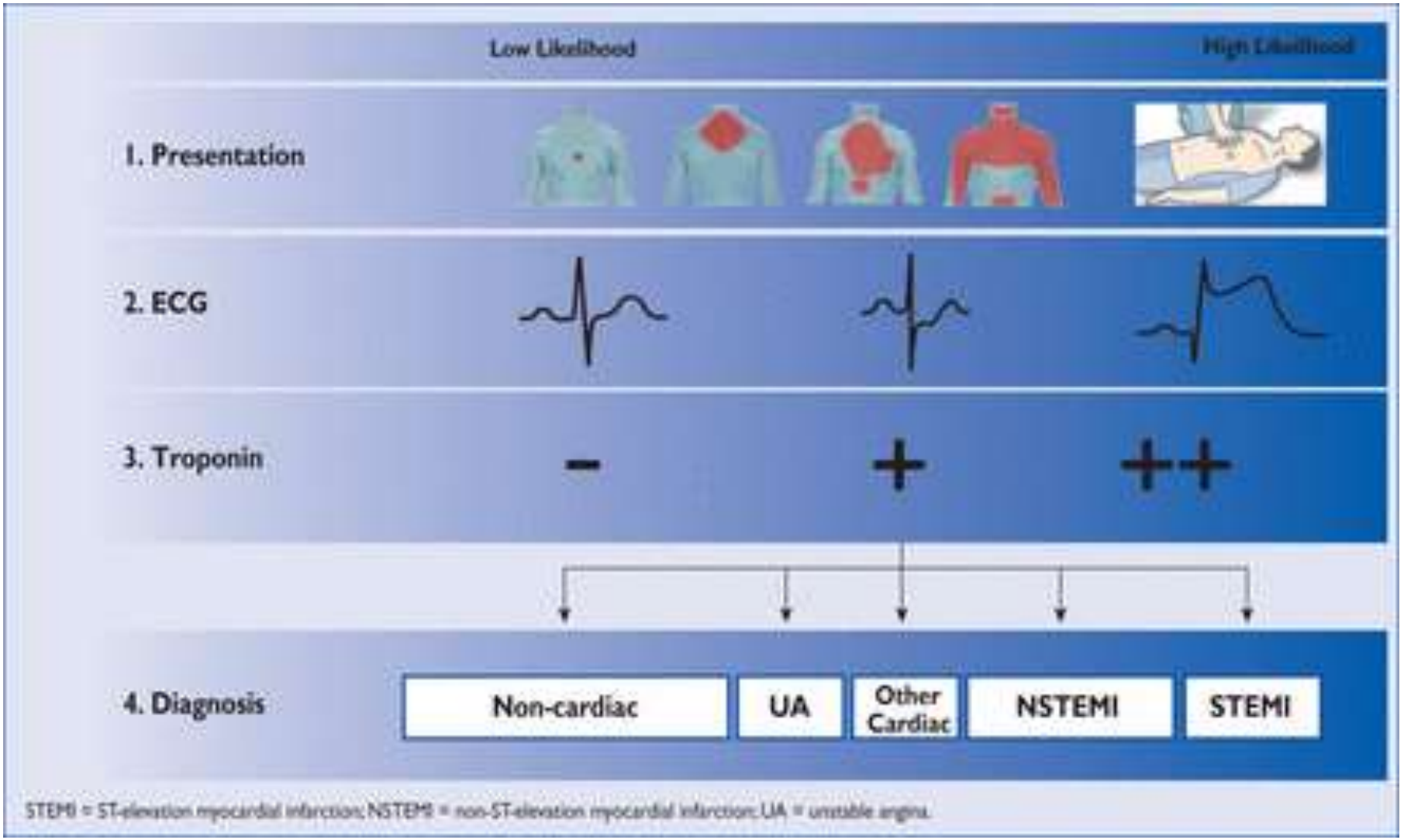
US MLE TEST

Correct Answer 5: This patient is suffering from an ST-elevation myocardial infarction (STEMI). Time to restoration of coronary blood flow following a myocardial infarction is the strongest predictor of long-term prognosis for these patients.
1,2,4: Patient age, medical comorbidities, and time to giving aspirin may all affect patient outcomes after MI; however, time to restoration of coronary blood flow is the strongest predictor of long-term prognosis., 3: The magnitude of troponin elevation has been shown to affect patient outcomes; however, time to restoration of coronary perfusion is a stronger predictor of patient prognosis.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

Diagnosis

(Initial Assessment of Patients with Suspected ACS)



