CHEST PAIN

KEVAL SHAH
INTERVENTIONAL CARDIOLOGY
4/24/2026

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DISCLOSURES

None.

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GOALS

- Typical vs atypical presentation
- Red flags vs low risk features
- Urgent Care vs Emergency Department vs Clinic work up
- Risk reduction prevention and when to start medications

- 2021 ACC/AHA Chest pain guidelines
- Recent patient cases

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- Second most common reason for emergency department visits in the country.
- Often the etiology of chest pain is benign.
- Goal is to rule out life threatening conditions –
  - Acute coronary syndrome
  - Aortic dissection
  - Pulmonary embolism
  - Esophageal rupture

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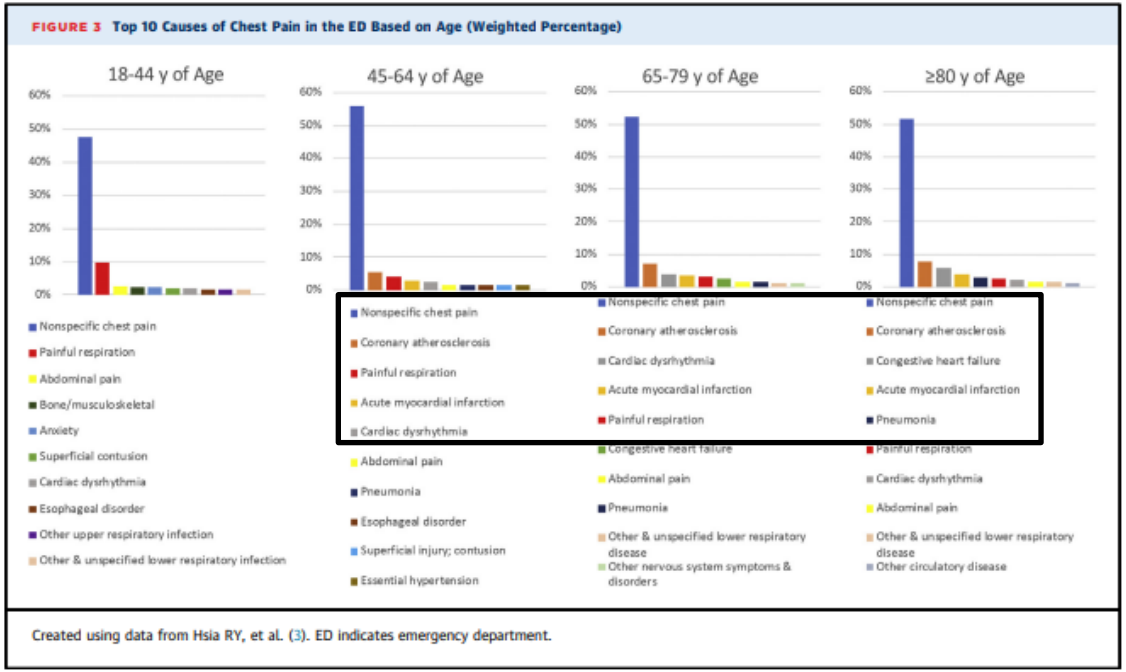

Created using data from Hsia RY, et al. (3). ED indicates emergency department.

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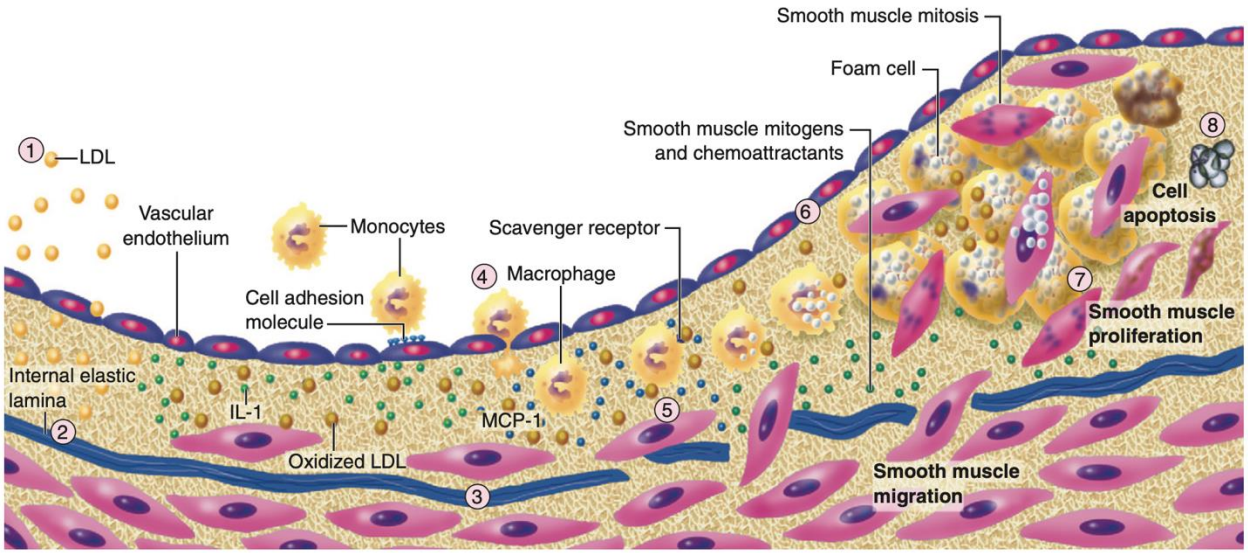

Evolution of atherosclerotic plaque

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- Acute coronary syndrome is caused by
    - Plaque rupture
    - Plaque erosion
    - Thrombosis of calcified nodule

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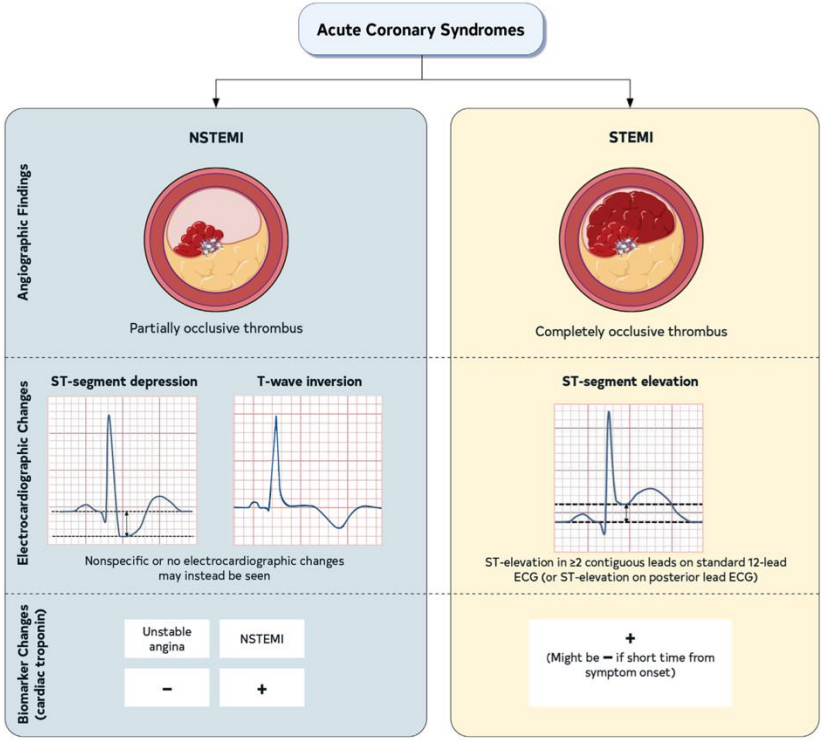

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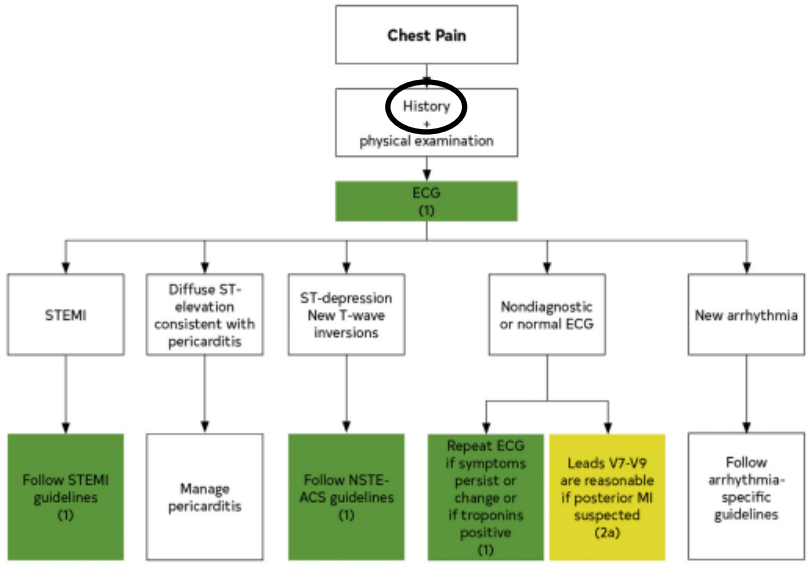
FIGURE 4 Electrocardiographic-Directed Management of Chest Pain



ECG indicates electrocardiogram; NSTE-ACS, non-ST-segment-elevation acute coronary syndrome; MI, myocardial infarction; and STEMI, ST-segment-elevation myocardial infarction.

