



Acute Coronary Syndromes

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Disclosures

- None

Objectives

- Review the underlying pathophysiology of coronary artery disease and acute coronary syndromes (ACS)
- Review the updated recommendations on medical and invasive management of ACS
- Discuss the management of cardiogenic shock

Objectives

2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/ American Heart Association Joint Committee on Clinical Practice Guidelines

Sunil V. Rao, MD, FACC, FSCAI, Michelle L. O'Donoghue, MD, MPH, FACC, FAHA, Marc Ruel, MD, MPH, FACC, FAHA, Tanveer Rab, MD, FACC, MSCAI, Jaqueline E. Tamis-Holland, MD, FACC, FAHA, FSCAI, John H. Alexander, MD, MHS, FACC, FAHA, Usman Baber, MD, MS, FACC, FSCAI, ... [SHOW ALL ...](#) , and Marlene S. Williams, MD, FACC | [AUTHOR INFO & AFFILIATIONS](#)

Epidemiology

Coronary Artery Disease



Affects >18 million adults in the United States

Leading cause of death annually

Every 40 seconds, someone is having an acute myocardial infarction (MI)

The annual incidence of acute MI is ~800,000

Gulati et al. 2021 Circulation; Tsao et al. Circulation 2023

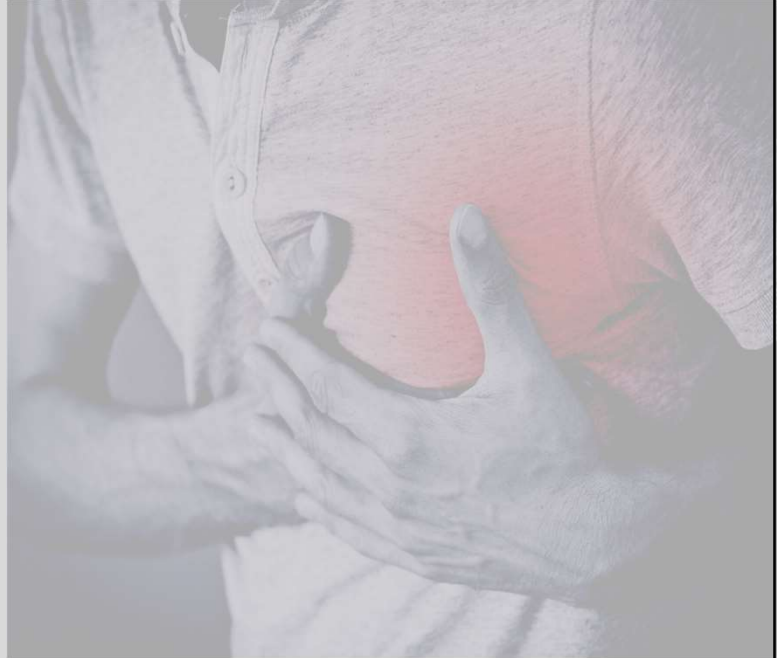
Chest Pain

The most common symptom of coronary disease

Annually:

- >6.5 million ED visits
 - 4 million outpatient visits
- ~5% of patients in the emergency department with chest pain will have an acute coronary syndrome

Gulati et al. 2021 Circulation



Pathophysiology

Plaque Rupture

Disruption of unstable atherosclerotic plaque

- Thin fibrous cap
- Large lipid core



Partial or complete thrombosis resulting in myocardial ischemia

- Fibrin-rich thrombus

Rao et al. 2025 Circulation; Libby, P. 2024 Circulation; Luo et al. 2021 Front Cardiovasc Med