eurheartj.oxfordjournals.org/content/37/3/267

Diagnosis

Criteria for Diagnosis of MI 1

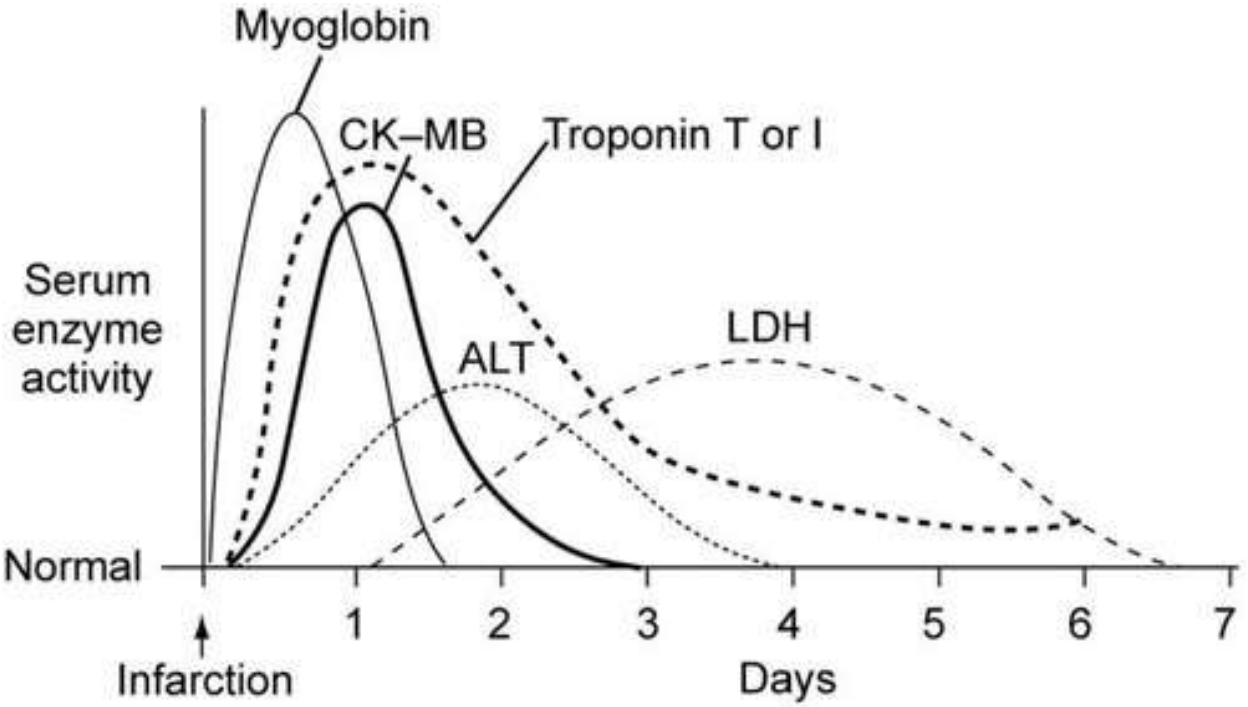
A combination of criteria is required to meet the diagnosis of MI, namely the detection of an increase and/or decrease of a cardiac biomarker, preferably high-sensitivity cardiac troponin, with at least one value above the 99th percentile of the upper reference limit and at least one of the following:

Diagnosis

Criteria for Diagnosis of MI 2

One of the following: symptoms of ischemia; new or presumed new significant ST-T wave changes or left bundle branch block on 12-lead ECG; development of pathological Q waves on ECG; imaging evidence of new or presumed new loss of viable myocardium or regional wall motion abnormality; intracoronary thrombus detected on angiography or autopsy.

Diagnosis
(Cardiac Biomarkers)



Biomarkers related to necrotic processes in myocardial infarction zone.

Diagnosis

Indications for Measurement of Troponins 1

- Cardiac troponin I and T have displaced myoglobin and creatine kinase-MB as the preferred markers of myocardial injury
- Serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days
- Troponin levels may not be detectable for six hours after the onset of myocardial cell injury. The most sensitive *early marker* for myocardial infarction is myoglobin.

patient.info/doctor/cardiac-enzymes-and-markers-for-myocardial-infarction

Diagnosis

Indications for Measurement of Troponins 2

- Troponin levels should be measured at presentation and again 10-12 hours after the onset of symptoms. When there is uncertainty regarding the time of symptom onset, troponin should be measured at twelve hours after the presentation.
- The risk of death from an ACS is directly related to troponin level and patients with no detectable troponins have a good short-term prognosis.