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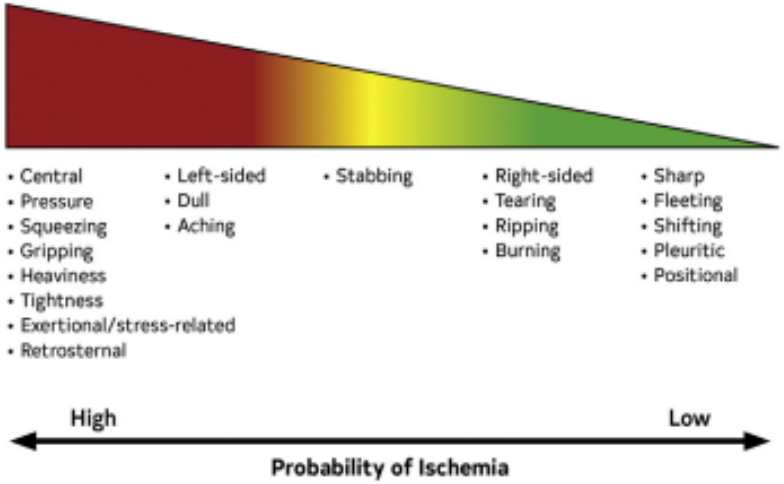
FIGURE 2 Index of Suspicion That Chest "Pain" Is Ischemic in Origin on the Basis of Commonly Used Descriptors



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-Cardiac symptoms
  - Symptoms gradually build up over a few minutes.
  - Symptoms can be triggered with physical exertion or emotional stress.
  - Associated symptoms include dyspnea, nausea, diaphoresis, near syncope or syncope.

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- Noncardiac symptoms
  - -Pleuritic chest pain.
  - - Sudden onset of severe/ripping chest pain more likely associated with aortic dissection.
  - - Chest pain lasting a few seconds.
  - - Chest pain that can be localized to a small area.
  - - Positional chest pain or pain with movements.
  - - Pain radiating to lower abdomen/hips.

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- Time of onset of symptoms
  - Recognized in the 80s that there was a circadian variation in presentation of acute MI and stroke, with an increase in incidence between 6am to noon.
  - Physician health study showed an increased incidence of AMI between 4 am to 10 am.
  - In ISIS-2 trial, time of onset was prospectively determined in ~12,000 patients, with a peak period from 6 am to 11 am.
  - Smaller studies showed an increased incidence in the 1st hour after waking up.

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Circadian variation thought to be secondary to an increase in –

- Plasma catecholamine levels
- Sympathetic activity
- Heart rate and blood pressure
- Vascular tone
- Platelet aggregation

There is also a decrease in protective factors such as vagal activity and fibrinolytic activity.

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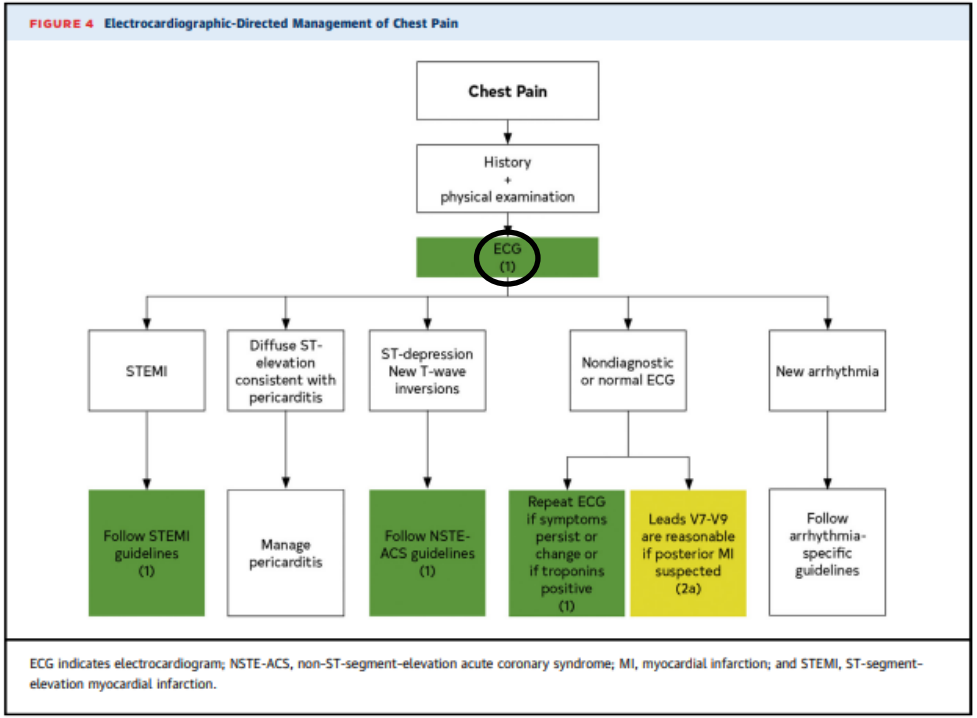

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81y/o male with dyslipidemia and diabetes mellitus type II, forgets bag at airport, and is hurrying back to look for it, when he develops chest pressure with burning, and dyspnea. He goes into the bathroom, and falls, found on the ground. EMS is called and he is brought into the emergency department.

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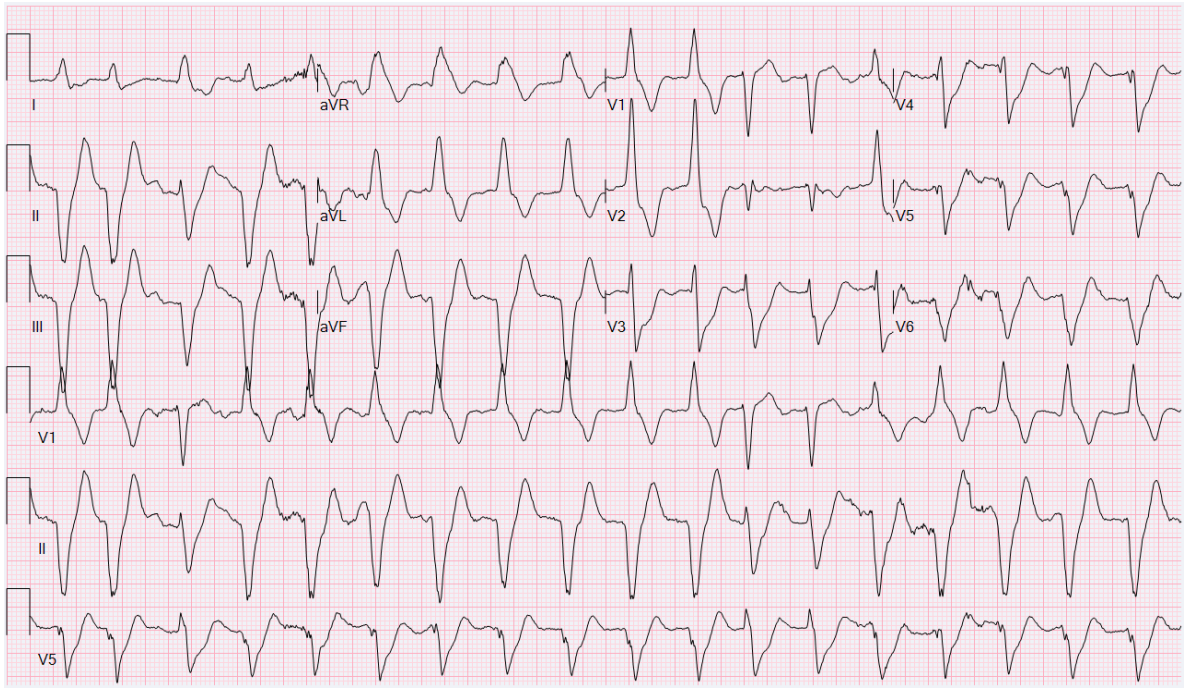