Plaque Erosion

Degradation of endothelial cells and collagen

- Thick fibrous cap
- Small or absent lipid core



Neutrophil activation leading to thrombus formation

- Platelet-rich thrombus

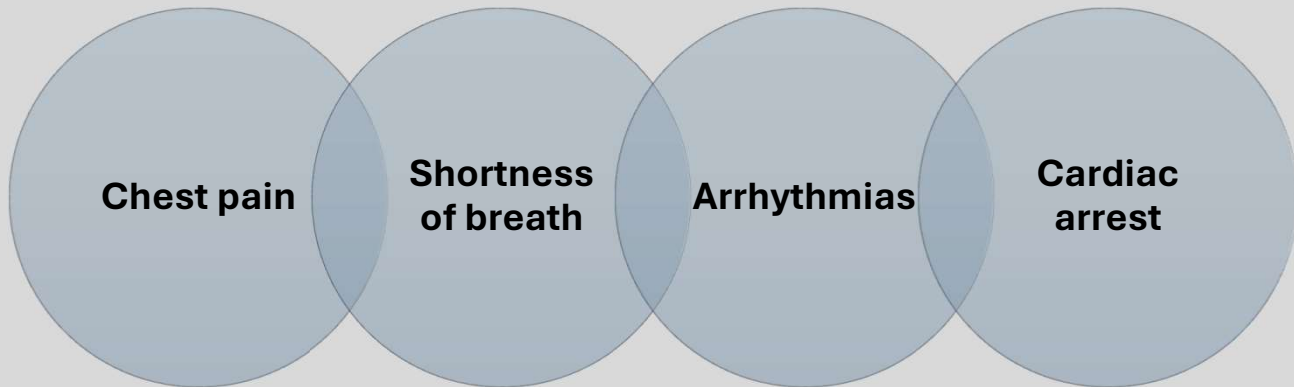
Luo et al. 2021 Front Cardiovasc Med

Other Mechanisms

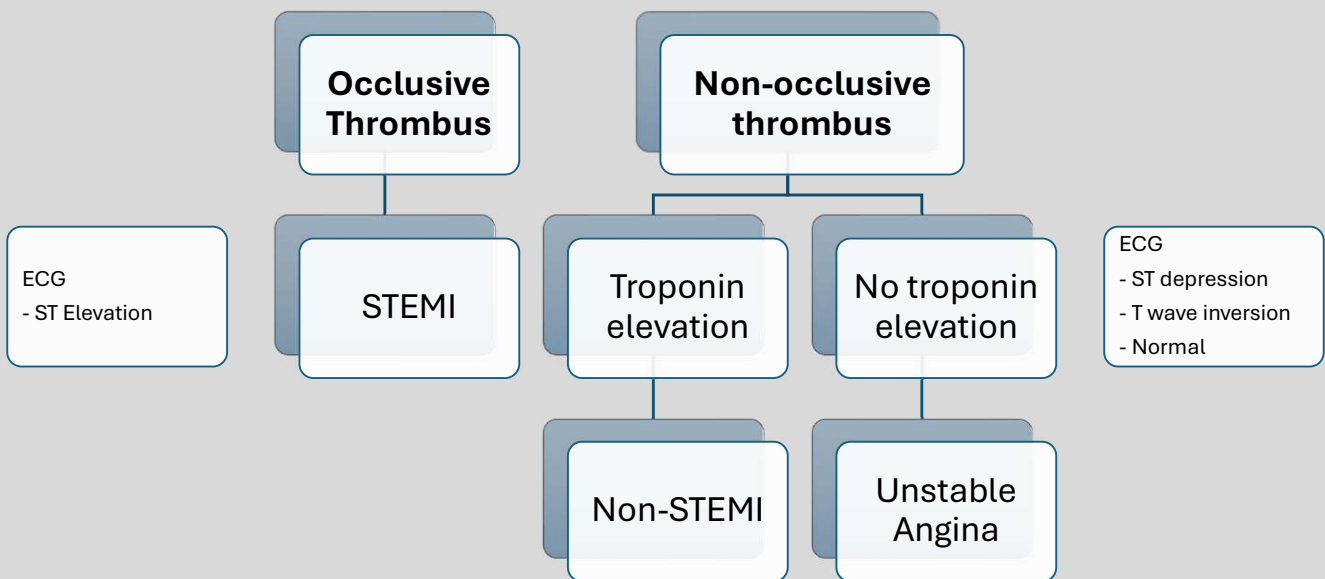
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- Spontaneous Coronary Artery Dissection**
 - Embolism**
 - Microvascular Dysfunction**
 - Vasospasm**

