CARDIOGENIC SHOCK

- Amy Shepard RN, MS, ACNS-BC, CCRN
- STEMI and Cardiogenic Shock Program Manager

LEARNING OBJECTIVES

By the end of this presentation learners will be able to:

1. Identify signs and symptoms of Acute Coronary Syndrome
2. Discuss recent and upcoming advancements in open heart surgery and interventional cardiology
3. Examine the causes and progression of cardiogenic shock

STATISTICS

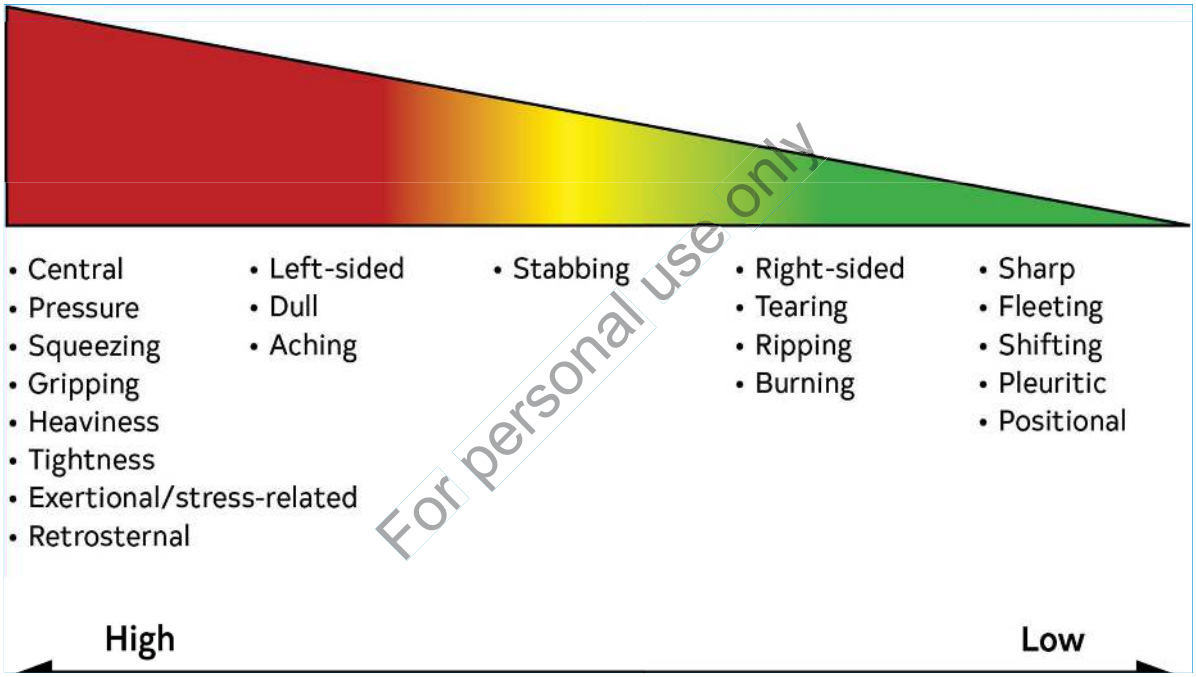
- Every 40 seconds an American will have a heart attack.
- According to data from 2005-2014, the estimated annual incidence of heart attack in the US was 605,000 new attacks and 200,000 recurrent attacks.
- Average age at the first heart attack was 65.6 years for males and 72 for females.
- Coronary heart disease caused 371,506 deaths in 2022.

American Heart Association, At-a glance statistics, 2025

WHAT IS ACUTE CORONARY SYNDROME?

- Acute coronary syndrome (ACS) is a spectrum of conditions compatible with acute myocardial ischemia and/or infarction that are usually due to an abrupt reduction in coronary blood flow.
- They are typically caused by disruption (rupture or erosion) of an unstable coronary artery atherosclerotic plaque with associated partial or complete coronary artery thrombosis and/or micro emboli, resulting in diminished blood flow to the myocardium and subsequent myocardial ischemia.
  - ST-Elevation Myocardial Infarction
  - Non-ST Elevation Myocardial Infarction (NSTEMI)
  - Unstable Angina

Rao, et al. Circulation, 2025

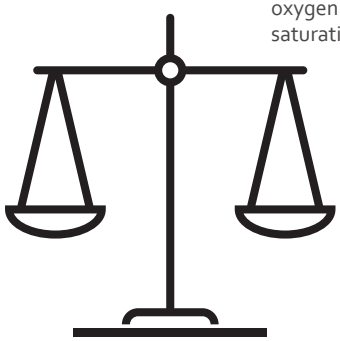


WHAT CAUSES ACS?