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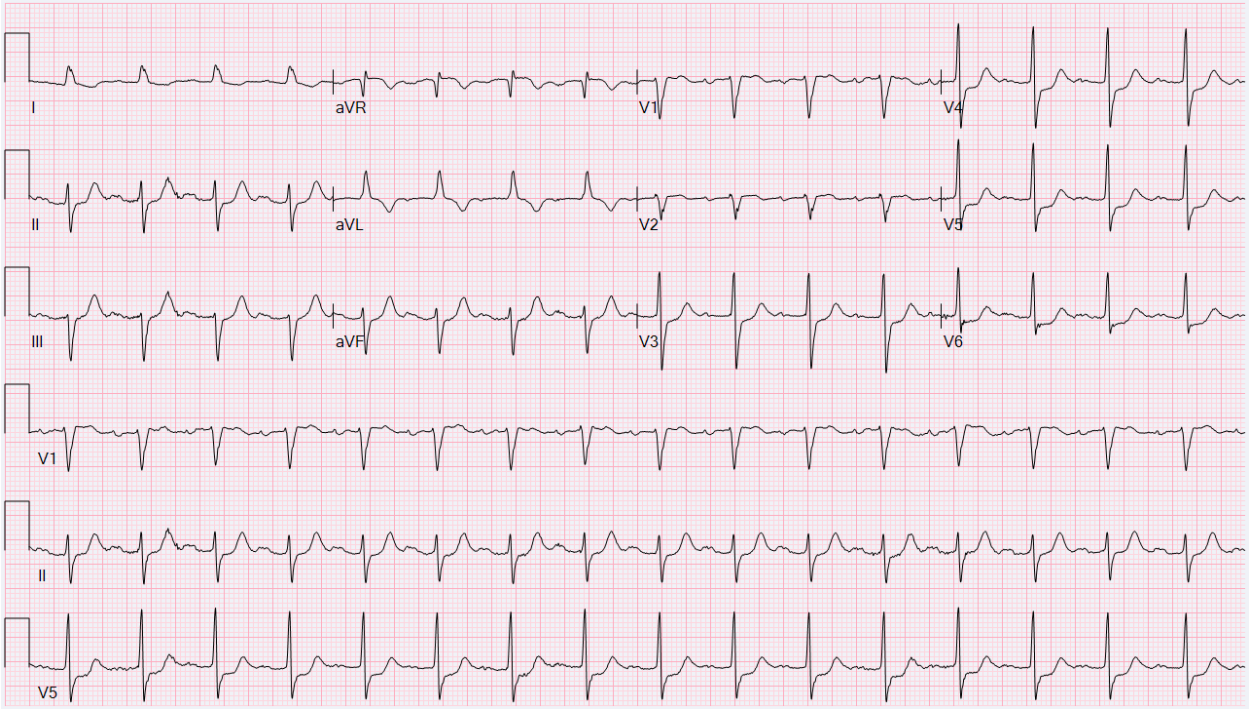

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- Admitted and treated per NSTEMI pathway.
- Develops rising high sensitivity troponin.
- Echocardiogram next day shows LVEF of 35-40% with wall motion abnormality in anterior, anteroseptal, and anterolateral leads.
- Coronary angiogram shows severe, thrombotic, distal left main stenosis extending into the left anterior descending and left circumflex coronary arteries.
- Has IABP placed and Cardiac surgery consulted.
- Turned down for CABG and undergoes Impella assisted PCI.

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- Diffuse ST depressions with ST elevation in lead aVR is indicative of diffuse cardiac ischemia.
- Concern for left main stenosis vs multivessel disease.
- Can be seen in patients with cardiac arrest.

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- 46y/o male with hypertension, dyslipidemia, tobacco use, with witnessed cardiac arrest at home, with bystander CPR, initial rhythm on EMS arrival ventricular fibrillation, ROSC after 20-30 mins. Brought into the emergency department and ECG obtained.

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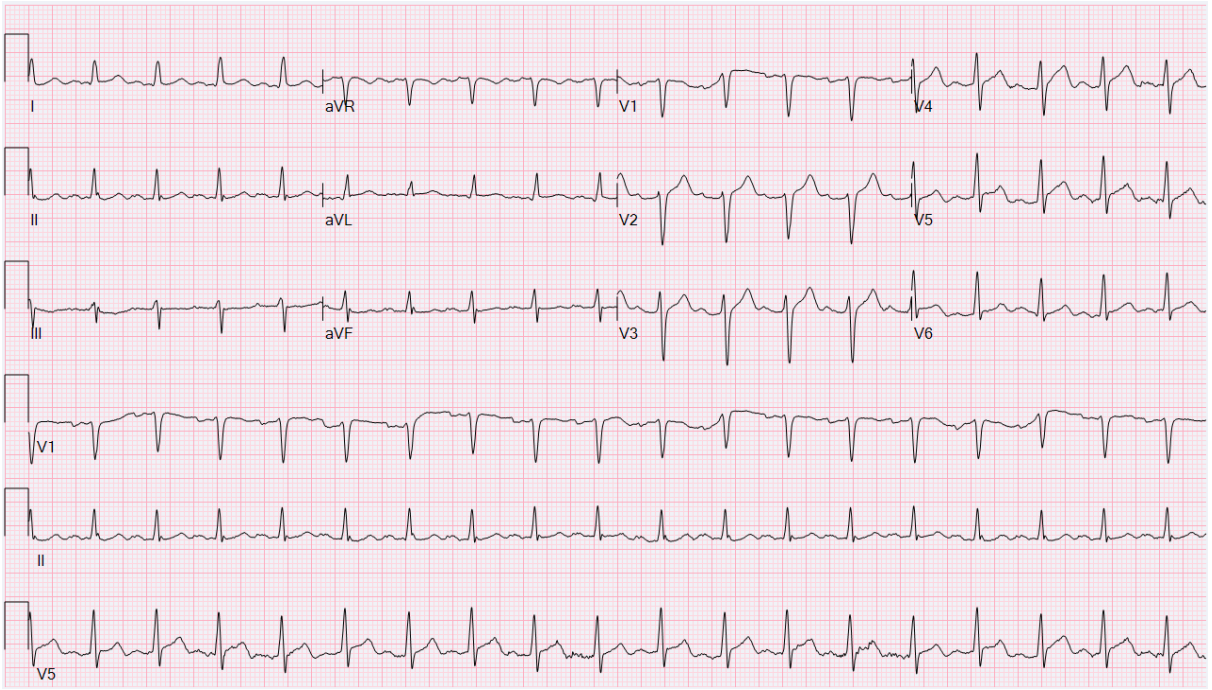

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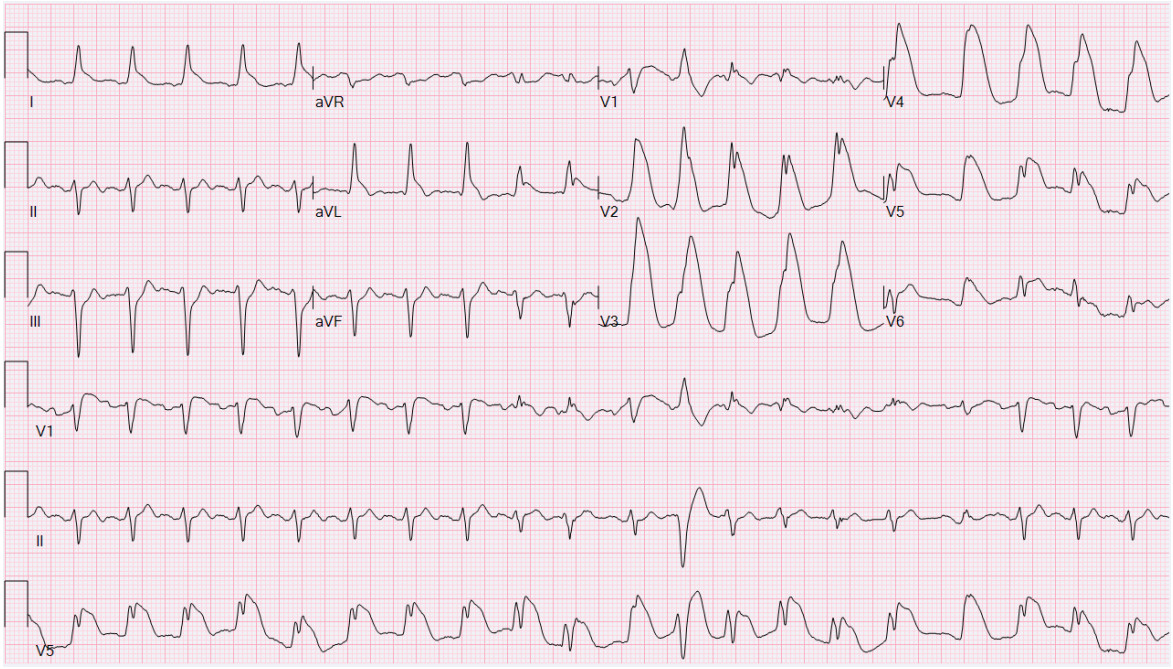