Initial Evaluation

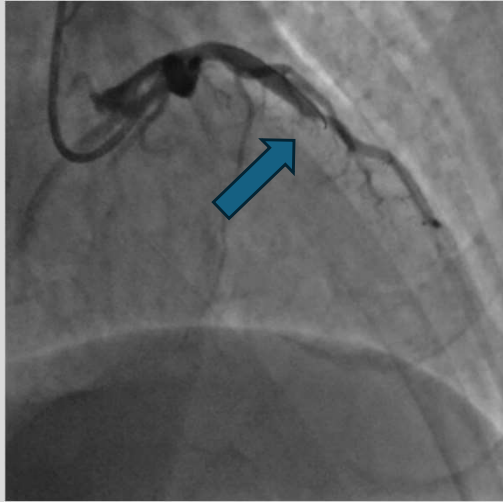
Symptoms



Presentation



Occlusive Thrombus

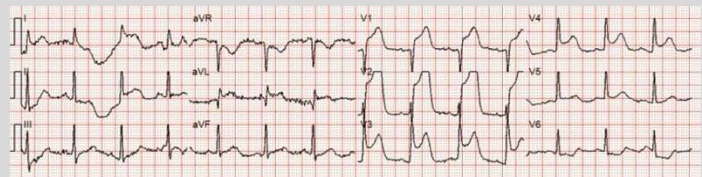
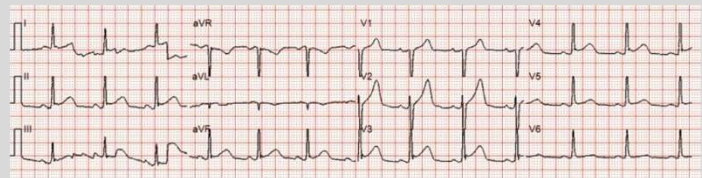
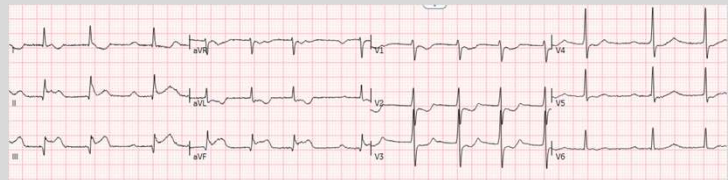


Non-occlusive thrombus

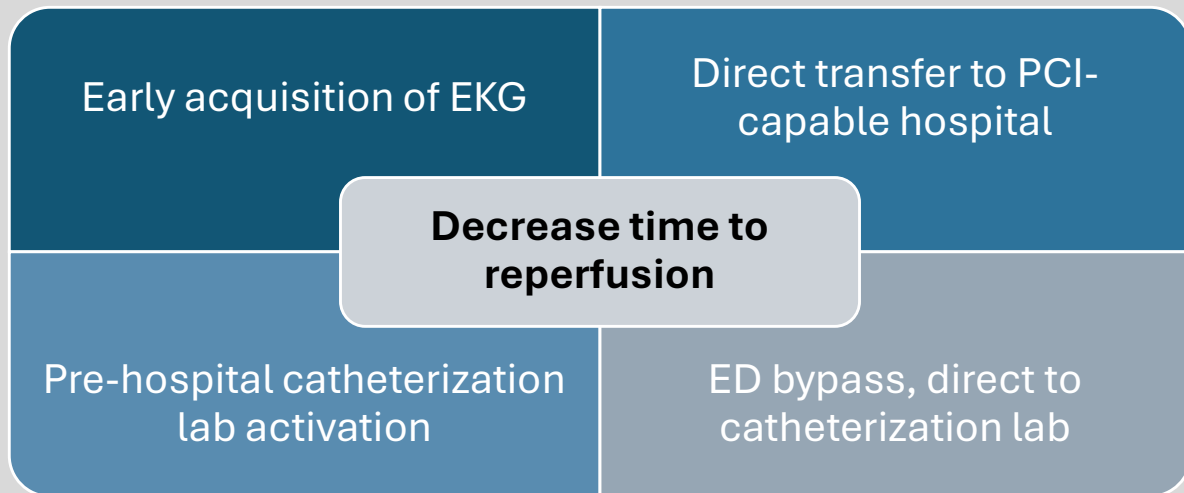


Electrocardiogram (ECG)

- 12-lead ECG should be obtained **within 10 minutes** of first medical contact (Class 1)
- If initial ECG is non-diagnostic, **serial 12-lead ECGs** should be performed if clinical suspicion is high (Class 1)