- ACS is when there is an imbalance between Myocardia oxygen consumption (MVO_2)and demand.
- Usually occurs due to a coronary artery obstruction.
- **BUT…. NOT ALWAYS!**

Increased Oxygen Demand
- Increased HR
- Increased Blood pressure
- Increased preload
- Increased afterload

Decreased Oxygen Supply
- Decreased coronary blood flow
- Vessel caliber
- Increased HR
- Decreased blood pressure
- Decreased oxygen saturation

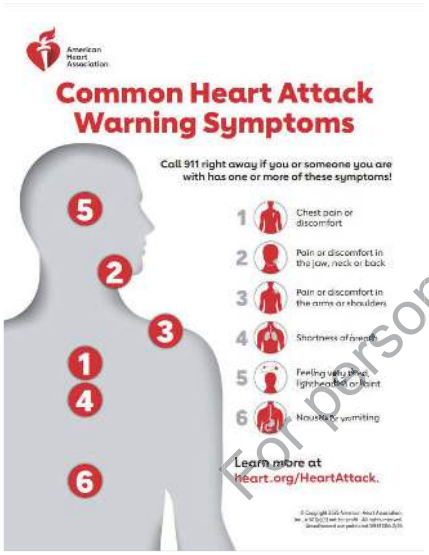


OTHER CORONARY CAUSES OF ACS

- Prinzmetal angina
- Coronary embolism
- Coronary arteritis

RISK FACTORS

- Positive family history
  - Males 55 or younger
  - Females 65 or younger
- Diabetes Mellitus
- Hypertension
- Hyperlipidemia
- Tobacco use
- Male

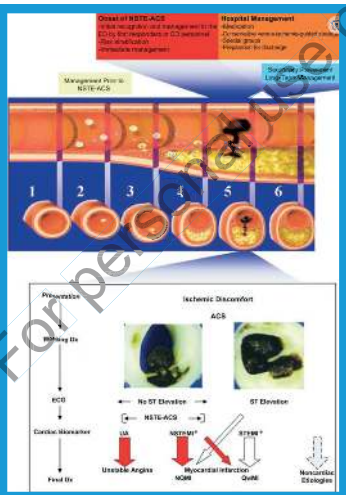


SYMPTOMS OF A HEART ATTACK

American Heart Association, 2025

PROCESS OF ATHEROGENESIS

1. Normal artery
2. Extracellular lipid in the sub intima
3. Fibrofatty Stage



4. Weakening of fibrous cap
5. Disruption of fibrous cap
6. Thrombus resorption

Amsterdam, et al, Circulation, 2014

UNSTABLE ANGINA

- A condition in which a patient may experience symptoms of a myocardial infarction without the release of cardiac biomarkers.
- Ischemia was not severe enough to cause myocardial damage.
- Unstable angina pain or discomfort:
  - Can happen at rest
  - Can come on suddenly
  - May last longer than 15 minutes
  - Usually isn't relieved by rest or medication
  - May get worse over time
- Other symptoms include:
  - Shortness of breath
  - Dizziness
  - Anxiety
  - Sweating
  - Nausea and vomiting

American Heart Association, 2025

Recommendations for Initial In-Hospital Assessment of Patients With Confirmed or Suspected ACS
Referenced studies that support recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	B-NR	1. In patients with suspected ACS, acquisition and interpretation of an ECG within 10 minutes is recommended to help guide patient management.[1,2]
1	B-NR	2. In patients with suspected ACS in whom the initial ECG is nondiagnostic, serial 12-lead ECGs should be performed to detect potential ischemic changes, especially when clinical suspicion of ACS is high, symptoms are persistent, or clinical condition deteriorates.[3]
1	B-NR	3. In patients with suspected ACS, cTn should be measured as soon as possible, preferably using a high-sensitivity cTn (hs-cTn) assay.[4–7]
1	B-NR	4. In patients with suspected ACS with an initial hs-cTn or cTn that is nondiagnostic, the recommended time intervals for repeat measurements after the initial sample collection (time zero) are 1 to 2 hours for hs-cTn and 3 to 6 hours for conventional cTn assays.[8–12]

*Adapted from the "2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain."[13]

NON-ST ELEVATION ACS

- Typically occurs at rest with minimal exertion and lasts ≥ 10 minutes.
- Pain often starts at the retrosternal area and can radiate to either or both arms, the neck, or the jaw.
- Pain may occur in the other areas without chest pain
- Patients may also present with diaphoresis, dyspnea, nausea, abdominal pain, or syncope

Amsterdam, et al., Circulation, 2014

NON-ST ELEVATION ACS (CONT.)