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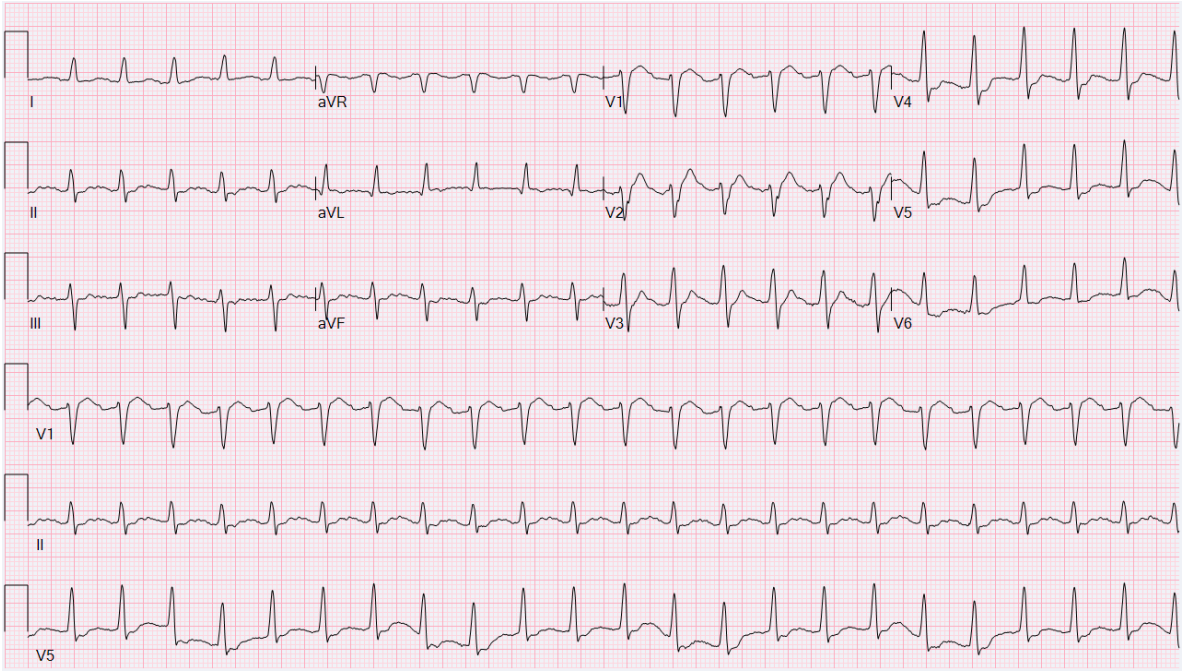

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- Patient in cardiogenic shock after ROSC.
- Undergoes Impella left ventricular assist device placement.
- Coronary angiogram shows severe thrombotic stenosis of mid left anterior descending coronary artery s/p PCI and stent placement.
- Impella weaned down and taken out two days later. Patient extubated shortly after Impella removal and has full neurologic recovery.

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- Importance of repeating ECGs if persistent chest discomfort/symptoms.

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42 y/o male with no prior medical history presents with dizziness and endorses chest discomfort.

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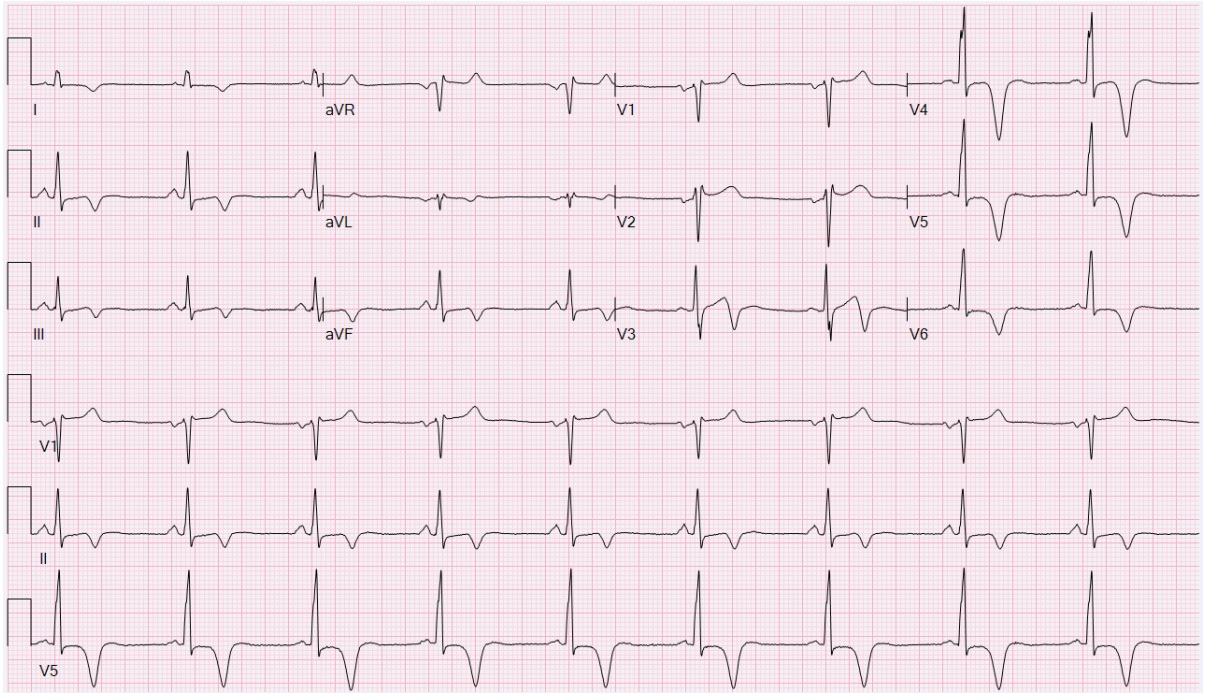

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- Serial high sensitivity troponin remain negative.
- Echocardiogram shows apical hypertrophy, consistent with apical hypertrophic cardiomyopathy.

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83y/o female s/p pacemaker placement presents with intermittent chest discomfort with nausea and vomiting, for two days. Initial high sensitivity troponin is elevated.

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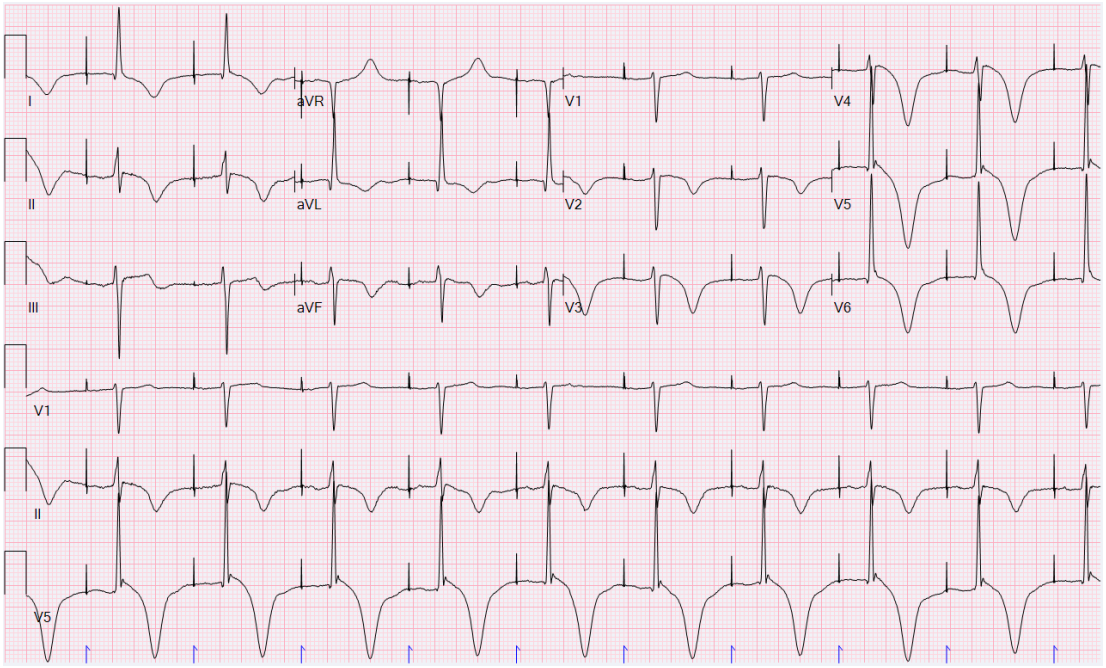

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- Echocardiogram shows LVEF 25-30%, with wall motion abnormality of mid and apical segments.
- Coronary angiogram shows mild coronary artery disease.
- Diagnosis of Takotsubo's cardiomyopathy is made.