Pre-Hospital Assessment



In-Hospital Assessment

Troponin should be measured as soon as possible

- High-sensitivity assay preferred

If the initial troponin is non-diagnostic, a repeat should be obtained

- 1-2 hours for high-sensitivity troponin
- 3-6 hours for conventional troponin

Additional Testing

Laboratory Testing

- Complete blood count
- Chemistry panel
- PT/INR

Chest X-ray

- Assess other causes of chest pain

Medical Management

Medical Therapy

Acute treatment

- Platelet activation and aggregation
- Coagulation cascade

Long-term prevention and risk reduction

Analgesia

Rapid and effective pain relief is an important treatment goal

- Nitroglycerin sublingual or intravenous
- Intravenous opioids

Nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided due to increased risk of MACE

Rao et al. 2025 Circulation

Aspirin

Initial loading dose of aspirin 324 mg followed by daily aspirin 81 mg is recommended to reduce death and adverse cardiovascular events (Class 1)

Blocks formation of cyclooxygenase (COX) dependent vasoconstrictors → improves endothelial function

Associated with:

- 23% decrease of vascular mortality rate
- 50% decrease in non-fatal reinfarction or stroke
- 50-70% decrease in fatal or non-fatal myocardial infarction

Rao et al. 2025 Circulation; Dai et al. 2011 Thrombosis

P2Y12 Inhibitors

P2Y12 inhibitors should be used in conjunction with Aspirin for patients with acute coronary syndrome

Blocks adenosine diphosphate (ADP)-mediated activation of platelets

- Decreases incidence of recurrent MACE
- Increased bleeding

Rao et al. 2025 Circulation

P2Y12 Inhibitors

	CLOPIDOGREL	PRASUGREL	TICAGRELOR	CANGRELOR
Platelet inhibition	40-60%	70%	80-90%	95-100%
Pharmacology	Irreversible, pro-drug	Irreversible, pro-drug	Reversible, active drug	Reversible, active drug
Onset	2-4h	30 min	30 min	2 min
Loading Dose	300-600 mg	60 mg	180 mg	30 mcg/kg
Maintenance Dose	75 mg QD	5-10 mg QD	90 mg BID	4 mcg/kg/min
Considerations	Triple therapy	TIA/CVA	Dyspnea, bradycardia	

P2Y12 Inhibitors

- **Prasugrel or ticagrelor** recommended in acute coronary syndrome (Class 1)
- **Clopidogrel** is recommended after fibrinolytic therapy or when prasugrel or ticagrelor are unavailable
- Given immediately prior to or during angiography for STEMI patients
- Routine pre-treatment not needed for patients with unstable angina or NSTEMI who have planned invasive management

Rao et al. 2025 Circulation

Anticoagulation

IV unfractionated heparin

- Upstream therapy reduces ischemic events in NSTEMI
- Should be continued until revascularization

Enoxaparin or fondaparinux

- Alternatives when early invasive approach not anticipated

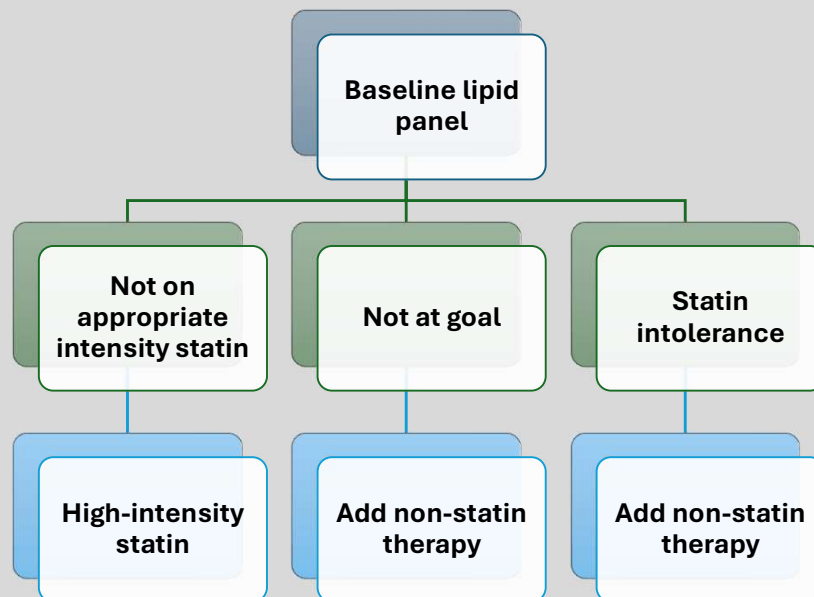
Glycoprotein IIb/IIIa Inhibitors

Block platelet aggregation by preventing platelet cross-linking via fibrinogen or von Willebrand factor binding

