Chapter 3
Acute Coronary Syndrome: ST-Segment Elevation Myocardial Infarction

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Name and Synonyms

ST-Segment Elevation Myocardial Infarction

- STEMI, ST-elevation MI, acute MI, transmural MI, Q-wave MI

Incidence/Epidemiology

- At the highest-acuity end of the continuum of diagnoses collectively called acute coronary syndrome (ACS)
- Highest pre- and in-hospital 30-day mortality of ACS spectrum
- Incidence varies widely by demographics and risk profiles
- More than 1.2 million MIs in US each year; about 20 % are STEMI

Differential Diagnosis

- Includes all other causes of chest pain

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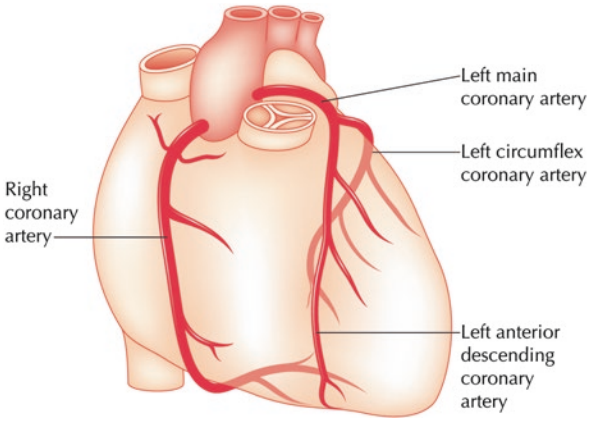
- Includes other causes of "anginal equivalents" in susceptible populations, e.g.:
- Dyspnea
- Back pain
- Jaw pain
- Shoulder pain
- Epigastric pain
- Palpitations
- Dizziness
- Weakness
- Nausea
- Syncope

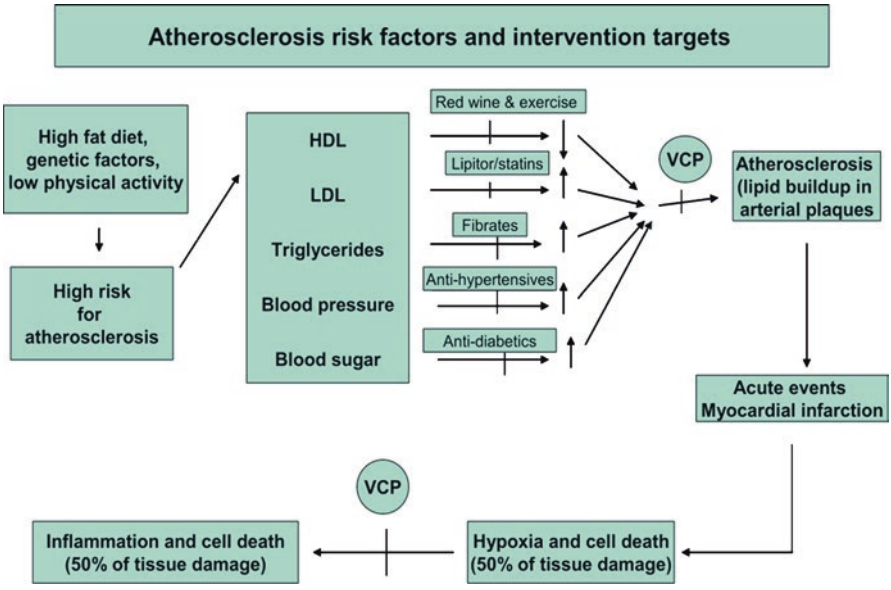
Pathophysiology and Etiology

- ACS is the result of ischemia (diminished blood flow / oxygen delivery) to myocardium
- If ischemia persists, frank infarction (muscle death) may occur
- Infarction results in breakdown of cell membranes, allowing release of intracellular proteins (such as myoglobin, troponins, and CPK-MB) into the circulation, where they may be detected in peripheral blood draw
- STEMI is most extreme and acute form of ACS, with "transmural" ischemia that results in characteristic acute findings on ECG (see below)
- Most STEMI occurs as a result of fracture or frank rupture of atherosclerotic plaque in an epicardial artery



Normal coronary anatomy. [Achenbach S. Normal coronary anatomy. In: Budoff MJ, Achenbach S, Narula J, editors. Atlas of cardiovascular computed tomography. Philadelphia: Current Medicine; 2007 (Braunwald E, editor. Atlas of heart diseases; vol. 1)] *Caption from original*

- Atherosclerosis is a product of diverse inherited and acquired conditions, including:
  - Family history
  - Hyperlipidemia
  - Hypertension
  - Diabetes mellitus
  - Tobacco abuse



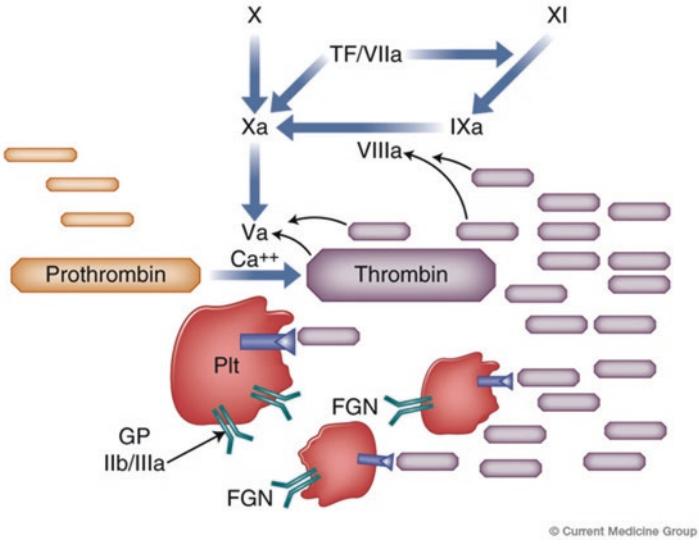
Atherosclerosis risk factors and intervention targets. [Thorbjornsdottir P, Thorgeirsson G, Kotwal GJ, Arason GJ. Control of inflammation with complement control agents to prevent atherosclerosis. In: Suri JS, Kathuria C, Molinari F, editors: Atherosclerosis disease management. New York: Springer. p. 633-75. Book https://doi.org/10.1007/978-1-4419-7222-4; Chapter: 20; Chapter https://doi.org/10.1007/978-1-4419-7222-4_20, 2011-01-01] *Caption adapted from original*

Risk factors	No. of patients
Individual	
Smoking	6
Hypertension	11
Hypercholesterolemia	9
Diabetes mellitus	4
Clusters	
Smoking and hypercholesterolemia	1
Hypertension and hypercholesterolemia[a]	4
Hypertension and diabetes	2
Smoking, hypertension and hypercholesterolemia	1
Smoking, hypertension, hypercholesterolemia and diabetes	2