patient.info/doctor/cardiac-enzymes-and-markers-for-myocardial-infarction

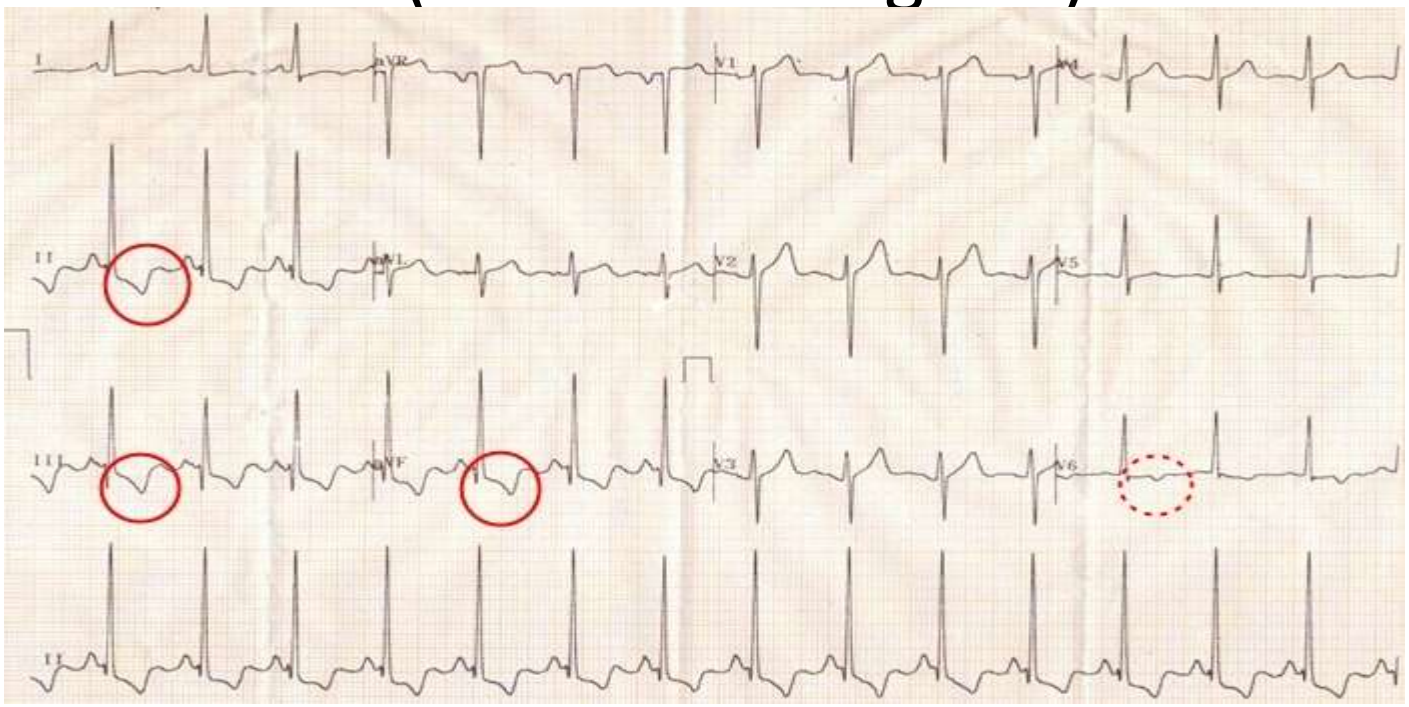
Diagnosis

Indications for Measurement of Troponins 3

- Elevated troponin levels can occur in patients without an ACS and are associated with adverse outcomes in many other clinical situations, including congestive heart failure, sepsis, acute pulmonary embolism and chronic kidney disease. Other cardiac causes include myocarditis and aortic dissection.

patient.info/doctor/cardiac-enzymes-and-markers-for-myocardial-infarction

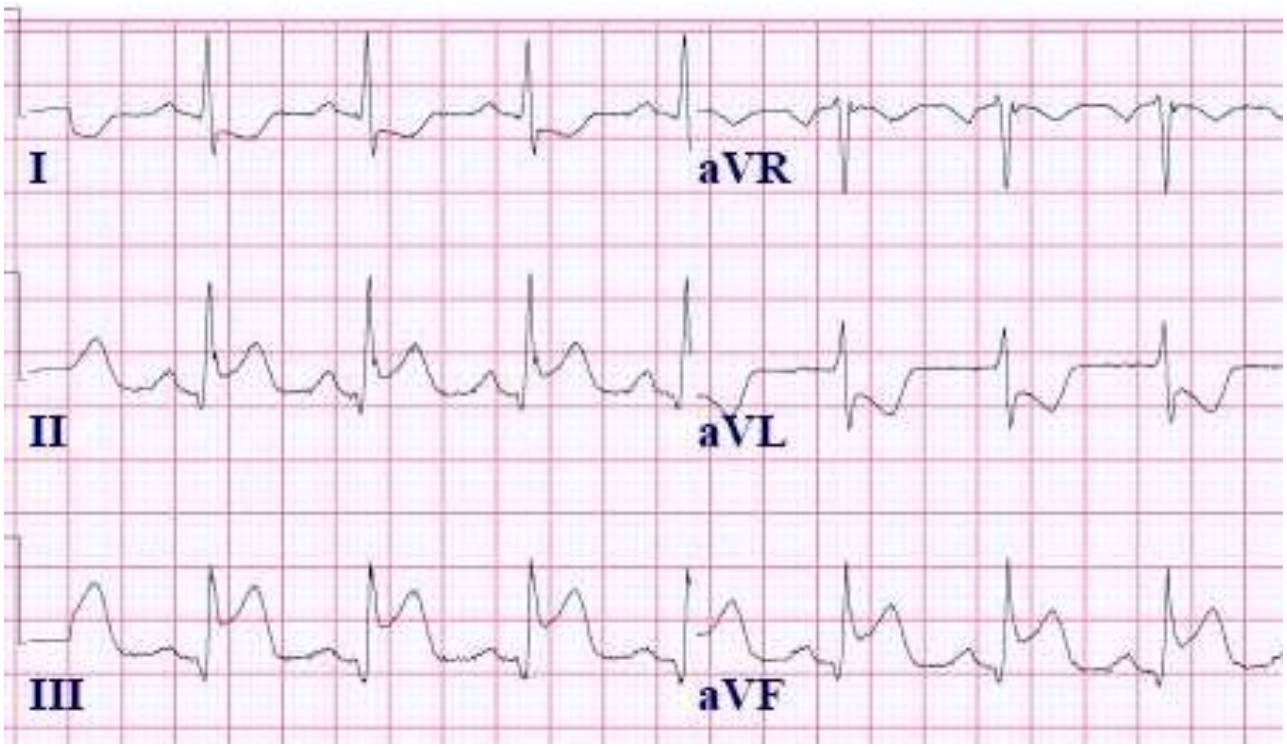
Diagnosis
(Electrocardiogram)