Can consider use in patients with large thrombus burden, no-reflow or slow flow (Class 2a)

Should not be used routinely due to lack of ischemic benefit and risk of bleeding (Class 3)

Lipid Management



Beta Blockers

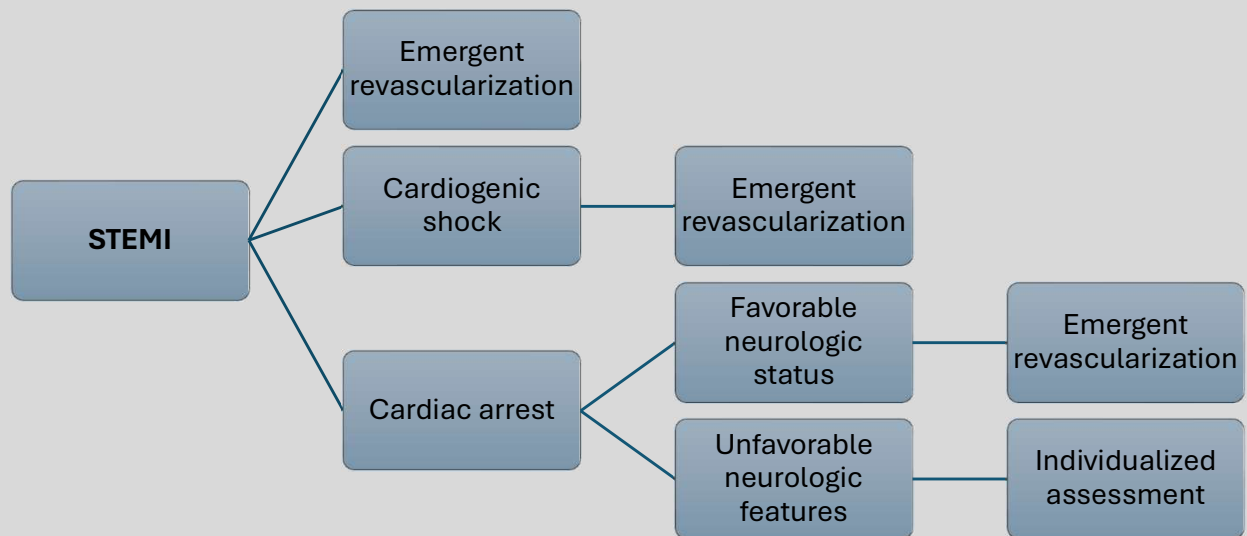
Early initiation of beta blockers to reduce risk of reinfarction and ventricular arrhythmias (Class 1)

Not used in patients with evidence of cardiogenic shock

Newer trial data suggest use of beta blockers may not be beneficial for patients with preserved ejection fraction

- REDUCE-AMI (EF >50%)
- REBOOT-CNIC & BETAMI-DANBLOCK (EF >40%)

Invasive Management



ST-Elevation Myocardial Infarction (STEMI)

Improved prognosis due to coronary reperfusion options

Delay in reperfusion is a determinant of outcome

- One-year mortality increases for each 30-minute delay
- Short- and long-term mortality increases for every 10-minute delay from reperfusion

Door to balloon time <90 minutes

- <120 minutes if being transferred

Pasquale, G. 2022 Int J Cardiol Heart Vasc; Kochan et al. 2023 Circulation: Card Interventions

Cardiac Arrest with STEMI

Approximately 10% of patients with STEMI transferred to the hospital had an out of hospital cardiac arrest

Resuscitated patients with favorable neurologic status who have evidence of STEMI should undergo PCI (Class 1)

Resuscitated patients with unfavorable neurologic prognostic features should undergo individualized assessment (Class 2b)