[a] Hypertension was defined as a diastolic arterial pressure >95 mm Hg and hypercholesterolemia >6 mmol/l

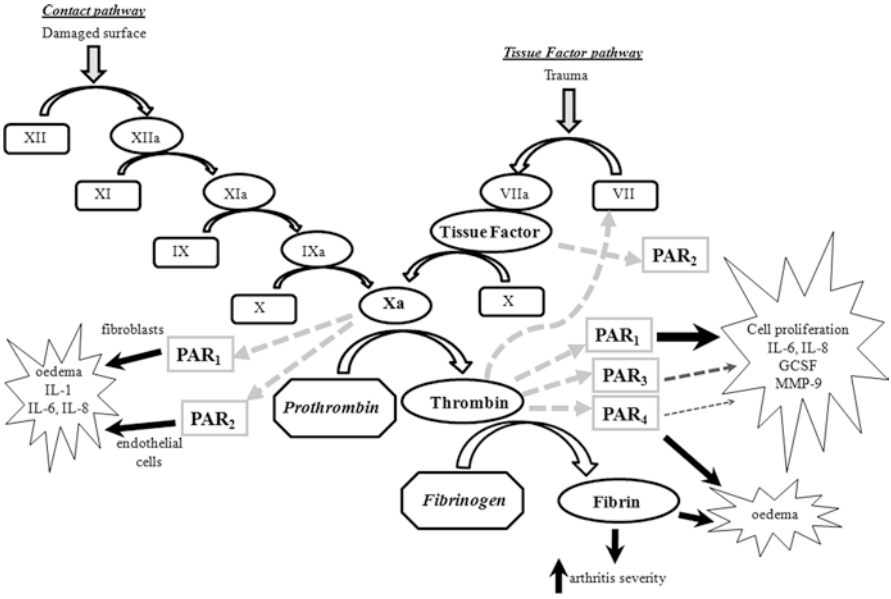
Main known risk factors for atherosclerosis in 16 patients studied. [Watt S, Aesch B, Lanotte P, Tranquart F, Quentin R. Viral and bacterial DNA in carotid atherosclerotic lesions. Eur J Clin Microbiol Infect Dis. 2003 Feb;22(2):99-105. https://doi.org/10.1007/s10096-002-0867-1, 2003-02-01] *Caption adapted from original*

- Plaque rupture initiates a complex thrombo-inflammatory response locally, including activation of:
  - Coagulation



Networking of coagulation cascade and aggregation of platelets (Plt). The extrinsic limb triggers activation of the coagulation cascade when tissue factor (TF) is exposed in a disrupted plaque. Coagulation factor VII is activated (VIIa) and can

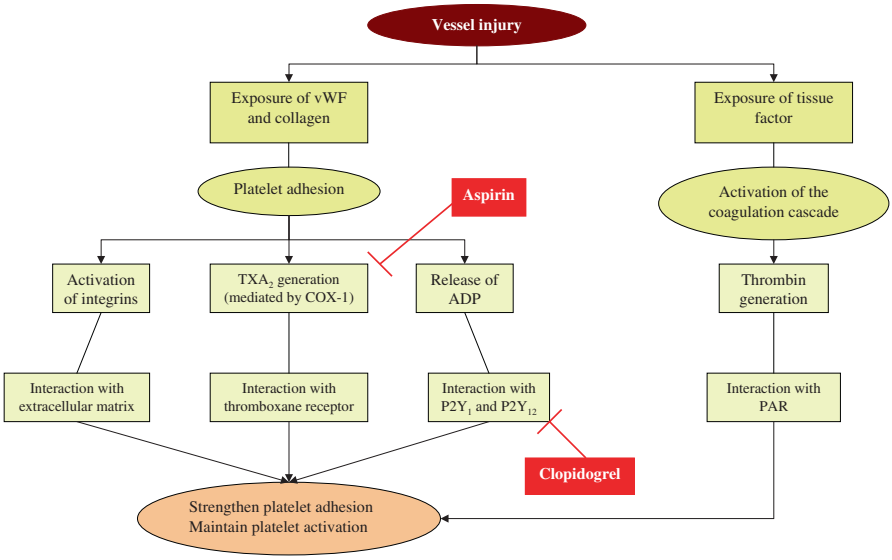
activate factor X to Xa and promote perpetuation of the coagulation process via the intrinsic limb that results in formation of IXa and VIIIa. The prothrombinase complex of Xa, Va, Ca^{++} forms on a phospholipid surface (*eg*, membrane of a platelet) and converts prothrombin to thrombin. The thrombin that is formed binds to the thrombin receptor on platelets promoting activation and aggregation of platelets, as well as amplifying the coagulation cascade by promoting formation of VIIIa and Va. This diagram depicts the amplification nature of the coagulation process because one molecule of Xa leads to the downstream production of a large number of thrombin molecules (stoichiometric relationship not completely depicted to prevent obscuring the diagram with thrombin molecules). Activated platelets express numerous copies of the active form of the fibrinogen receptor GP IIb/IIIa on their surface. GP IIb/IIIa recognizes specific amino acid sequences on circulating ligands. One such ligand is fibrinogen (FGN), which has multiple copies of the RGD amino acid sequence and serves to bridge platelets together, promoting formation of aggregates. The more aggregates formed, the greater the surface area for the prothrombinase complex and amplification of the reactions of the coagulation cascade. [Antman E. Acute coronary syndromes. In: Libby P, editor. Essential atlas of cardiovascular disease. Philadelphia: Current Medicine; 2009. ISBN: 978-1-57340-309-2, 2009-05-21; Antman, Elliott] *Caption from original*



A simplified diagram of the coagulation cascade and its links with PARs and inflammation. Two pathways are involved in coagulation, with the major one being the tissue factor pathway. Most of the coagulation factors are serine proteinases that are present as inactive zymogens (rectangles) that when activated (ovals; lowercase 'a' indicates an active form) can catalyze the next reaction in the cascade. The two