There are ST and T changes in leads II, III, aVF (solid red circles) – it is a suspicion of ischemia of the inferior myocardial wall. Similar change is outlined in the lead V6 (dashed circle).

health-tutor.com/simple-cardiac-ischemia-ecg.html

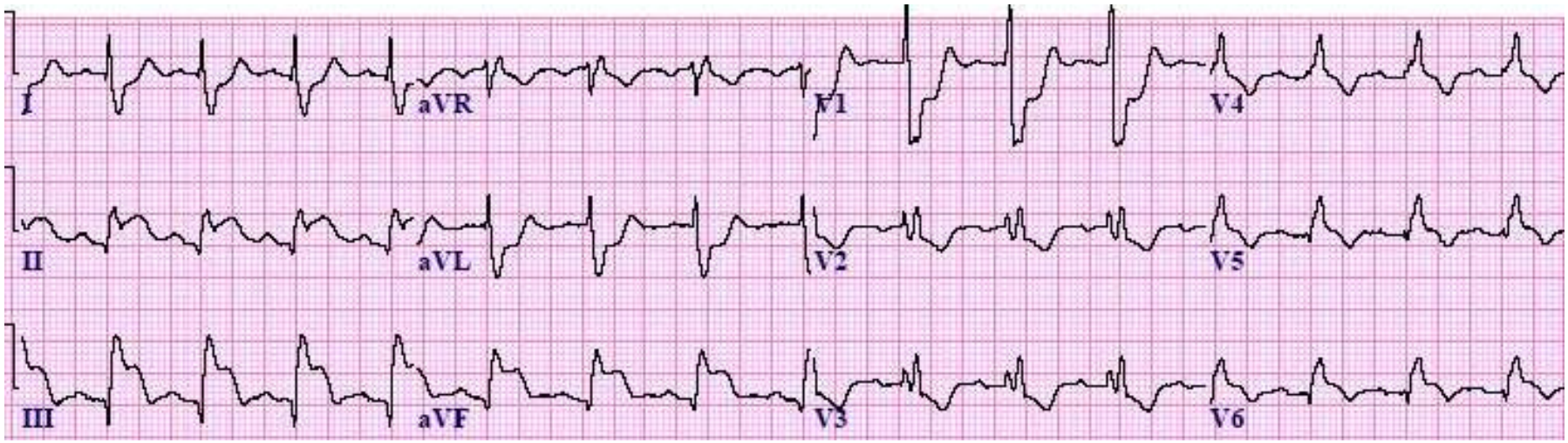
Diagnosis
(Electrocardiogram)



Inferior STEMI in acutest phase.

healio.com/cardiology/learn-the-heart/blogs/stemi-mi-ecg-pattern

Diagnosis
(Electrocardiogram)



An inferior STEMI with a right bundle branch block on the ECG. Ehe reciprocal depression in lead I and aVL.

healio.com/cardiology/learn-the-heart/blogs/stemi-mi-ecg-pattern

Diagnosis

(Criteria of Differentiation Noncomplicated and Complicated MI)

Criterion	Type of acute myocardial infarction		
	Noncomplicated with eureactive stress	Complicated with hyperreactive distress	Complicated with hyporeactive distress
Blood leukocytes, E 9 1/l.: 1st day results and further dynamics	7-11, rapid decrease	>11, slow decrease	<7, increase, then slow decrease
Shift of leukocytes, arb. units: 1st day results and further dynamics	3.0-6.5, rapid decrease	>6.5, slow decrease	<3.0, increase, then slow decrease
Time of maximum activity of enzymes, hrs	16-22, rapid decrease	>16, slow decrease	>22, slow decrease
Initial period of protein-carbohydrate complex growth, days	1-2	>2	>2
Period of reaching maximum protein-carbohydrate complex concentrations, days	4-5	>5	>5
Interval between maxima of enzymes and protein-carbohydrate complexes, days	>4	>6	>6
Return of the ST segment on ECG to the isoline, hrs	>36	>36	>36

meteopathy.com/zdorovye-i-bolezn/my-myocardial-infarction-phylosophy/2/

Treatment

Prehospital Care and Initial Management 1

- All patients should be managed as if the pain is ischemic in origin, unless clear evidence to the contrary is established
- Specific care includes intravenous access; supplemental oxygen; pulse oximetry; immediate administration of aspirin; nitroglycerin for active chest pain; telemetry and prehospital ECG, if available.