Rao et al. 2025 Circulation

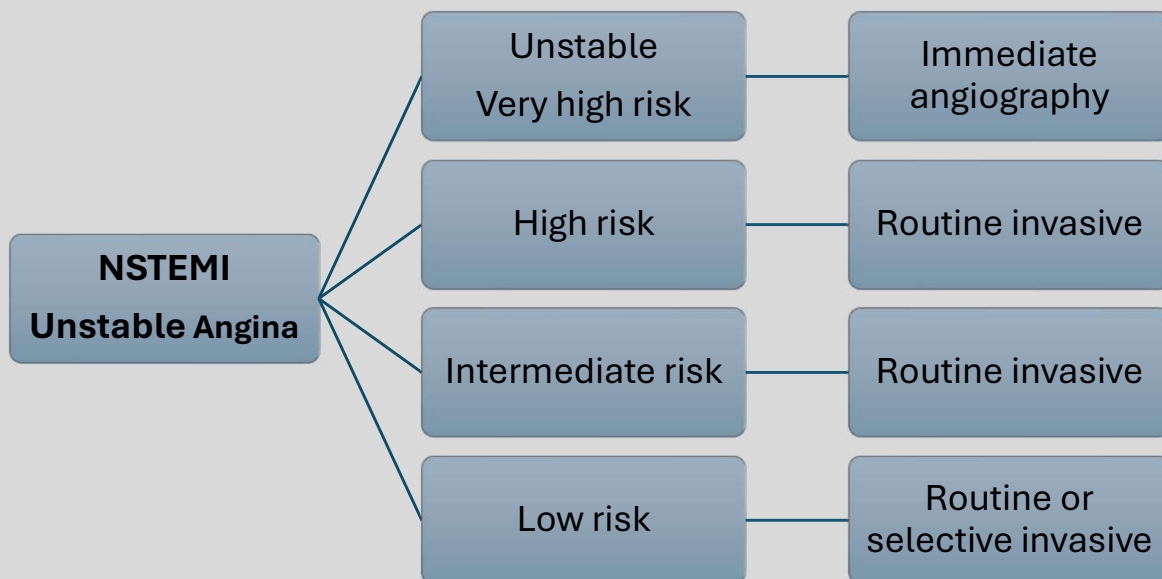
Cardiac Arrest without STEMI

Resuscitated patients who are comatose, without evidence of STEMI should not undergo immediate angiography (Class 3: No Benefit)

Multiple studies show no benefit to early angiography compared to delayed / no angiography

Ischemic evaluation can be performed once patient is improved

Rao et al. 2025 Circulation



Risk Stratification

Global Registry of Acute Coronary Events (GRACE) Risk score

- Estimates mortality after MI

Thrombolysis in Myocardial Infarction (TIMI) Risk Score

- Used to estimate 14-day risk of all-cause mortality, new or recurrent MI or severe ischemia

When NOT to Pursue Routine Angiography

- High bleeding risk
- Advanced kidney disease and acute renal failure
- Limited life expectancy
- Patient preference

Procedural Considerations

Radial approach is preferred

- Associated with significant relative risk reduction of all-cause death and major bleeding
- Decreased vascular complications