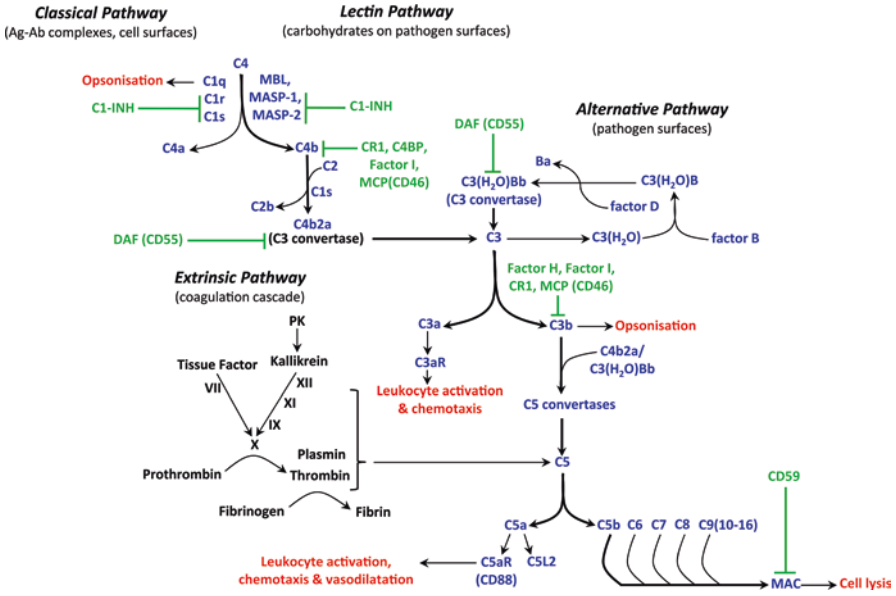
pathways converge to activate factor X, thrombin and fibrin. [Russell FA, McDougall JJ. Proteinase-activated receptors and arthritis. In: Vergnolle N, Chignard M, editors. Proteases and their receptors in inflammation. Basel: Springer; 2011. p. 217-42. Book https://doi.org/10.1007/978-3-0348-0157-7; Chapter: 9; Chapter https://doi.org/10.1007/978-3-0348-0157-7_9, 2011-01-01] *Caption adapted from original*

- Platelets



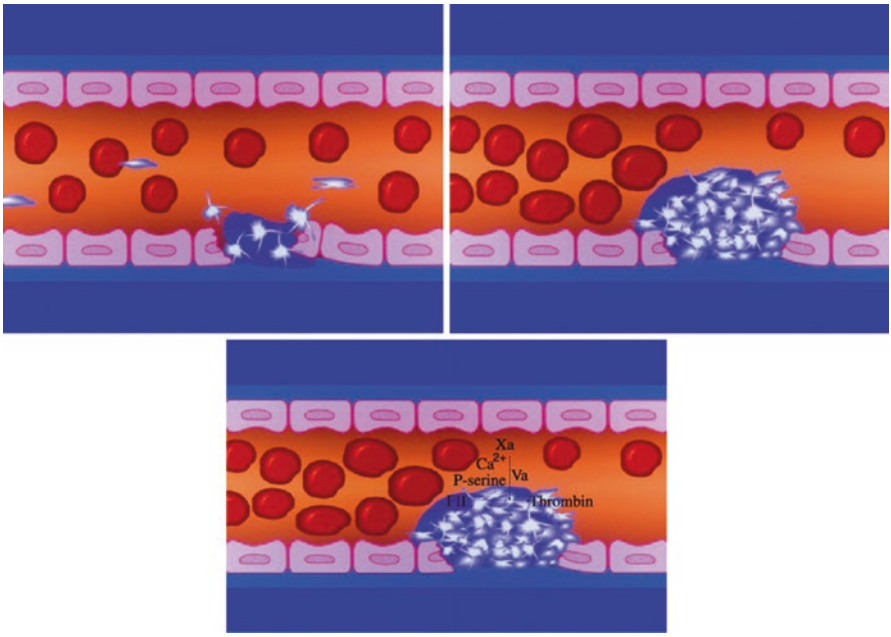
Mechanisms of platelet activation. [Ajjan R, Storey RF, Grant PJ. Aspirin resistance and diabetes mellitus. Diabetologia. 2008 Mar;51(3):385-90. https://doi.org/10.1007/s00125-007-0898-3, 2008-02-01] *Caption adapted from original*

- Complement



Common pathways for complement activation. [From article: Complement activation in the injured central nervous system: another dual-edged sword? J Neuroinflammation. 2012 Jun 21;9:137. https://doi.org/10.1186/1742-2094-9-137, at http://link.springer.com/article/10.1186%2F1742-2094-9-137; by Faith H Brennan, Aileen J Anderson, Stephen M Taylor, Trent M Woodruff, Marc J Ruitenberg, © Brennan et al.; licensee BioMed Central Ltd. 2012; licensed under Creative Commons Attribution License http://creativecommons.org/licenses/by/2.0] *Caption adapted from original*

- This response results in local aggregation of activated platelets that decreases and ultimately occludes downstream blood flow, leaving muscle distal to lesion ischemic and subject to infarction unless there is sufficient downstream collateral perfusion.

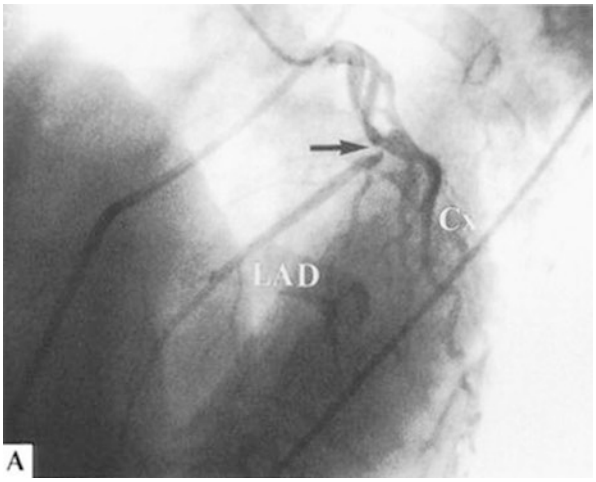


Platelet adhesion at the site of injury and aggregation with one another (a). Platelet plug consolidation (b) and platelets expressing procoagulant activity on their surface with subsequent thrombin generation (c). Procoagulant factors are represented by roman numbers. P-serine, phosphatidylserine. [Tripodi A. Haemostasis abnormalities in chronic liver failure. In: Ginès P, Kamath PS, Arroyo V, editors. Chronic liver failure. New York: Springer; 2011. p. 289-303. Book https://doi.org/10.1007/978-1-60761-866-9; Chapter: 14; Chapter https://doi.org/10.1007/978-1-60761-866-9_14, 2011-01-01] *Caption from original*

- Sudden acute ischemia from complete upstream artery occlusion results in characteristic ECG findings (see below).
- Alternative and much rarer etiologic considerations

- Pure arterial spasm without plaque rupture
- Arteritis

  - Lupus



Anteroapical myocardial infarction in a patient with SLE. A, Cranial left anterior oblique view on arteriography showing severe proximal stenosis (arrow) of the left anterior descending (LAD) coronary artery that led into an anteroapical infarction in a 41-year-old woman with flaring SLE. [Roldan CA. Rheumatic and connective tissue diseases and the heart. In: Crawford MH, editor. Heart disease in the presence of disorders of other organ systems. Philadelphia: Current Medicine; 1996 (Braunwald E, editor. Atlas of heart diseases; vol. 6). ISBN: 1-878132-28-8, 2002-01-23] *Caption adapted from original*