Treatment

Prehospital Care and Initial Management 2

- Additional objectives include adequate analgesia (morphine); pharmacologic reduction of excessive sympathoadrenal and vagal stimulation; treatment of ventricular arrhythmias; support of cardiac output, and respiration
- Prehospital fibrinolytic therapy (tissue-type plasminogen activator) , aspirin, and heparin may be given by paramedics, as guided by electrocardiographic findings, within 90 minutes of the onset of symptoms.

emedicine.medscape.com/article/155919-treatment#d5

Treatment

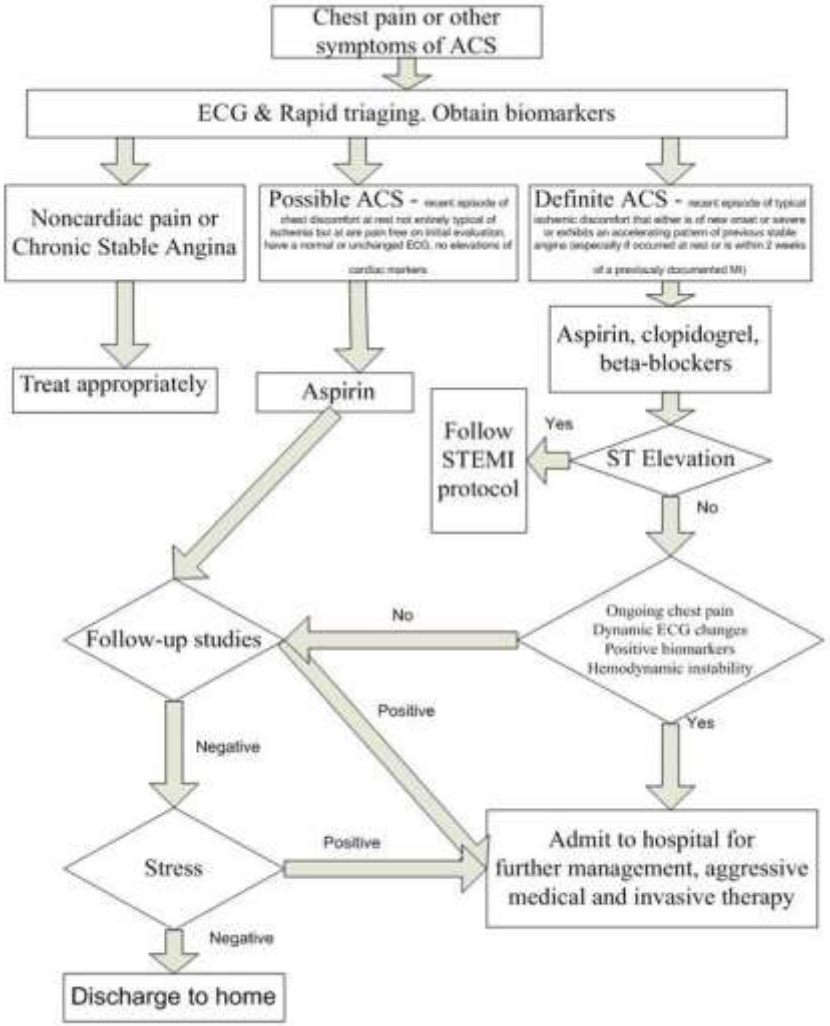
Emergency Department Care and In-Hospital Management

- Triage and evaluation: because ACS is a spectrum of conditions, initial evaluation to establish a working diagnosis is crucial, as this will dictate management owing to some differences in management steps and timelines for each component of the ACS spectrum
- Initial management: restoration of the balance between oxygen supply and demand to prevent further ischemia; pain relief, prevention and treatment of complications.

emedicine.medscape.com/article/155919-treatment#d5

Treatment

Suggested Algorithm for Triaging Patients with Chest Pain



emedicine.medscape.com/article/1910735-overview#showall

Treatment
Presumed ACS and NSTEMI 1

- The accepted management is empirical treatment with aspirin, a second platelet inhibitor such as clopidogrel, and heparin (usually a low-molecular weight heparin such as enoxaparin), with intravenous glyceryl trinitrate and opioids if the pain persists
- A blood test is generally performed for cardiac troponins twelve hours after onset of the pain and if this is positive, coronary angiography is typically performed on an urgent basis

/en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

Treatment
Presumed ACS and NSTEMI 1

- If there is no evidence of ST segment elevation on the electrocardiogram, delaying urgent angioplasty until the next morning is not inferior to doing so immediately.

/en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

Treatment
STEMI

- If the ECG confirms changes suggestive of MI, thrombolytics may be administered or primary coronary angioplasty may be performed
- The time frame for door-to-needle thrombolytic administration should be within 30 minutes, whereas the door-to-balloon Percutaneous Coronary Intervention (PCI) time should be less than 90 minutes.

/en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

Treatment

Absolute and Relative Contraindications to Fibrinolytic Therapy

Absolute Contraindications	Relative Contraindications
Prior intracranial hemorrhage Structural cerebral vascular lesion Intracranial neoplasm Ischemic stroke within the past 3 months (except for acute stroke within 4.5 hours) Suspected aortic dissection Active bleeding or bleeding diathesis (excluding menses) Significant closed-head or facial trauma within 3 months Intracranial or intraspinal surgery within 2 months Severe uncontrolled hypertension (unresponsive to emergency therapy) For streptokinase (no longer marketed in the US): Prior treatment within previous 6 months	Chronic, severe, poorly controlled hypertension Systolic pressure >180 mm Hg or diastolic pressure >110 mm Hg History of prior ischemic stroke >3 months Dementia Intracranial pathology Traumatic or prolonged CPR (>10 minutes) Recent (within 2-4 weeks) internal bleeding Noncompressible vascular punctures Pregnancy Active peptic ulcer disease Current use of anticoagulants For streptokinase: prior exposure (>5 days previously) or prior allergic reaction to these agents