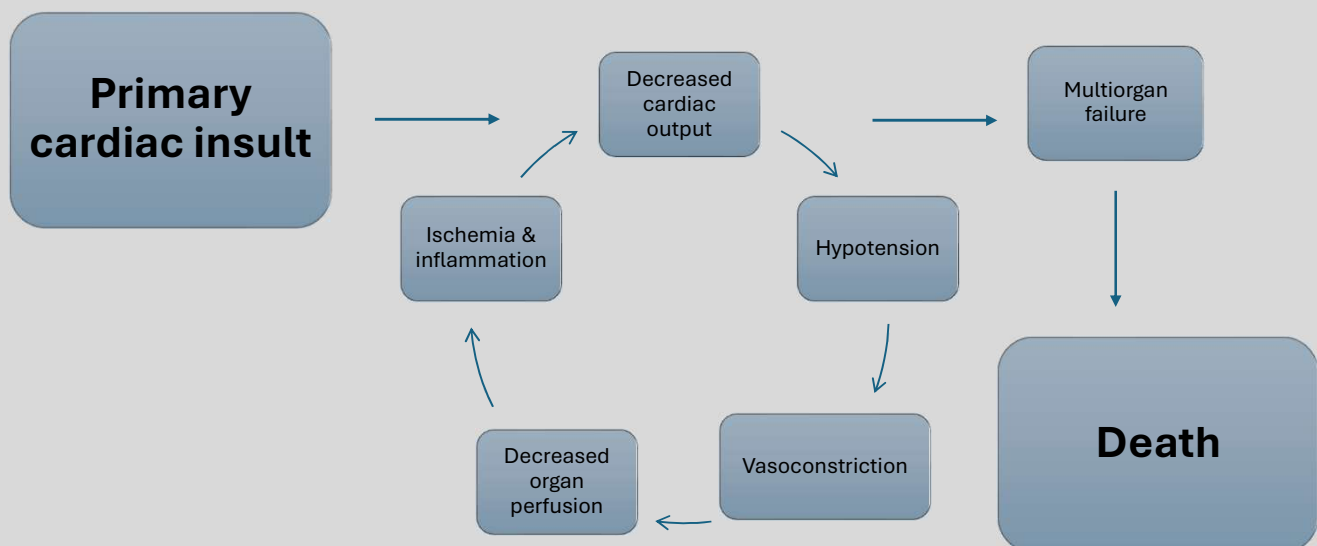
Additional In-Hospital Testing

Assessment of the left ventricular ejection fraction is recommended prior to hospital discharge (**Class 1**)

- Assess for any potential complications
- Help guide medical therapy
- Future need for primary prevention ICD

Cardiogenic Shock

Cardiogenic Shock



Sinha et al. JACC 2025; Tehrani et al. JACC HF 2020

Cardiogenic Shock in Acute MI

- Approximately 80% of cardiogenic shock is due to acute MI
- 5-10% of patients with acute MI will develop cardiogenic shock
- STEMI patients twice as likely to develop shock vs NSTEMI
- Short- and long-term mortality remains ~50%
- Worse outcomes for more advanced stages of shock

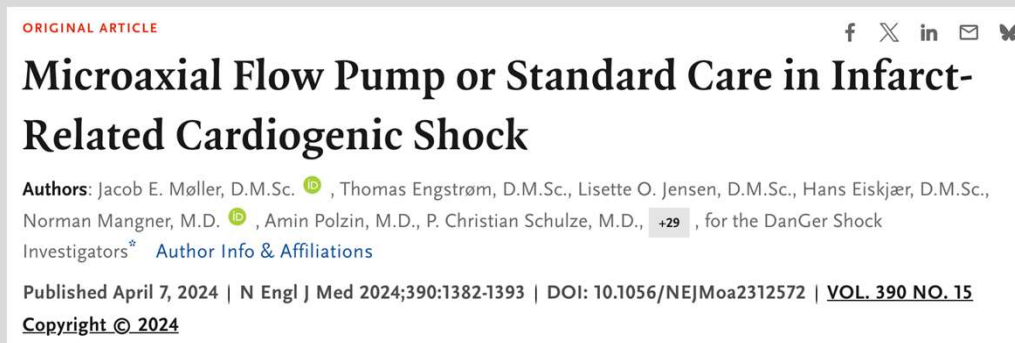
Osman et al. 2021 JAHA; Vahdatpour et al. 2019 JAHA

Management of Cardiogenic Shock

- Recognizing signs and symptoms of cardiogenic shock
- Initiating appropriate pharmacologic therapy
- Interdisciplinary, team-based approach (Shock Team) to deliver individualized management
- Transferring the patient to the appropriate level of care

Temporary Mechanical Support

- In select patients with STEMI-cardiogenic shock with left ventricular dominant shock, evidence of clinical hypoperfusion and/or hemodynamic deterioration, **escalation to microaxial flow-pump may be considered (Class 2a)**



Temporary Mechanical Support

- Promote ventricular unloading and restore perfusion
- Bridge to recovery or advanced therapies
- Vascular access and complications
- Routine use in all cardiogenic shock patients not recommended