- Takayasu's disease
- Kawasaki's disease

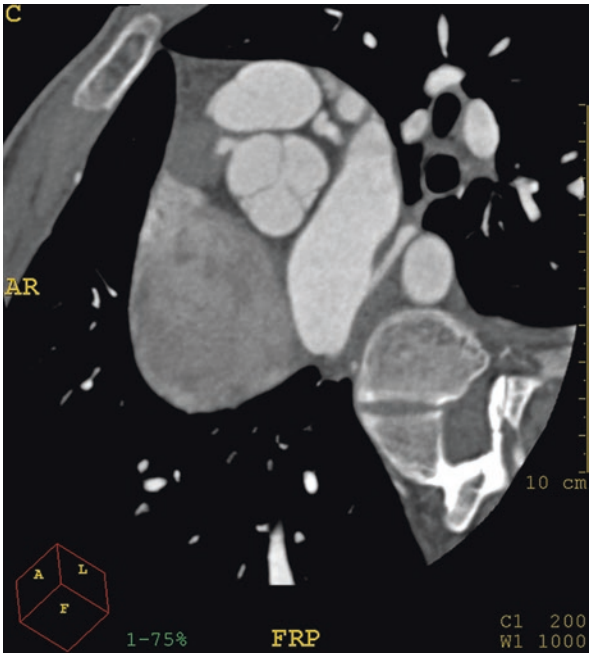


Image obtained from a patient with an acute myocardial infarction caused by thrombosis of a right coronary artery Kawasaki aneurysm (*arrow*). Kawasaki disease results from a mucocutaneous viral infection acquired during childhood. Typically, multiple aneurysms may develop in the coronaries and in other systemic vessels. Thrombosis of these aneurysms may occur later in life. [Garcia MJ. Intracardiac, myocardial and extracardiac abnormalities. In: Budoff MJ, Achenbach S, Narula J, editors. Atlas of cardiovascular computed tomography. Philadelphia: Current Medicine; 2007] *Caption from original*

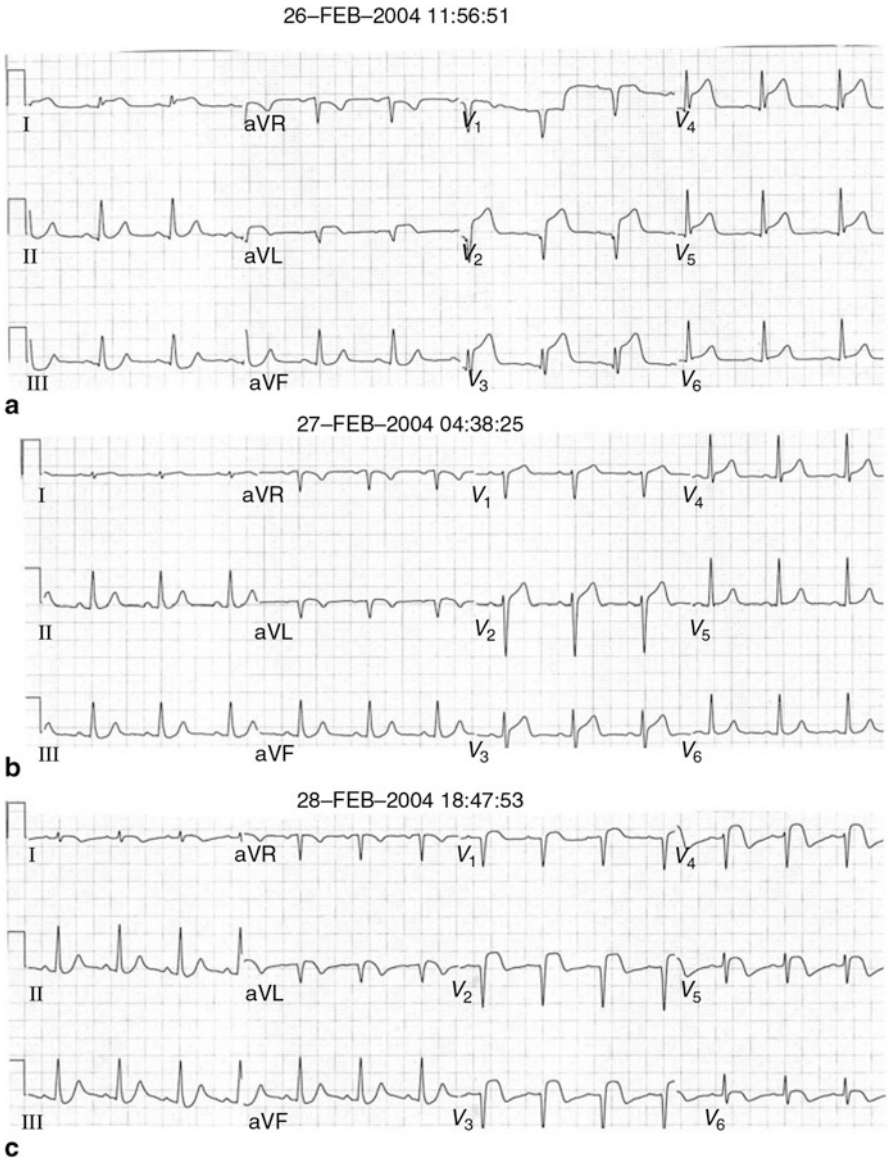
- Cocaine- or methamphetamine-induced vasospasm

Presentation

Typical/"Classic"

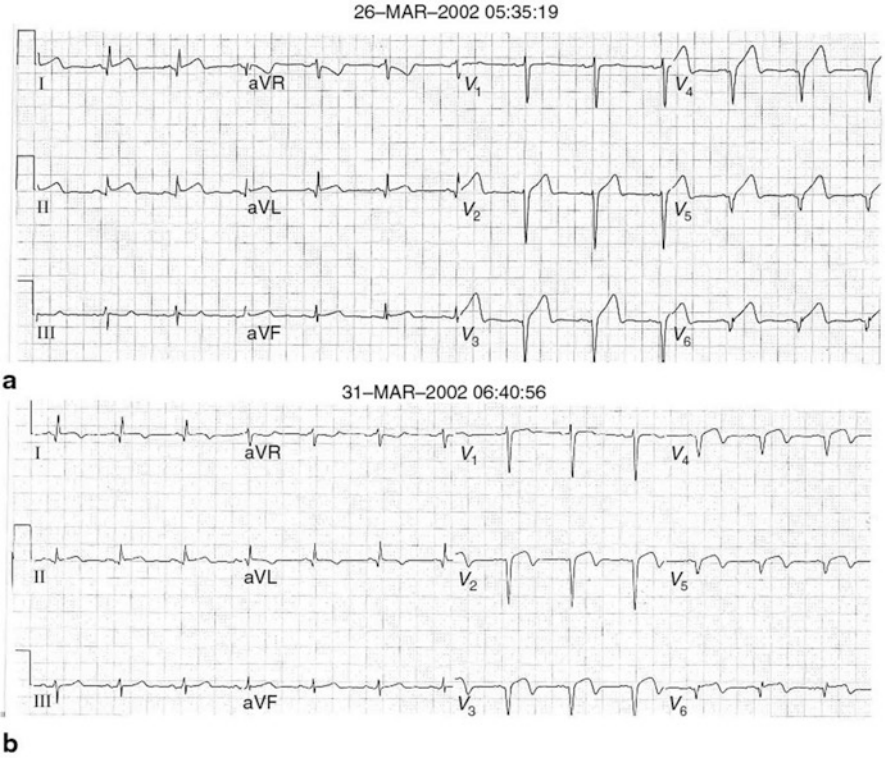
- Pressure-like chest pain starting substernally and radiating toward left shoulder or left jaw
- Pain is classically associated with diaphoresis, dyspnea, and nausea
- Pain often accompanied by tachycardia; blood pressure is variable with very high and very low presenting blood pressures associated with poorer prognosis
- Pain often starts with exertion; may be improved with rest or with use of nitroglycerin. May also start at rest and often persists >20 minutes before presentation
- Pain unchanged with movement, positioning, or deep breathing