CPR = cardiopulmonary resuscitation

emedicine.medscape.com/article/155919-treatment#d25

Treatment

Fibrinolytic Agents

Fibrinolytic Agent	Dose	Fibrin Specificity	Antigenic	Patency Rate
Streptokinase (no longer marketed in the US)	1.5 million units IV given over 30–60 min	No	Yes	60%–68%
Tenecteplase (TNK-tPA)	30 mg for weight <60 kg 35 mg for 60–69 kg 40 mg for 70–79 kg 45 mg for 80–89 kg 50 mg for >90 kg	++++	No	85%
Reteplase (rPA)	10-U IV boluses given 30 min apart	++	No	84%
Alteplase (tPA)	Bolus 15 mg followed by infusion 0.75 mg/kg for 30 min (maximum 50 mg), then 0.5 mg/kg (maximum 35 mg) over the next 60 min; total dose not to exceed 100 mg.	++	No	73%-84%

IV = intravenous; rPA = recombinant human tissue plasminogen activator; STEMI = ST-elevation myocardial infarction; tPA = tissue plasminogen activator; US = United States of America.

emedicine.medscape.com/article/155919-treatment#d25

US MLE TEST

An intern covering the night shift at an ICU is called to the room of a 75-year-old hypotensive man. The patient experienced an anterolateral myocardial infarction four days prior and underwent cardiac catheterization with successful revascularization of his left anterior descending artery. He was doing well prior to 45 minutes ago when he began to complain of weakness, fatigue, and renewed chest pain. A nurse obtained his blood pressure, which was 80/40, and was not able to auscultate his heart sounds due to muffling. The intern found the patient lethargic and confused and noted distended neck veins. He checked for pulsus paradoxus and noted a 15 mmHg difference in the systolic pressure during respiration. He runs out of the room to gather supplies. Which of the following emergent therapies is warranted?

1. Pericardiocentesis, 2. Repeat cardiac catheterization , 3. Mitral valve repair, 4. Intra-aortic balloon pump, 5. Cardioversion.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

US MLE TEST

Correct Answer 1: This man has pulsus paradoxus and is experiencing cardiac tamponade caused by an anterior wall rupture and requires emergent pericardiocentesis.
Incorrect Answers:
2: This patient is not having another myocardial infarction and so would not benefit from a repeat cardiac catheterization., 3: This patient did not have a papillary muscle rupture and so would not benefit from a mitral valve repair., 4: An intra-aortic balloon pump is not indicated for a free wall rupture emergently., 5: There is no indication that the patient is having an issue with his rhythm and so would not benefit from cardioversion.

https://www.medbullets.com/step1-endocrine/9019/pheochromocytoma

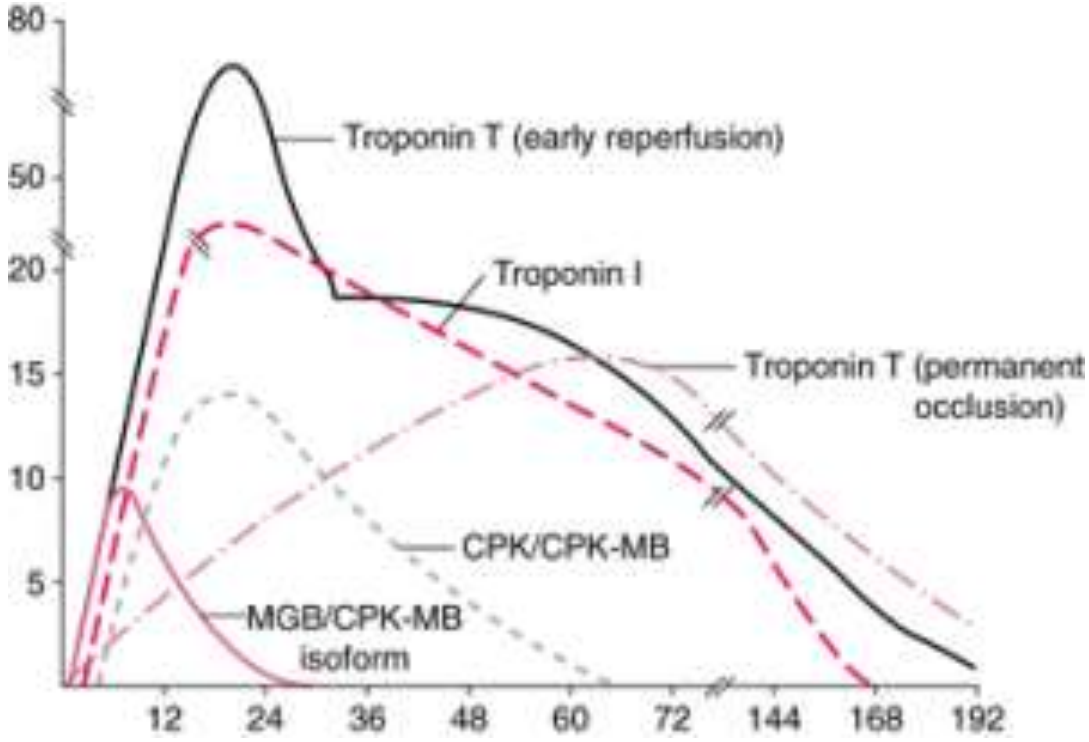
Treatment
Cardioprotective Medications

- Inhibitors of the renin-angiotensin-aldosterone (RAA) system
- Beta blockers
- Statins