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Differential for 'Wellens' T wave abnormality

- Left main vs proximal left anterior descending coronary artery stenosis
- Takotsubos or stress induced cardiomyopathy
- Apical hypertrophic cardiomyopathy
- Acute neurologic process – subarachnoid hemorrhage vs ischemic stroke

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54y/o male with diabetes mellitus type 2, presents with substernal chest pressure, with burning, worse with inspiration.

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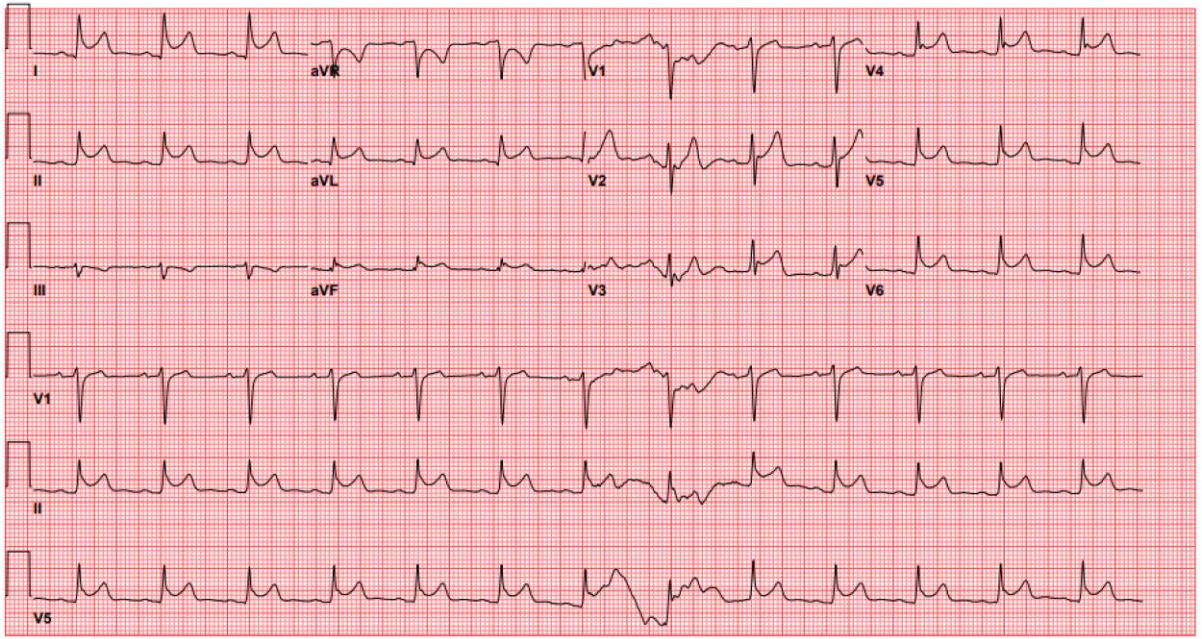

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- CT chest showed inflammation of ascending aorta and small pericardial effusion .
- Suspicion of giant cell arteritis, and treated with steroids.

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29y/o female with prior bioprosthetic mitral valve replacement, presents with sudden onset dyspnea and chest pressure, starting when climbing stairs, and now present at rest.

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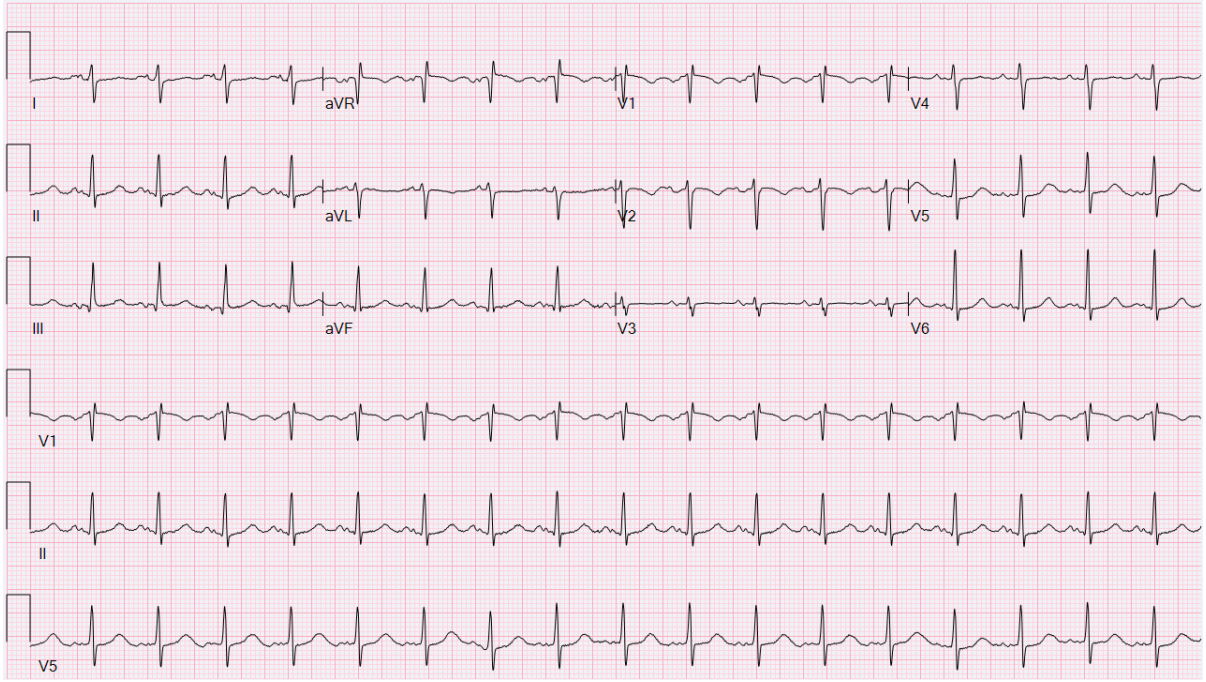

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- Echocardiogram shows flail mitral leaflet with severe mitral regurgitation.
- Right heart catheterization shows mildly elevated right sided filling pressures, severely elevated left sided filling pressures, severe pulmonary hypertension, and low cardiac output.
- Patient undergoes IABP placement.
- Undergoes mitral valve replacement via right minithoracotomy.

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- Sinus tachycardia in patients with acute cardiac abnormality is not benign.

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60y/o female with no prior cardiac history, presents with chest pressure of 2 days, radiating to back, and worse with deep breathing. Serial high sensitivity troponin are negative.