- In describing pain, patient may hold clenched fist over chest ("Levine sign")
- Defined (as name implies) by specific ECG findings:
  - ST-segment elevation in two or more contiguous leads OR



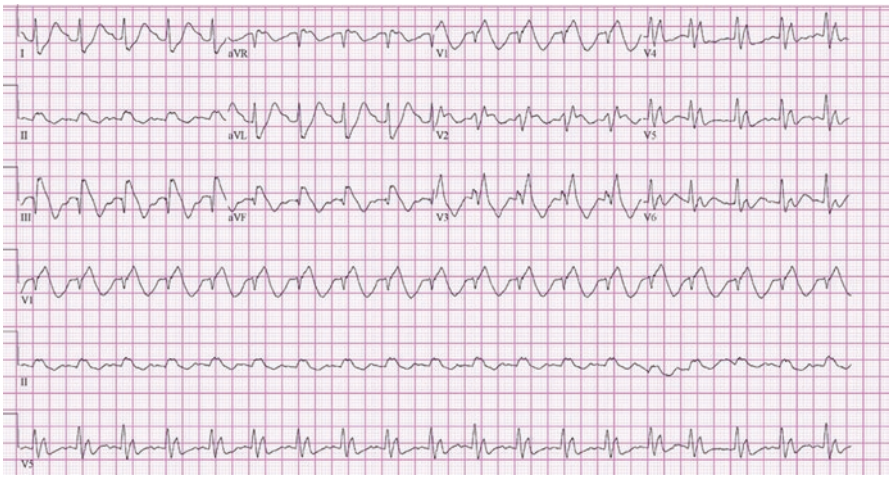
Electrocardiogram of a 51-year-old man with acute anterior-superior myocardial infarction caused by total occlusion of left anterior descending coronary artery proximal to the first septal perforator and the first diagonal branch; there was also

40% occlusion of the first obtuse marginal branch of the left circumflex artery and 70% occlusion of the right coronary artery. Kinesis involved large portion of the anterior wall and the apex. with an estimated left ventricular ejection fraction of 15%. In (a): ST elevation in leads aVL and V1–6, with reciprocal ST depression in leads III and aVR. In (b): one day later "pseudonormalization" with slight ST elevation in the leads V 2–3 and T wave inversion in lead aVL as the only abnormalities. In (c): on the following day when chest pain resolved. ST elevation and T wave inversion in leads I, aVL and V 1–6 with reciprocal ST depression in the leads III, aVF compatible with the evolution of the infarction pattern. Incipient T wave inversion in leads a VL, V 2–6. [Surawicz B. Ventricular repolarization in myocardial ischemia and myocardial infarction: theory and practice. In: Macfarlane PW, van Oosterom A, Pahlm O, Kligfield P, Janse M, Camm J, editors. Comprehensive electrocardiology. London: Springer; 2011. p. 803-31. Book https://doi.org/10.1007/978-1-84882-046-3; Chapter: 18; Chapter https://doi.org/10.1007/978-1-84882-046-3_18, 2010-01-01] *Caption from original*



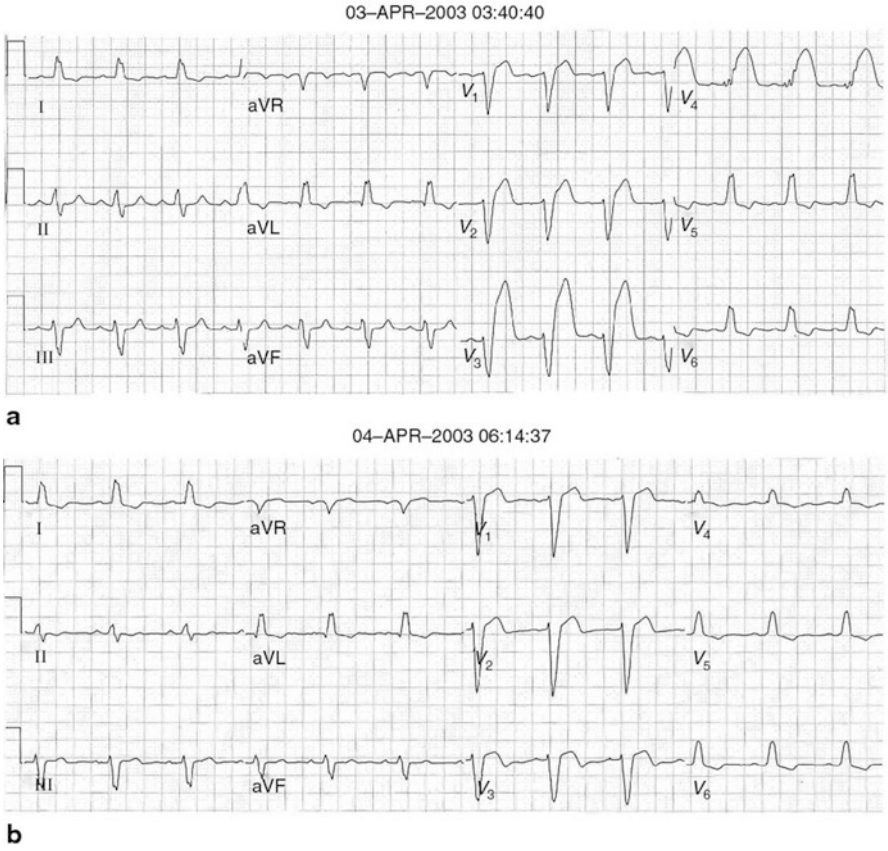
Electrocardiogram of a 47-year-old woman with anterolateral myocardial infarction (MI) caused by total occlusion of left anterior descending coronary artery in midportion associated with 50–70% occlusion of the co-dominant left anterior circumflex artery. Anterior wall and apex were akinetic with an estimated left ventricular