en.wikipedia.org/wiki/Acute_coronary_syndrome#Signs_and_symptoms

Treatment

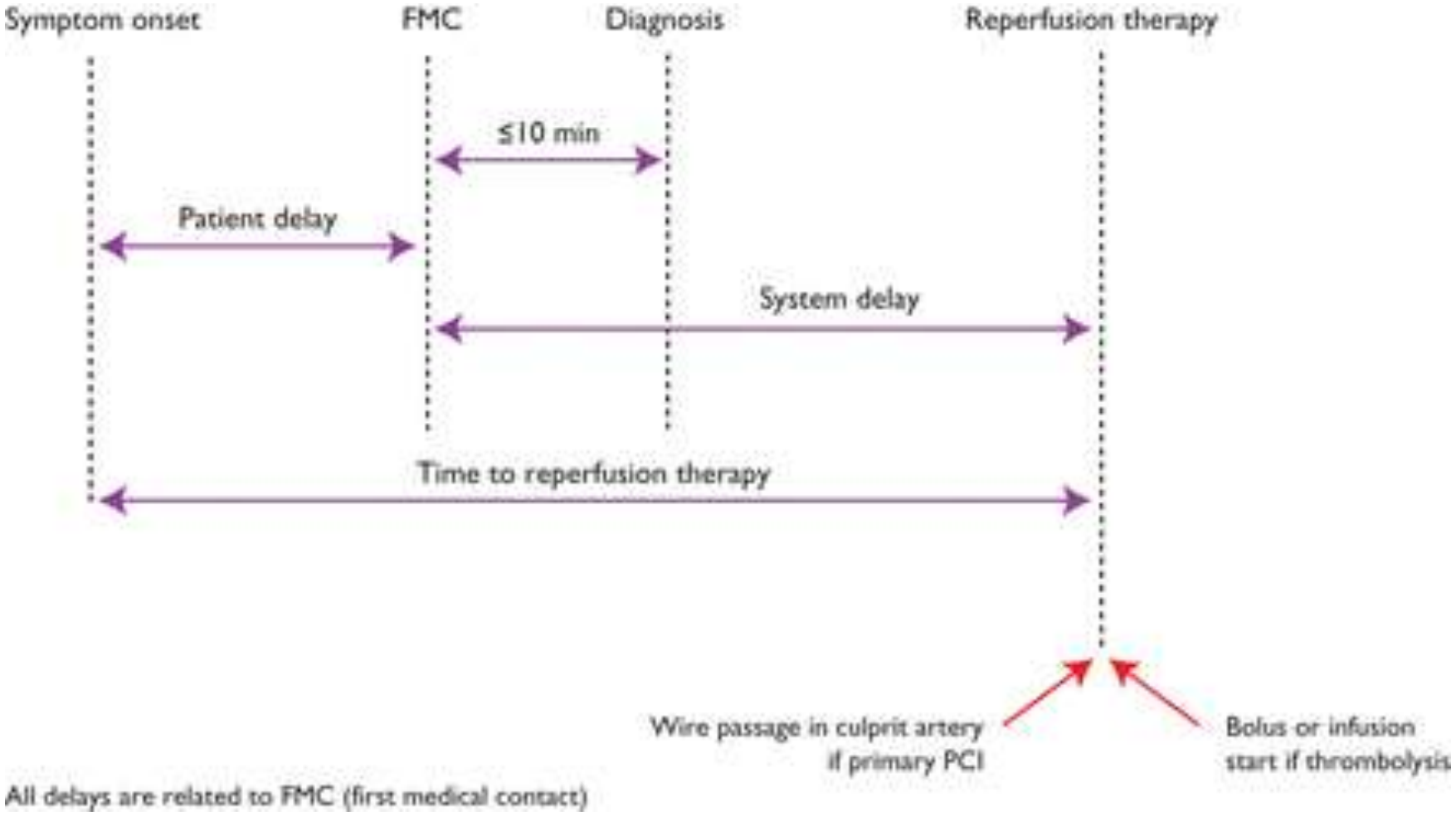
Troponin in MI reperfusion



dpmls89.wordpress.com/2012/09/09/a-quick-review-of-troponins/

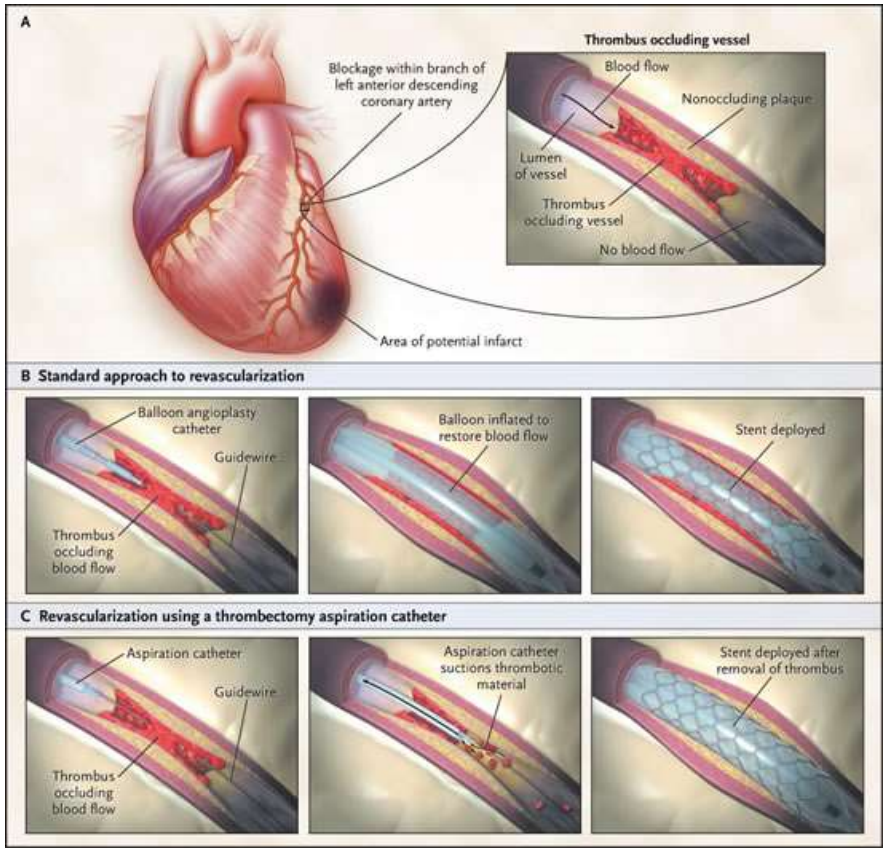
Treatment

Components of Delay in ACS and Ideal Time Intervals for Intervention



eurheartj.oxfordjournals.org/content/33/20/2569#T3

Treatment
Revascularization



- Percutaneous coronary intervention
- Coronary artery bypass surgery
- Revascularization vs. medical therapy

hemodinamiadelsur.com.ar/journals/journal_130.asp

Treatment

Stress' Optimization 1

- Upon hyperreactive distress and accelerated necrotic processes, our aim is to decrease the former and to retard the latter, using beta blockaders and anti-inflammatory drugs administered enterally and/or parenterally
- Hyporeactive distress and retarded necrotic processes require the prescription of alpha -and beta- adrenostimulators, bacterial and leukocytic pyrogens, leukopoiesis stimulants, using several ways of administration.

meteopathy.com/zdorovye-i-bolezn/my-myocardial-infarction-phylosophy/3/

Treatment
Stress' Optimization 2

- Upon intermittent distress, the attitude should be even more careful to prevent wave recurrence of necrotic processes.

meteopathy.com/zdorovye-i-bolezn/my-myocardial-infarction-phylosophy/3/

Lifestyle Modifications and Cardiac Rehabilitation 1

- Extensive patient education that includes providing easily understood and culturally sensitive written and verbal instructions about symptoms of ACS, as well as how and when to seek emergency care, in addition to providing instructions about medication types, purposes, doses, frequency, and side effects.

emedicine.medscape.com/article/155919-treatment#d7

Lifestyle Modifications and Cardiac Rehabilitation 2

- Dietary changes that adopt a low-fat and low-salt diet with dietary counseling, smoking cessation, up-to-date vaccination, and an increase in physical activity and exercise
- Aerobic exercise training within a cardiac rehabilitation programs, with the need for an evaluation of both exercise capacity and exercise-associated risk.