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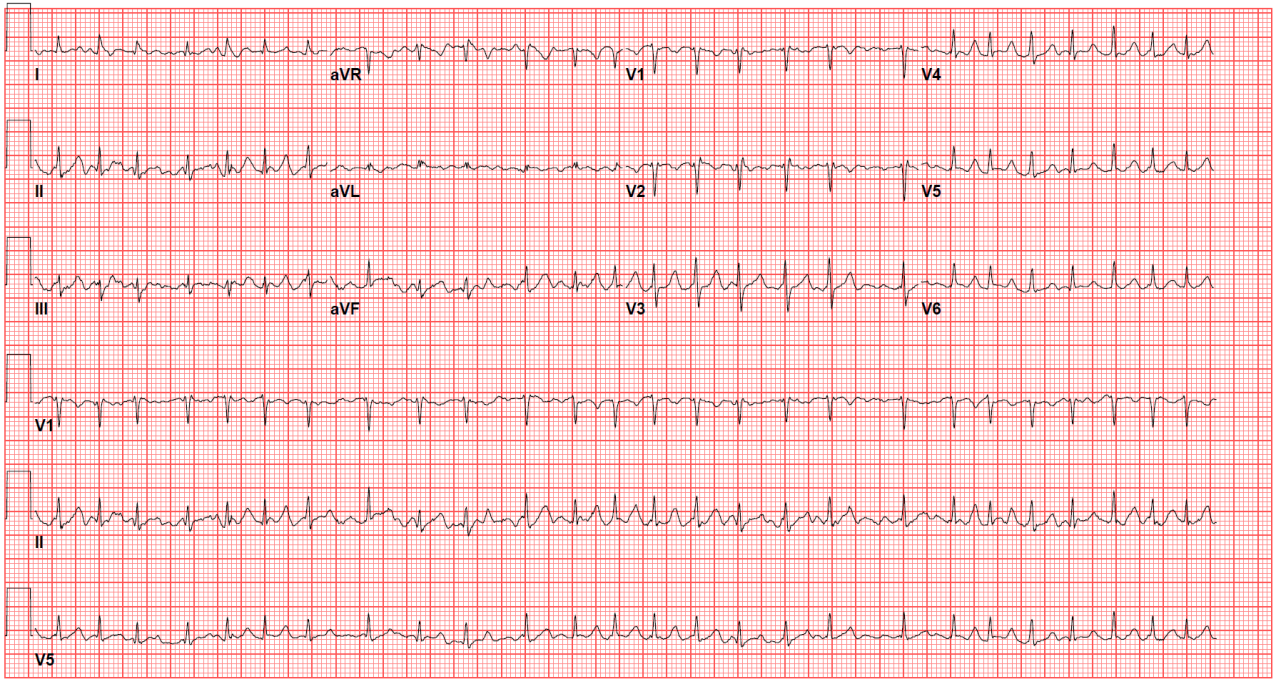

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- Echocardiogram shows normal LVEF and no significant valvular heart disease.
- Patient discharged on metoprolol, digoxin and rivaroxaban.
- Readmitted two weeks later for continued chest pain.
- CT chest shows large pericardial effusion. Confirmed on echocardiogram, with evidence for tamponade.
- Underwent pericardiocentesis with removal of ~400 mls of serous fluid.

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- Importance of history in evaluating chest pain/discomfort.

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35y/o male with hyperlipidemia and family history of premature CAD, presents with two episodes of shock like chest discomfort, associated with dyspnea an diaphoresis. Endorses fatigue with daily activities since the episodes.

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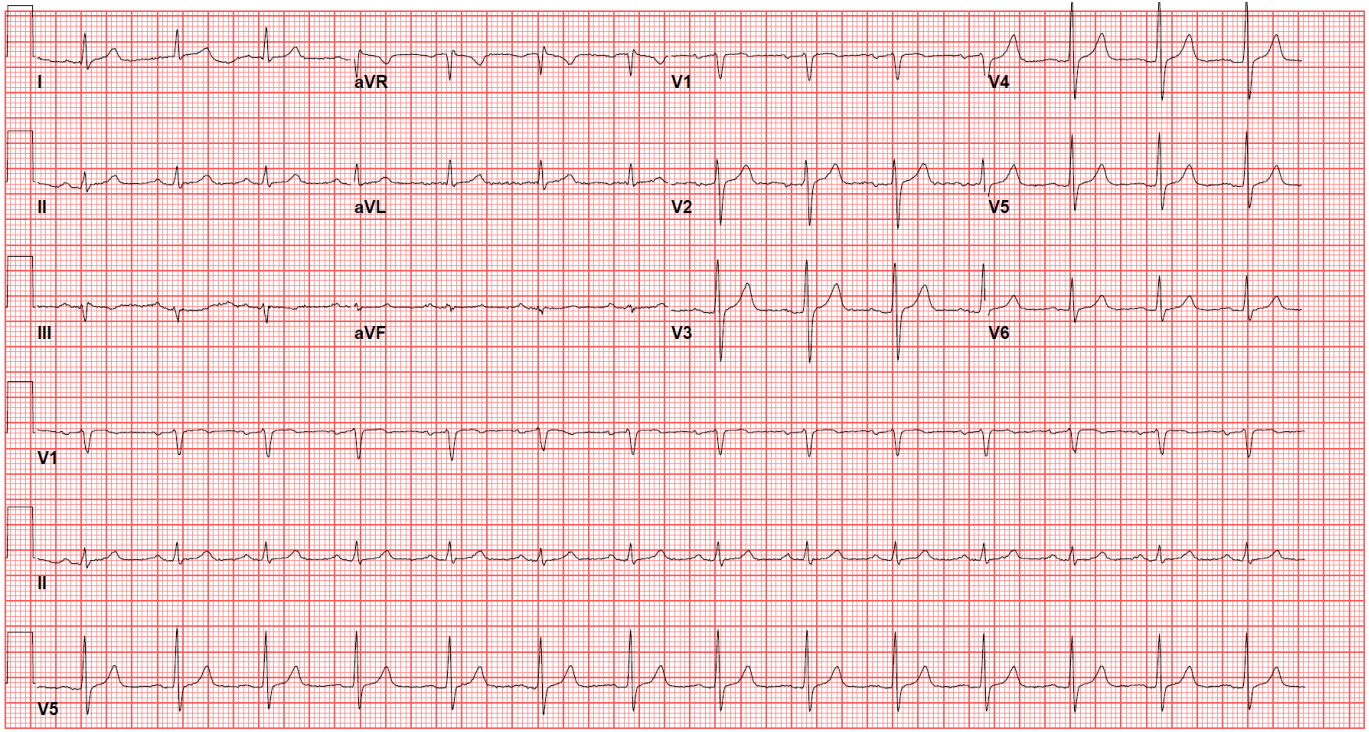

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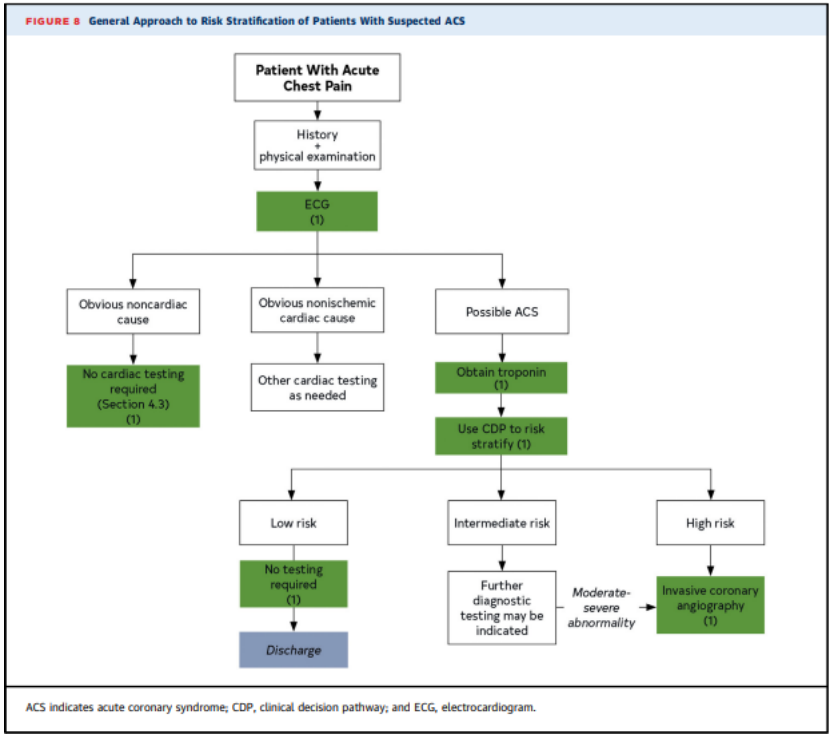

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TABLE 6 Sample Clinical Decision Pathways Used to Define Risk