ejection fraction of 30%. In (a): Q waves with ST elevation in leads I, aVL, V 3–6 and reciprocal ST depression in aVR. In (b) 5 days later after an emergent percutaneous intervention evolution of ECG pattern of anterior MI with T wave inversion in leads I, aVL and V 2–6. Residual ST elevation is present in the above leads. [Surawicz B. Ventricular repolarization in myocardial ischemia and myocardial infarction: theory and practice. In: Macfarlane PW, van Oosterom A, Pahlm O, Kligfield P, Janse M, Camm J, editors. Comprehensive electrocardiology. London: Springer; 2011. p. 803-31. Book https://doi.org/10.1007/978-1-84882-046-3; Chapter: 18; Chapter https://doi.org/10.1007/978-1-84882-046-3_18, 2010-01-01] *Caption from original*



Electrocardiogram on presentation showing coved ST-segment elevation in precordial leads with simultaneous ST-segment elevation in leads II, III, and aVF. [Sheikh M, Kanjwal K, Kasmani R, Chutani S, Maloney JD. Simultaneous ST-segment elevation in inferior and precordial leads following ingestion of a lethal dose of desipramine: a novel Brugada-like EKG pattern. J Interv Card Electrophysiol. 2010 Jun;28(1):35-8. https://doi.org/10.1007/s10840-009-9412-9, 2010-04-13] *Caption from original*

- Known-to-be-new left bundle branch block in presence of anginal symptoms



Electrocardiogram of a 58-year-old man with left bundle branch block (LBBB) and acute anterior myocardial infarction (MI). In (a), in addition to secondary ST and T changes of LBBB, there is primary ST elevation in leads $V_{2–4}$-indicative of acute injury pattern. No reciprocal ST depression is discernible. In (b), after percutaneous intervention, primary ST elevation subsided, but primary T-wave inversion in leads $V_{2–3}$ is compatible with evolution of anterior MI pattern. [Surawicz B. Ventricular repolarization in myocardial ischemia and myocardial infarction: theory and practice. In: Macfarlane PW, van Oosterom A, Pahlm O, Kligfield P, Janse M, Camm J, editors. Comprehensive electrocardiology. London: Springer; 2011. p. 803-31. Book https://doi.org/10.1007/978-1-84882-046-3; Chapter: 18; Chapter https://doi.org/10.1007/978-1-84882-046-3_18, 2010-01-01] *Caption from original*

Atypical

- STEMI has been discovered in patients with virtually any complaint localized above the umbilicus.
- "Anginal equivalents" listed above include different distributions of pain (e.g., epigastric, jaw, neck, back) and some presentations that are not painful at all (e.g., palpitations, nausea, syncope).
  - Patients with STEMI who present without chest pain frequently experience delays in diagnosis and therefore delays in treatment.
  - Atypical presentations more common in women, in elderly, and in diabetics.
- Diagnostic ECG findings are the defining and unifying feature of all STEMI presentations.

Primary Differential Considerations

- Primary differential considerations include pain mimics to STEMI and are diverse, including life-threatening and more benign causes:
  - Aortic dissection
  - Pulmonary embolism
  - Peptic and esophageal disease, hiatal hernia
  - Costochondritis
  - Pneumonia
  - Pneumothorax
  - Pleurisy
  - Anxiety and panic disorders
  - Biliary colic
  - Herpes zoster

	Helpful clinical features
Cardiovascular	
Angina	Retrosternal chest pressure, squeezing, heaviness. Associated with exertion or emotional stress, relieved by rest or nitroglycerin. Usually between 2 and 20 min in duration.
Aortic stenosis	Similar features as for angina, but with late-peaking systolic murmur radiating to carotids. May be associated with syncope or signs of left heart failure.
Pericarditis	Sharp, retrosternal, pleuritic chest pain lasting hours to days. May be associated with friction rub and may be alleviated by leaning forward.
Aortic dissection	Sudden onset of tearing, ripping chest pain radiating to back. Associated with underlying hypertension.
Pulmonary embolism	Ipsilateral pleuritic pain associated with dyspnea, tachycardia, possible cor pulmonale. May have irritative cough or hemoptysis or present with syncope. Usually sudden onset.
Pulmonary	
Pneumonia/ pleuritis/pleural effusion	Pleuritic pain, lateralizing to side of infection/ inflammation. May be associated with fevers, dyspnea, cough. Exam with pleural rub, consolidation, or dullness to percussion.
Asthma/COPD exacerbation	Chest "tightness" associated with more prominent findings of dyspnea, tachypnea and diffuse wheezing.
Spontaneous pneumothorax	Sudden onset of pleuritic pain. Unilateral and associated with dyspnea. More common in thin, young males or patients with emphysematous disease. Decreased breath sounds and hyperresonance on side of pneumothorax.
Chest wall	
Muscle spasm/ strain	Associated with prior increased physical activity/ weight lifting. Pain variable in character but usually reproducible with palpation.

	Helpful clinical features
Costochondritis	Sharp, sudden onset pain that is short in duration. May be reproducible with palpation.
Herpes zoster	Sharp, burning, superficial neuropathic pain. May have allodynia, vesicular rash on exam. Unilateral dermatomal distribution.
Rib fracture	Prior trauma or known metastatic disease of bone. Point tenderness over affected rib(s). Pain is usually pleuritic.
Cervical/thoracic nerve root compression	Intermittent neuropathic pain often associated with neck movement or position. Usually unilateral.
Gastrointestinal	
Mediastinitis/ esophageal rupture	Often preceded by esophageal procedure or forceful vomiting. Pt. may have fever, associated septic shock. Symptoms vary from burning chest discomfort to severe dyspnea.
Esophageal reflux	Burning pain, often associated with nausea, belching. Usually worse at night and after large meals. Alleviated by antacids.
Esophageal spasm	Sudden onset, sharp, retrosternal pain. May be relieved by nitroglycerine and exacerbated by cold liquids. Sometimes associated with dysphagia.
Pancreatitis	Sharp epigastric pain, usually constant and prolonged. Exacerbated by food and often associated with nausea/vomiting. Alcohol and gallstones are risk factors.
Peptic ulcer	Sharp or burning epigastric pain. Often relieved by food or antacids. May be associated with occult GI bleeding or massive acute blood loss.
Psychogenic	
Anxiety/panic disorder	May be unable to distinguish from anginal pain, but usually has atypical features such as prolonged duration and no exertional component. Should be a diagnosis of exclusion at initial workup.

