NOTES
ACUTE CORONARY SYNDROMES

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Signs and symptoms caused by decreased blood flow in the coronary arteries to the extent that the muscle cannot function properly, or even dies
- Acute coronary syndromes are almost always due to atherosclerosis

SIGNS & SYMPTOMS

- See individual disorders

DIAGNOSIS

- See individual disorders

TREATMENT

MEDICATIONS

- Oxygen as needed
- Pain control with nitrates and/or morphine
- Antiplatelets
- Anticoagulants
- *Nitrates:* decreases preload by vasodilation
- *Beta blockers:* reduces cardiac demand by decreasing heart rate, BP and contractility (first-line choice: metoprolol)
- *Statins:* HMG-CoA reductase inhibitor that reduces production of cholesterol to improve lipid profile

OTHER INTERVENTIONS

- Hospital admission with continuous monitoring
- Reestablish blood flow via catheterization / revascularization
- *Lifestyle changes:* improve diet (reduce intake of saturated fat), smoking cessation, control blood pressure, strict management of diabetes mellitus, increase exercise, weight loss, improve lipid profile

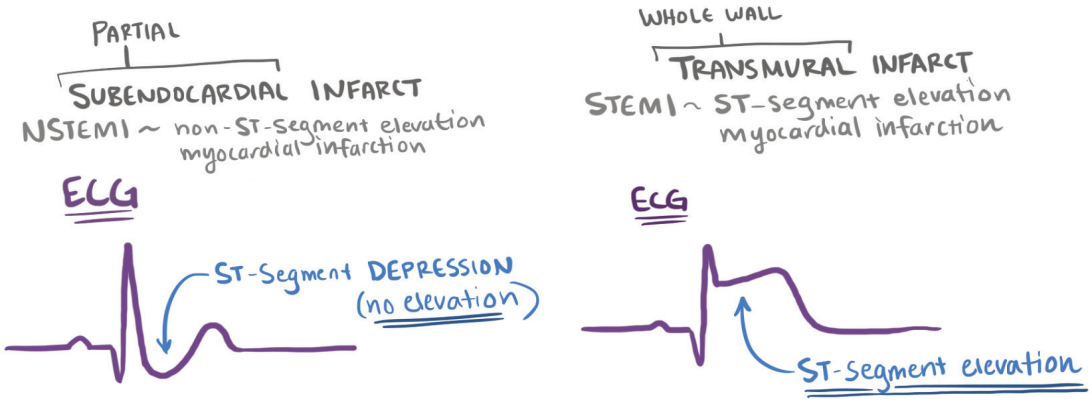

Figure 1.1 Illustration depicting ST depression and ST elevation seen in myocardial infarctions.

MYOCARDIAL INFARCTION (MI)

osms.it/myocardial-infarction

PATHOLOGY & CAUSES

- Death of heart muscle cells due to lack of oxygen-rich blood flow
- Plaque buildup (fat, cholesterol, proteins, calcium, white blood cells) takes years to form in lumen
- Blood platelets adhere to plaque and enhance clotting process, creating blockage
- Necrosis of myocardial cells follows series of events
  - *< 24 hrs:* early coagulative necrosis, cell debris in blood, edema, wavy fibers and hemorrhage
  - *1–3 days:* extensive necrosis, tissue has acute inflammation with neutrophils
  - *3–14 days:* macrophages and granulation tissue in margins
  - *> 14 days:* contracted scar forms

TYPES

ST segment elevation myocardial infarction (STEMI)

- Coronary artery completely blocked; full thickness of myocardial wall involved
- ECG shows ST elevation, possible Q waves

Non-ST segment elevation MI (NSTEMI)

- Coronary artery not completely blocked, subendocardium may be especially vulnerable to ischemia
- ECG shows ST depression

RISK FACTORS

- *Modifiable risk factors:* older age, smoking, high blood pressure, diabetes mellitus, high cholesterol, low levels of physical activity, obesity, excessive alcohol use, illegal drug use (e.g., cocaine, amphetamines), chronic stress
- *Non-modifiable risk factors:* family history, biological male