- Unexplained or new-onset increased exertional dyspnea is the most common anginal equivalent.

Amsterdam, et al., Circulation, 2014

ATYPICAL SYMPTOMS

The following patients usually experience typical symptoms but the incidence of non-cardiac symptoms such as epigastric pain, indigestion, stabbing, pleuritic pain, and dyspnea without chest pain.

- Older patients ≥ 75 years of age
- Women
- Diabetes
- Impaired renal function
- Dementia

Amsterdam, et al., Circulation, 2014

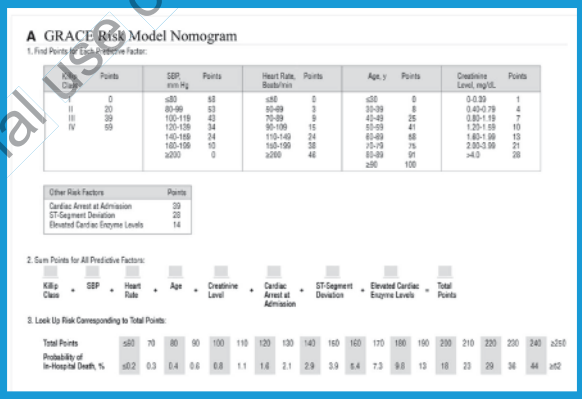
RISK SCORES

Table 3. TIMI Risk Score* for NSTE-ACS

TIMI Risk Score	All-Cause Mortality, New or Recurrent MI, or Severe Recurrent Ischemia Requiring Urgent Revascularization Through 14 d After Randomization, %
0–1	4.7
2	8.3
3	13.2
4	19.9
5	26.2
6–7	40.9

COLLAPSE

The TIMI risk score is determined by the sum of the presence of 7 variables at admission; 1 point is given for each of the following variables: ≥65 y of age; ≥3 risk factors for CAD; prior coronary stenosis ≥50%; ST deviation on ECG; ≥2 anginal events in prior 24 h; use of aspirin in prior 7 d; and elevated cardiac biomarkers.



A GRACE Risk Model Nomogram

1. Find Points for Each Discrete Factor:

Killip Class	Points	SBP, mm Hg	Points	Heart Rate, Beats/min	Points	Age, y	Points	Creatinine Level, mg/dL	Points
I	0	≤80	58	≤50	0	≤30	0	0-0.39	1
II	20	80-99	53	50-69	3	30-39	8	0.40-0.79	4
III	39	100-119	43	70-89	9	40-49	25	0.80-1.19	7
IV	59	120-139	34	90-109	15	50-59	41	1.20-1.59	10
		140-159	24	110-149	24	60-69	58	1.60-1.99	13
		160-199	10	150-199	38	70-79	75	2.00-3.99	21
		≥200	0	≥200	46	80-89	91	>4.0	28
						≥90	100		

Other Risk Factors	Points
Cardiac Arrest at Admission	39
ST-Segment Deviation	28
Elevated Cardiac Enzyme Levels	14

2. Sum Points for All Predictive Factors:

Killip Class + SBP + Heart Rate + Age + Creatinine Level + Cardiac Arrest at Admission + ST-Segment Deviation + Elevated Cardiac Enzyme Levels = Total Points

3. Look Up Risk Corresponding to Total Points:

Total Points	≤80	70	80	90	100	110	120	130	140	150	160	170	180	190	200	210	220	230	240	≥250
Probability of In-Hospital Death, %	≤0.2	0.3	0.4	0.6	0.8	1.1	1.6	2.1	2.9	3.9	5.4	7.3	9.8	13	18	23	29	36	44	≥52

Amsterdam, et al., Circulation, 2014

NSTEMI DIAGNOSIS

- ECG
  - 12 Lead ECG may show ST depression or T wave inversion
    - ***Caution: ST depression in V1-V3 may be a posterior STEMI***
  - Cardiac Biomarkers
    - Troponin I or T should be measured at presentation and 3 to 6 hours after symptom onset – Positive troponin = >0.04
    - Creatine Kinase (CK-MB) and myoglobin are not useful for diagnosis of ACS (class III recommendation by AHA –No benefit)
  - Imaging
    - Chest x-ray
    - Chest CT
    - Transthoracic Echocardiogram

Rao, et al. Circulation, 2025

ACS TREATMENT

- Fentanyl – 50 mcg-100 mcg
- Oxygen – Administer for oxygen saturation <90%
- Nitroglycerin – 0.4mg every 5 minutes up to 3 doses
  Do not administer nitroglycerin to the following patients:
  - Patients with hypotension, ≤90mmHg systolic
  - Patients with RV infarct
  - Patients with severe aortic stenosis (AS)
  - Patients with LV outflow tract obstruction
  - Patients who have taken a phosphodiesterase inhibitor (i.e, Viagra, Sildenafil, etc.) within the last 24 hours. *** 48 hours for Tadalafil**
- Aspirin – 162mg - 324 mg chewed