Differential diagnosis of acute chest pain. [McClintic BR, Rosenblatt RL. Approach to the patient with chest pain. In: Bisognano JD, Beck R, Connell R, editors. Manual

of outpatient cardiology. London: Springer. p. 349-71. Book https://doi.org/10.1007/978-0-85729-944-4; Chapter: 13; Chapter https://doi.org/10.1007/978-0-85729-944-4_13, 2012-01-01] *Caption from original*

History and Physical Exam

Findings That Confirm Diagnosis

- None

Factors That Suggest Diagnosis

- Nature and history of pain with presence of risk factors.

Factors That Exclude Diagnosis

- None

Ancillary Studies

Laboratory

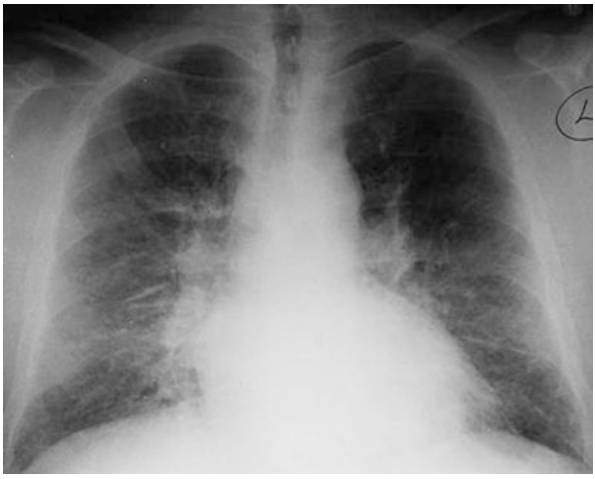
- CBC: no diagnostic findings
- Electrolytes: no diagnostic findings
- Renal function: no diagnostic findings, but renal insufficiency/failure is a risk factor for ACS and may complicate treatment
- Coagulation studies: no diagnostic findings but should be performed before initiation of anticoagulation therapy
- Cardiac enzymes: will be elevated in STEMI, but presentation often precedes abnormal test values. Elevation of enzymes is NOT required for diagnosis of STEMI.
    - Elevation in troponin (I or T) levels is part of universal diagnosis of myocardial infarction

Electrocardiography

- As above, STEMI is defined by the ECG
- Per national quality standards in the United States, ECG should be obtained within 10 minutes of arrival in an emergency department

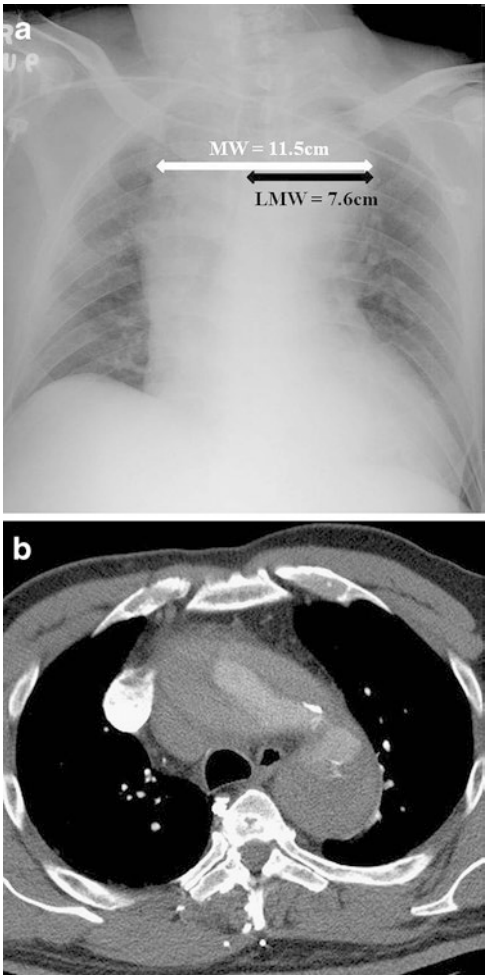
Imaging

- No diagnostic findings for STEMI on chest x-ray (CXR)
- Findings of heart failure (cardiomegaly, pulmonary congestion) on CXR portend a poorer prognosis on STEMI



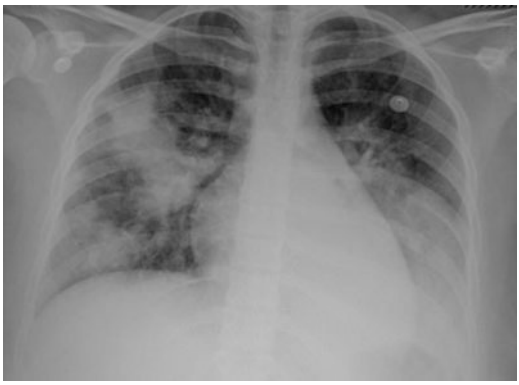
Frontal chest radiograph reveals typical features of pulmonary interstitial edema in a patient with congestive heart failure, manifested by peribronchial cuffing, indistinctness of the pulmonary vessels, and Kerley B lines. Note the cephalization or redistribution of the pulmonary vasculature and mild cardiomegaly. [Boiselle PM, Dass C, Steiner RM. Radiologic imaging in the critically ill patient. In: Criner GJ, Barnette RE, D'Alonzo GE, editors. Critical care study guide. New York: Springer; 2010. p. 181-207. Book https://doi.org/10.1007/978-0-387-77452-7; Chapter: 11; Chapter https://doi.org/10.1007/978-0-387-77452-7_11, 2010-01-01] *Caption from original*

- Findings of a widened mediastinum suggest consideration of aortic dissection but do not exclude STEMI. Check differential blood pressures in the upper extremities.



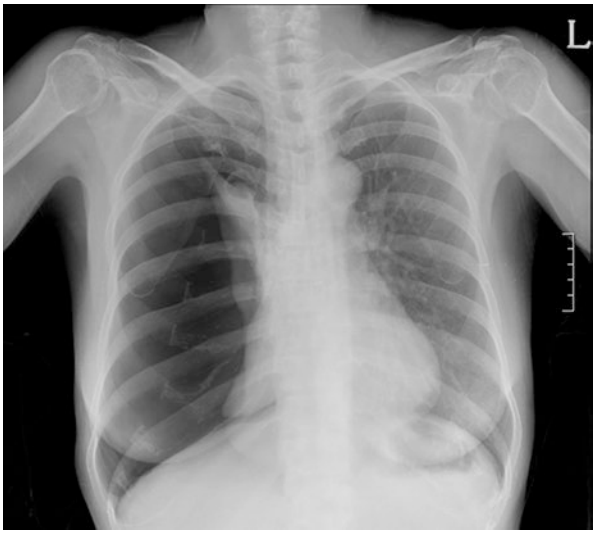
Acute type A aortic dissection in a 46-year-old man. A) AP chest radiograph showing marked widening of the mediastinum with MW and LMW measuring 11.5 and 7.6 cm, respectively. B) Corresponding selected image of CT aortogram confirms type A aortic dissection. [From article: Diagnostic accuracy of mediastinal width measurement on posteroanterior and anteroposterior chest radiographs in the depiction of acute nontraumatic thoracic aortic dissection. Emerg Radiol. 2012 Aug;19(4):309-15. https://doi.org/10.1007/s10140-012-1034-3, at http://link.springer.com/article/10.1007%2Fs10140-012-1034-3; by Vincent Lai, Wai Kan Tsang, Wan Chi Chan, Tsz Wai Yeung, © The Author(s) 2012; licensed under Creative Commons Attribution License https://creativecommons.org/licenses/by/2.0/] *Caption from original*

- Findings of pneumonia suggest consideration of that differential but do not exclude STEMI. Check for signs of infection.