	HEART Pathway (31)	EDACS (44)	ADAPT (mADAPT) (45)	NOTR (34)	2020 ESC/hs-cTn* (46,47)	2016 ESC/GRACE (11,38)
Target population	Suspected ACS	Suspected ACS, CP >5 min, planned serial troponin	Suspected ACS, CP >5 min, planned observation	Suspected ACS, ECG, troponin ordered	Suspected ACS, stable	Suspected ACS, planned serial troponin
Target outcome	↑ ED discharge without increasing missed 30-d or 1-y MACE	↑ ED discharge rate without increasing missed 30-d MACE	↑ ED discharge rate without increasing missed 30-d MACE	↑ Low-risk classification without increasing missed 30-d MACE	Early detection of AMI; 30-d MACE	Early detection of AMI
Patients with primary outcome in study population, %	6–22	12	15	5–8	9.8	10–17
Troponin	cTn, hs-cTn	hs-cTn	cTn, hs-cTn	cTn, hs-cTn	hs-cTn	cTn, hs-cTn
Variables used	History ECG Age Risk factors Troponin (0, 3 h)	Age Sex Risk factors History Troponin (0, 2 h)	TIMI score 0-1 No ischemic ECG changes Troponin (0, 2 h)	Age Risk factors Previous AMI or CAD Troponin (0, 2 h)	History ECG hs-cTn (0, 1 or 2 h)	Age HR, SBP Serum Cr Cardiac arrest ECG Cardiac biomarker Killip class

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HEART score for chest pain patients

History (Anamnesis)	Highly suspicious	2	
	Moderately suspicious	1	
	Slightly suspicious	0	
ECG	Significant ST-deviation	2	
	Non-specific repolarisation disturbance / LBBB / PM	1	
	Normal	0	
Age	≥ 65 years	2	
	45 – 65 years	1	
	≤ 45 years	0	
Risk factors	≥ 3 risk factors *or* history of atherosclerotic disease	2	
	1 or 2 risk factors	1	
	No risk factors known	0	
Troponin	≥ 3x normal limit	2	
	1-3x normal limit	1	
	≤ normal limit	0	
		Total	

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HEART	~ % pts	MACE/n	MACE	Death	Proposed Policy
0-3	32%	38/1993	1.9%	0.05%	Discharge
4-6	51%	413/3136	13%	1.3%	Observation, risk management
7-10	17%	518/1045	50%	2.8%	Observation, treatment, CAG

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- ESC 2023 ACS guidelines recommends use of 0h/1h high sensitivity troponin algorithm.
- ACC/AHA 2025 ACS guidelines endorses use of 0h/1h or 0h/2h high sensitivity troponin algorithm.
- For 0h/1h high sensitivity troponin algorithm, Positive predictive value is >70%, and, Negative predictive value is >99%.
- For our hospital's high sensitivity troponin assay, patients are ruled out if delta is <20 or if <50.

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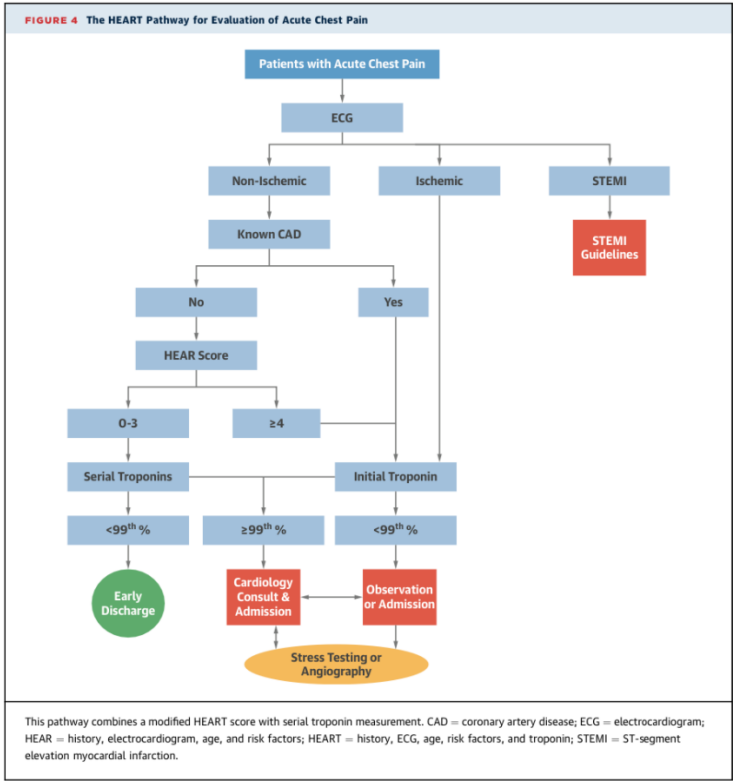

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Pretest likelihood of CAD	Low →	No testing necessary →	Option for CAC for ASCVD risk stratification		
	Intermediate-high →	Younger patient (<65 y of age)	**OR**	Less obstructive CAD suspected →	CCTA favored
	Intermediate-high →	Older patient (≥65 y of age)	**OR**	More obstructive CAD suspected →	Stress testing favored

	Favors use of CCTA	Favors use of stress imaging
Goal	• Rule out obstructive CAD • Detect nonobstructive CAD	• Ischemia-guided management
Availability and expertise	• High-quality imaging and expert interpretation routinely available	• High-quality imaging and expert interpretation routinely available
Likelihood of obstructive CAD	• Age <65 y	• Age ≥65 y
Prior test results	• Prior functional study inconclusive	• Prior CCTA inconclusive
Other compelling indications	• Anomalous coronary arteries • Require evaluation of aorta or pulmonary arteries	• Suspect scar (especially if PET or stress CMR available) • Suspect coronary microvascular dysfunction (when PET or CMR available)

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Stress testing information					
	ETT	Stress echocardiography	SPECT MPI	PET MPI	Stress CMR MPI
Patient capable of exercise	✓	✓	✓		
Pharmacologic stress indicated		✓	✓	✓	✓
Quantitative flow				✓	✓
LV dysfunction/scar		✓	✓	✓	✓

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35y/o male with hyperlipidemia and family history of premature CAD, presents with two episodes of shock like chest discomfort, associated with dyspnea an diaphoresis. Endorses fatigue with daily activities since the episodes.

- Cholesterol of 249, LDL-C of 141
- High sensitivity troponin of 343
- Heart score of 4
- Underwent a coronary angiogram, negative for obstructive CAD

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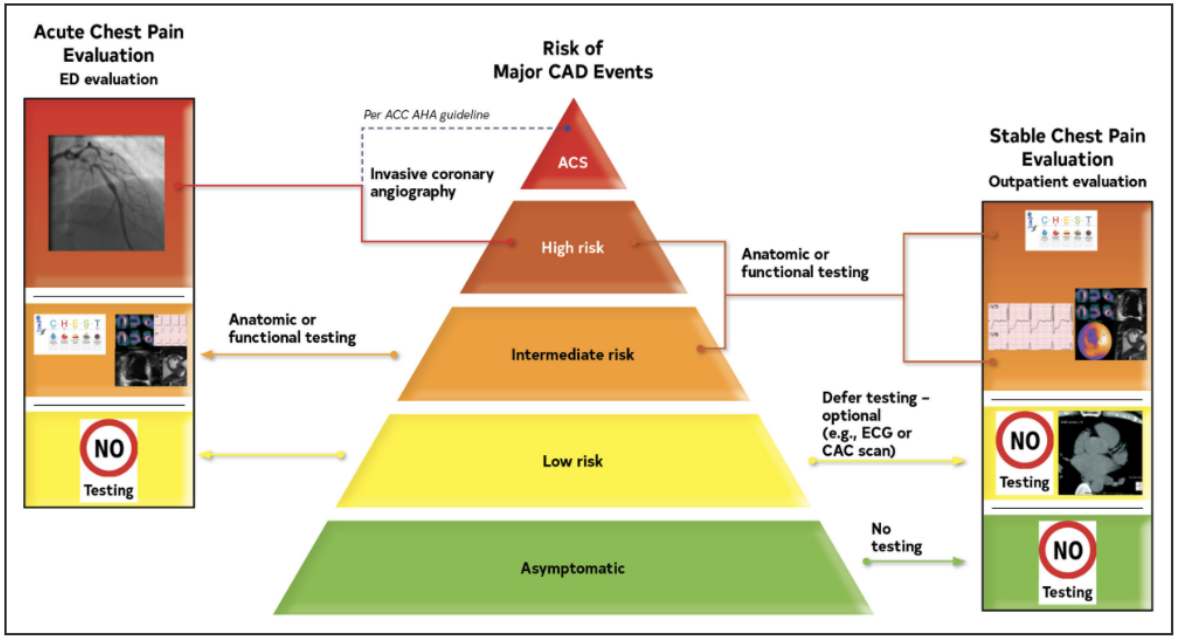

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Pretest Probabilities of Obstructive CAD in Symptomatic Patients
(A) according to age, sex, and symptoms;
(B) according to age, sex, symptoms, and CAC