COMPLICATIONS

- *Heart failure:* heart muscle fails to compensate for damage; risk related to size/territory of infarct and individual's baseline cardiac function
- *Cardiac arrhythmia:* may be seen within minutes after MI or years later. If undiagnosed MI, may be cause of death
- *Left ventricular (LV) failure and pulmonary edema:* happens after left ventricular infarction, free wall rupture, ventricular septal defect, papillary muscle rupture with mitral regurgitation
- Postinfarction pericarditis, papillary muscle rupture (might lead to acute, severe mitral regurgitation), interventricular septal rupture, ventricular pseudoaneurysm formation, ventricular free wall rupture (might lead to ventricular free wall rupture leading to pericardial tamponade/ventricular pseudoaneurysm), true ventricular aneurysm, Dressler syndrome

MNEMONIC: DARTH VADER

Complications of MI

Death
Arrythmia
Rupture (free ventricular wall/ ventricular septum/papillary muscles)
Tamponade
Heart failure (acute or chronic)
Valve disease
Aneurysm of ventricles
Dressler's syndrome
thrombo**E**mbolism (mural thrombus)
Recurrence/ mitral **R**egurgitation

SIGNS & SYMPTOMS

- Acute chest pain lasting > 20 min, radiating to arm/jaw
- Uncomfortable chest/back/neck/jaw/stomach pain, dyspnea, fatigue, diaphoresis, and/or nausea
- Feeling of fullness/indigestion

DIAGNOSIS

LAB RESULTS

- Usually detected with diagnostic laboratory work for cardiac enzymes
  - *Troponin I, troponin T most specific, sensitive markers:* rise apparent within 2–4 hrs, peaking ~24 hrs
  - *CK-MB can detect reinfarction after initial MI:* levels increased 4 hrs after infarction, peak at 24 hrs, return to normal after 48 hrs

OTHER DIAGNOSTICS

ECG

- Can confirm diagnosis; time sensitive, not accurate after 6 hours
- *< 30 min:* ST segment elevation
  - Only seen in STEMIs
  - ST depression/no ST segment deviation would be seen in NSTEMIs
- *< 24 hrs:* T wave inversion
- *> 24 hrs:* Q waves appear

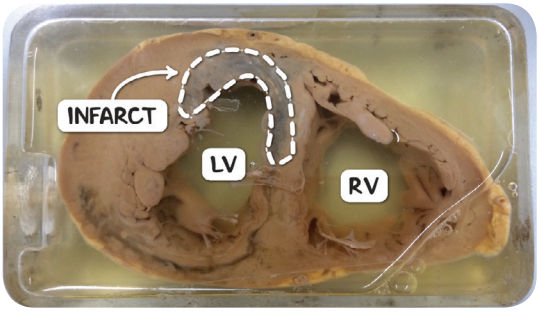

Figure 1.2 A pathology pot containing a heart exhibiting an anterior myocardial infarction, usually the result of left anterior descending artery disease.

TREATMENT

- *STEMI first priority:* emergent reperfusion via percutaneous coronary intervention (e.g. catheterization)/thrombolysis
  - Very time sensitive
- *NSTEMI:* reperfusion via percutaneous coronary intervention (not thrombolysis)
  - Less time sensitive than in STEMI

MEDICATIONS

- Heparin, aspirin + clopidogrel, beta blockers, ACE inhibitors, statins
- Control symptoms with morphine and nitroglycerin

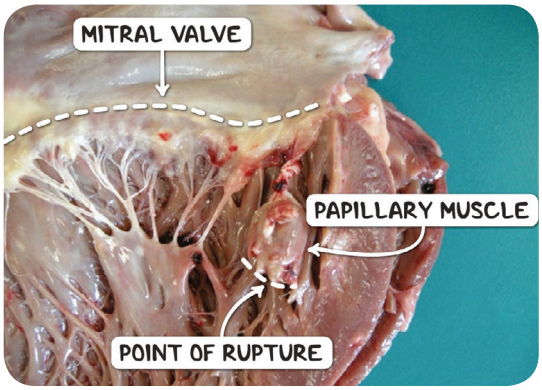

Figure 1.3 Gross pathology of a ruptured papillary muscle, a serious complication of myocardial infarction.