Chest radiograph showing right upper/lower lung field opacities and left lower lung field opacity consistent with pneumonia. [Tsigrelis C, Mohammad M, Fraimow HS, Dellinger RP, Marchesani D, Reboli AC. Secondary bacterial pneumonia due to Staphylococcus aureus complicating 2009 influenza A (H1N1) viral infection. Infection. 2010 Jun;38(3):237-9. https://doi.org/10.1007/s15010-010-0009-0, 2010-06-01] *Caption adapted from original*

- Findings of pneumothorax suggest consideration of that differential but do not exclude STEMI. Evaluate oxygenation status.



Posteroanterior upright chest X-ray shows large pneumothorax of the right lung. [Kim SH, Yoo WH. Recurrent pneumothorax associated with pulmonary nodules after leflunomide therapy in rheumatoid arthritis: a case report and review of the

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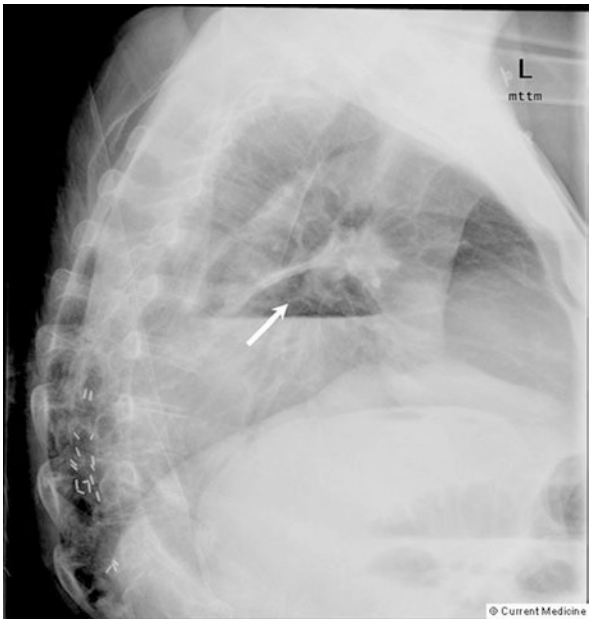
- Findings of hiatal hernia suggest consideration of that differential but do not exclude STEMI



Radiograph of large hiatal hernia. [Aurigemma G, Tighe D, Oh J, Espinoza R. Pericardial disease and cardiac masses. In: Solomon S, editor. Atlas of echocardiography. Philadelphia: Current Medicine; 2008.] *Caption from original*

Special Populations

Age

- Risk for STEMI increases with age, starting at age 40 in men and at menopause in women.
  - Age is a risk factor for mortality from STEMI.
  - Likelihood of atypical presentation with STEMI increases with age.

Pediatric Considerations

- A congenital abnormality of the coronary vasculature, in which there is an anomalous origin of the left coronary artery arising from the pulmonary artery (ALCAPA), may occur in infancy. It presents with various symptoms of cardiac ischemia (diaphoresis when feeding) and if not recognized and corrected surgically, may cause irreversible myocardial damage.

Co-morbidities

- Hypertension, diabetes, and renal failure are important comorbidities in STEMI risk and prognosis.
- Obesity is a weaker predictor of STEMI risk.

Pitfalls in Diagnosis

Critical Steps Not to Miss

- ECG must be performed as soon as the diagnosis of STEMI is even considered.

Mimics

- All differential considerations listed above.
  - Of these, only aortic dissection and pulmonary embolism are also life-threatening.

Time-Dependent Interventions

- Restoration of myocardial perfusion is time sensitive.
  - If possible, STEMI should be managed in the cardiac cath laboratory within 90 minutes of initial evaluation.
  - Outcomes worsen with each 30–60-minute delay in restoration of flow.
  - If interventional management is not possible, evaluation for fibrinolytic therapy should be performed immediately, with a goal of delivering lytic therapy to appropriate STEMI patients within 30 minutes of initial evaluation.

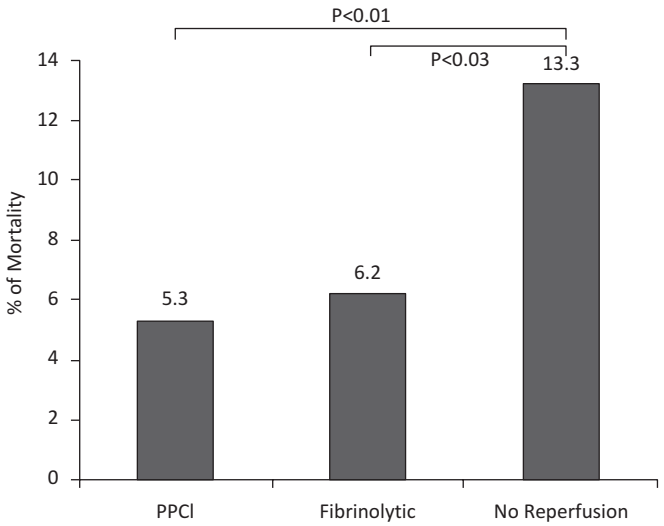
- Unless contraindicated (allergy, active bleeding), 324-325 mg aspirin should be administered immediately upon suspicion of an acute coronary syndrome.
- Extremes of blood pressure should be promptly treated to avert shock (hypotension) or undue myocardial oxygen demand (hypertension).
- Airway and oxygenation should be monitored and supported as necessary.
- Patients with STEMI should be on continuous cardiac monitoring to evaluate for dangerous arrhythmias.

Overall Principles of Treatment

- Immediate stabilization of the patient with control of blood pressure, pulse rate, and pain is critical. The patient may require resuscitation, intubation, and intensive support.
- Measures to reperfuse infarcting myocardium are essential and are time sensitive.

Disease Course

- STEMI mortality is highest in the prehospital setting.
- Once at the emergency department, in-hospital STEMI mortality among patients receiving reperfusion therapy is around 6 %.



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Related Evidence

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Use PubMed Clinical Queries to find the most recent evidence. Use this search strategy:

"ST segment elevation myocardial infarction" OR "STEMI"