Age, y	Chest Pain		Dyspnea	
	Men	Women	Men	Women
30–39	≤4	≤5	0	3
40–49	≤22	≤10	12	3
50–59	≤32	≤13	20	9
60–69	≤44	≤16	27	14
70+	≤52	≤27	32	12

A **Pretest probability based on age, sex, and symptoms**

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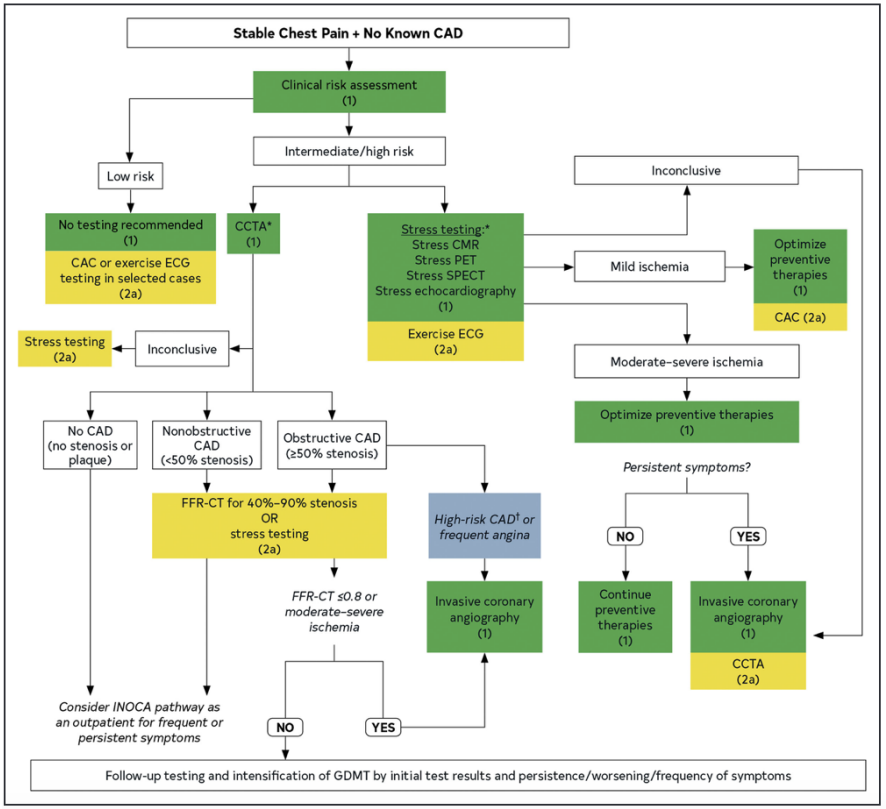

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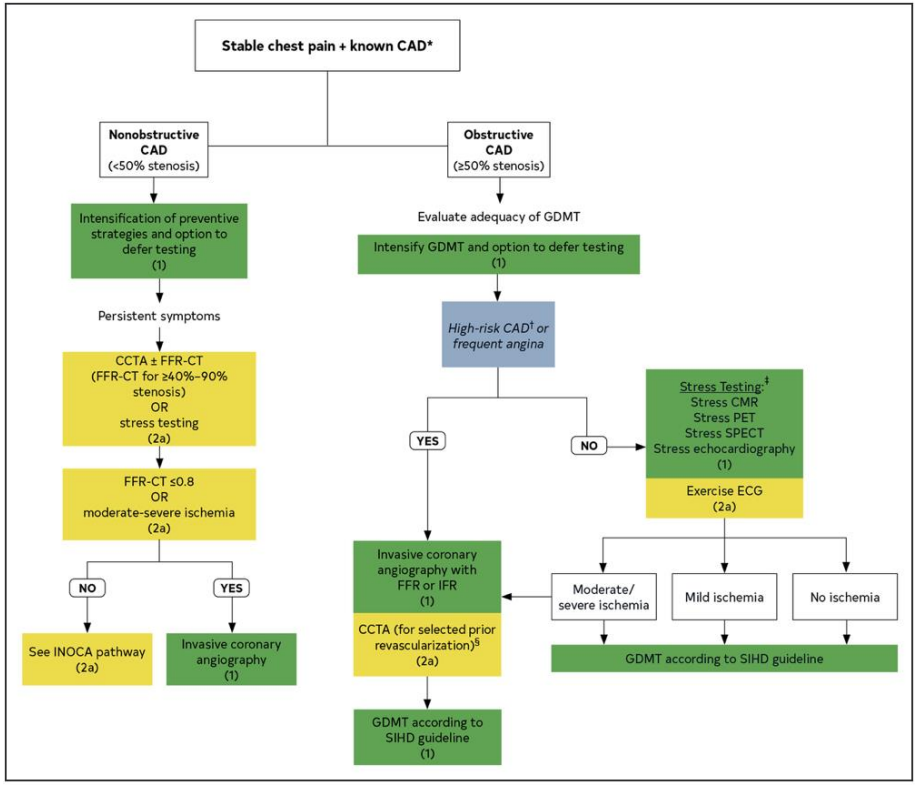

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Coronary artery calcium score.

- Coronary artery calcium score of 0 does not rule out soft plaque.
- Coronary artery calcification is present in 15-20% of patients with coronary atherosclerosis.
- Increased incidence seen in patients on statins and PCSK9 inhibitors, thought to signify stable plaque.
- As previously mentioned, <5% of patients presenting with ACS, develop it secondary to thrombosis of calcified nodules.

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- There has been no randomized control trial that has shown using coronary artery calcium score to guide treatment decisions improves survival, reduces heart attack or strokes, or lowers healthcare costs compared with simpler approaches.

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Medications to consider
- High intensity statin
- Aspirin
- Cardio selective beta blockers
- Calcium channel blockers

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- Aspirin is no longer recommended for primary prevention.
- IVUS based studies showing decrease in plaque volume after ~18 months of high intensity statin therapy.
- ISCHEMIA trial showed medical therapy is equivalent to revascularization.

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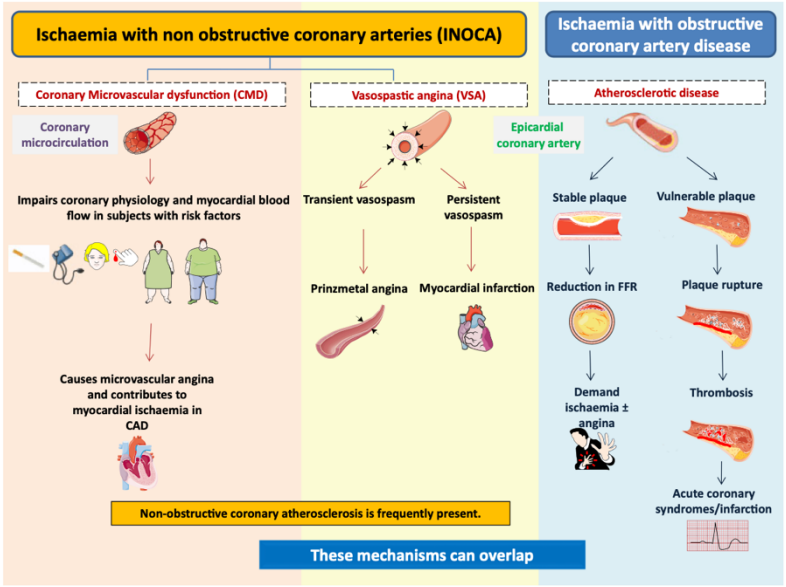
Incidence ~50% in women, and, ~30% in men.



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Microvascular dysfunction

- Coronary flow reserve is the ratio of hyperemic coronary flow to resting coronary flow – utilize to diagnose microvascular dysfunction.
- Can be measured non-invasively using Cardiac PET – Gold standard.
- Invasive measurements can be done using doppler velocity wire vs thermodilution technique.

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Coronary artery spasm

- Endothelial dysfunction and primary hyperreactivity of vascular smooth muscle cells predispose to coronary artery spasm.
- ~75% of patients with coronary artery spasm are active smokers.
- Acetylcholine provocation testing can help assess for coronary artery spasm.

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CONCLUSIONS

- Importance of history and ECG in evaluating for cardiac causes of chest discomfort.
- Consider emergency department/inpatient evaluation for acute chest pain, and, outpatient evaluation for stable chest pain.
- Use of clinic decision pathways and/or high sensitivity troponin for risk stratification of patients with acute chest pain.
- Medications for primary and secondary prevention.

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QUESTIONS

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