Advanced Therapies

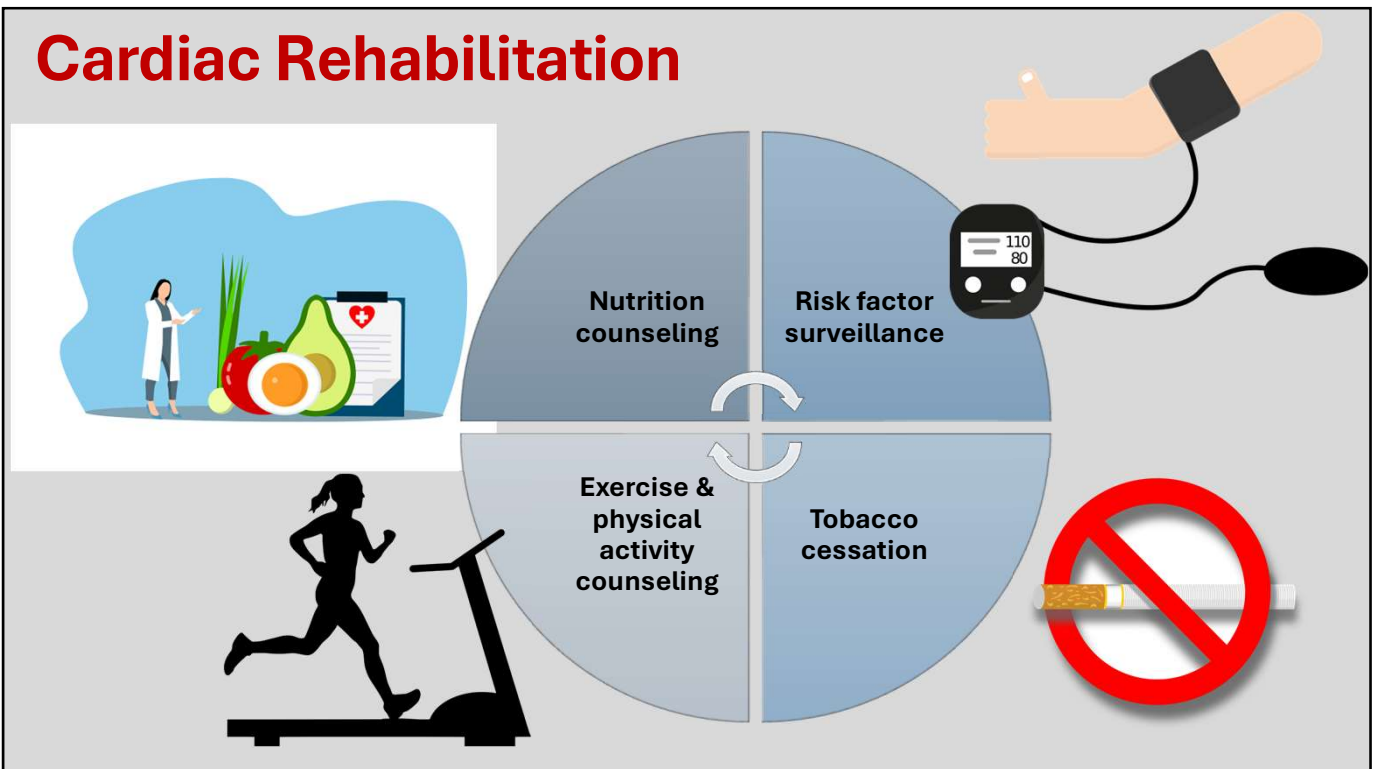
- Multidisciplinary team members including advanced heart failure & transplant can help determine a patient's candidacy for therapies such as durable left ventricular support devices or transplant
- Palliative care & hospice can help with conversations regarding goals of care

Post-Hospital Management

Duration of Dual Anti-Platelet Therapy (DAPT)

Default	Bleeding reduction strategies			High Bleeding Risk
12 months	DAPT 1-3 months	DAPT 1 month	Triple therapy (DAPT + OAC) 1-4 weeks	DAPT 1 month
	Ticagrelor monotherapy	Aspirin + Clopidogrel	Clopidogrel + OAC	Aspirin or P2Y12 monotherapy

Cardiac Rehabilitation



In Summary

- Acute coronary syndrome represents a spectrum of disorders
- Prompt recognition of STEMI improves mortality
- Management of UA/NSTEMI should be tailored to each individual patient
- Cardiogenic shock represents a small percentage of patients with acute MI, but continues to have a high mortality
- Post hospital care and management is crucial to ensure improved outcomes