emedicine.medscape.com/article/155919-treatment#d7

Prognosis

- Six-month mortality rates in the Global Registry of Acute Coronary Events (GRACE) were 13% for patients with NSTEMI ACS and 8% for those with unstable angina
- The prognosis varies greatly depending on a person's health, the extent of the heart damage, and the treatment given.

emedicine.medscape.com/article/1910735-overview#a6

Prophylaxis 1

- Lifestyle recommendations include the adoption of a Mediterranean-type diet, maintaining alcohol intake within recommended limits, exercising to the point of mild breathlessness for 20–30 minutes every day, stopping smoking, and trying to achieve a healthy weight.
- Exercise is both safe and effective even if people have had stents or heart failure.

en.wikipedia.org/wiki/Myocardial_infarction

Prophylaxis 2

- People are usually started on several long-term medications: aspirin, warfarin, beta blockers, ACE inhibitors, angiotensin II receptor antagonist, statins, aldosterone antagonists.
- Previous studies suggested a benefit from omega-3 fatty acid supplementation but this has not been confirmed.

en.wikipedia.org/wiki/Myocardial_infarction

Abbreviations

- ACE - angiotensin converting enzyme
- ACS - acute coronary syndrome
- ALT - alanine aminotransferase
- ECG – electrocardiography
- ICD - International Classification of Diseases
- LDH - lactate dehydrogenase
- NQMI non-Q-wave myocardial infarction
- NSTEMI - non–ST-segment elevation myocardial infarction
- MI – acute myocardial infarction
- MVO_2 - myocardial oxygen consumption
- STEMI - ST-segment elevation myocardial infarction
- QMI Q-wave myocardial infarction

Diagnostic and treatment guidelines

- 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation
- 2014 AHA/ACC Guideline for the Management of Patients With Non–ST-Elevation Acute Coronary Syndromes
- 2014 ESC/EACTS Guidelines on myocardial revascularization
- ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation
- Recommendations for best-practice STEMI management in Ontario