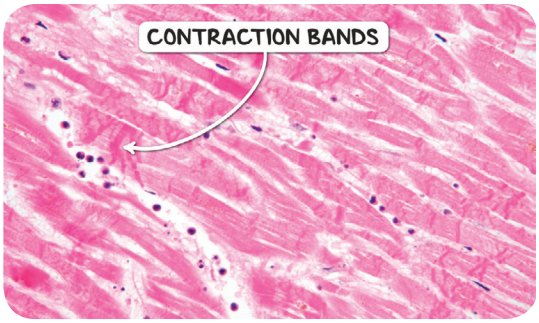

Figure 1.4 Histological appearance of the myocardium following a myocardial infarct.

PRINZMETAL'S ANGINA

osms.it/prinzmetals-angina

PATHOLOGY & CAUSES

- AKA vasospastic angina/variant angina

CAUSES

- Coronary artery vasospasms; occur spontaneously even at rest
- *Vasospasms:* constriction of the smooth muscle surrounding the artery, reducing blood flow through the vessel
  - Cause of vasospasms not well understood; likely due to vasoconstrictors such as platelet thromboxane A2
  - Coronary artery vasospasms cause ischemia throughout all of the heart layers

SIGNS & SYMPTOMS

- Same as stable angina, except pain may occur at rest
- Pain described as pressure, squeezing, burning, or tightness; can radiate to the either/both arms, jaw, shoulders, and back; lasts less than 20 minutes
- *Other symptoms:* Levine's sign (a clenched fist held over the chest), dyspnea, diaphoresis, fatigue, nausea, and epigastric pain

DIAGNOSIS

DIAGNOSTIC IMAGING

- Transient ST segment elevation
- Illustrates transmural ischemia

TREATMENT

MEDICATIONS

Calcium channel blockers

Vasodilators

- Increases coronary blood flow to heart

LOCATION OF STEMI IN ECG

INFARCT LOCATION	LEADS WITH ST ELEVATION OR Q WAVES
ANTEROSEPTAL (LAD)	V1 - V2
ANTEROAPICAL (DISTAL LAD)	V3 - V4
ANTEROLATERAL (LAD or LCX)	V5 - V6
LATERAL (LCX)	I - aVL
INFERIOR (LCX)	II, III, aVL
POSTERIOR (PDA)	V7-V9, ST depression in V1-V3 with tall R waves

UNSTABLE ANGINA

osms.it/unstable-angina

PATHOLOGY & CAUSES

- Episodic chest pain that either
  - Is new in onset
  - Occurs at rest unpredictably
  - Rapidly worsens over time
  - Occurs within 48 hrs after acute MI
- Usually caused by ruptured atherosclerotic plaque → formation of thrombosis on top of plaque → almost complete blockage in blood vessel → ischemia → pain
- *Medical emergency:* high risk of progression to MI
  - *Angina:* myocytes still alive
  - *Myocardial infarction:* death of myocytes

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- *Other symptoms:* Levine's sign (a clenched fist held over the chest), dyspnea, diaphoresis, fatigue, nausea, and epigastric pain

DIAGNOSIS

LAB RESULTS

- Serial troponins measured for individuals with unstable angina to rule out myocardial infarction
- Troponin released after injury to myocytes is a marker for myocardial injury

OTHER DIAGNOSTICS

ECG

- Can present with ST segment depression
  - May also present with T wave inversions
  - Illustrates subendocardial ischemia

TREATMENT

- Unstable angina, NSTEMIs are indistinguishable at initial evaluation
  - Elevated troponins indicating myocardial infarction not detectable for hours
  - Initial management identical

MEDICATIONS

Clopidogrel

Low-molecular-weight-heparin (LMWH)

- Prevents clot formation

Enoxaparin

- Drug of choice based on empirical evidence