Rao, et al. Circulation, 2025

OTHER DRUGS

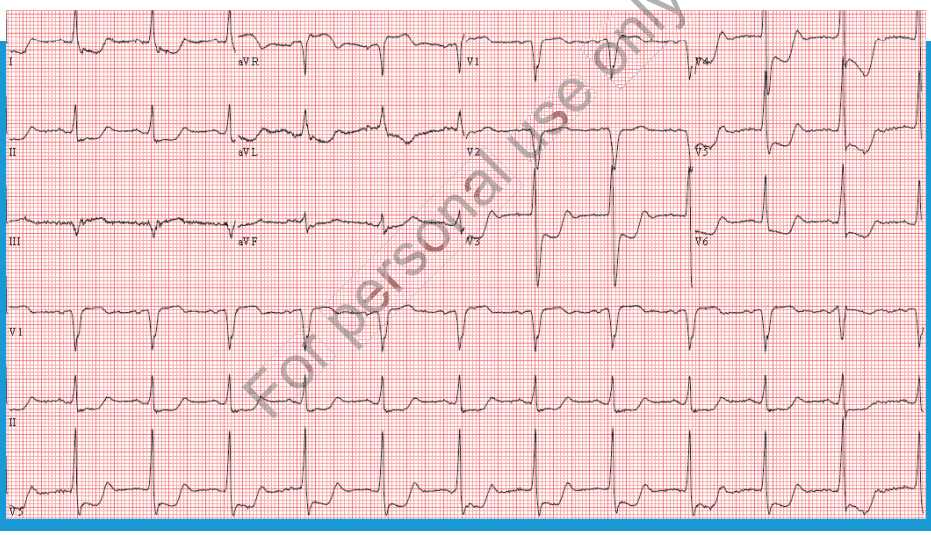
- Enoxaparin 1mg/kg SQ, every 12 hours

OR

- Heparin – 60 U/kg IV bolus followed by 12 IU/Kg/hr infusion
- P2y12 Inhibitors
  - **Ticagrelor (Brillinta)** –180mg, po loading dose, 90mg twice daily. ***Patients on Brillinta should only take 81 mg of ASA per day.***
  - **Clopidogrel (Plavix)** – 600mg, po loading dose, 75 mg once daily
  - **Prasugrel** (Effient) – 60mg,po loading dose, 10mg once daily

Rao, et al. Circulation, 2025

NSTEMI ECG



STEMI

- STEMI is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent electrocardiographic (ECG) ST elevation and subsequent release of biomarkers of myocardial necrosis.

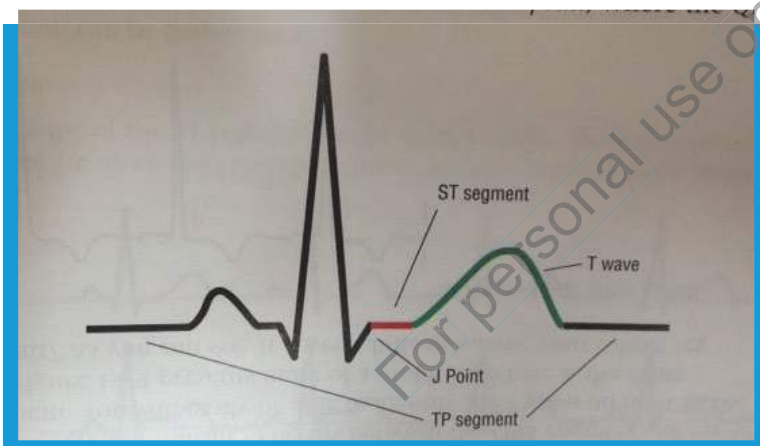
O'Gara, et al., Circulation, 2013

STEMI CRITERIA

- ST elevation at the J point in at least 2 contiguous leads.
- Men ≥2 mm Women ≥1.5 mm in V2-V3
- ≥1 mm in other contiguous chest leads or the limb leads
- ST depression in V1-3 without ST elevation in other leads may indicate a posterior myocardial infarction

O'Gara, et al., Circulation, 2013

WHY IS THE ST SEGMENT SO IMPORTANT?



The ST Segment represents the section of the complex in which the ventricles are between electrical depolarization and repolarization.

It is a key indicator as to whether the patient may be experiencing ischemic insult or injury to the myocardium.

WHAT CAUSES A STEMI?

ST segment

Elevation

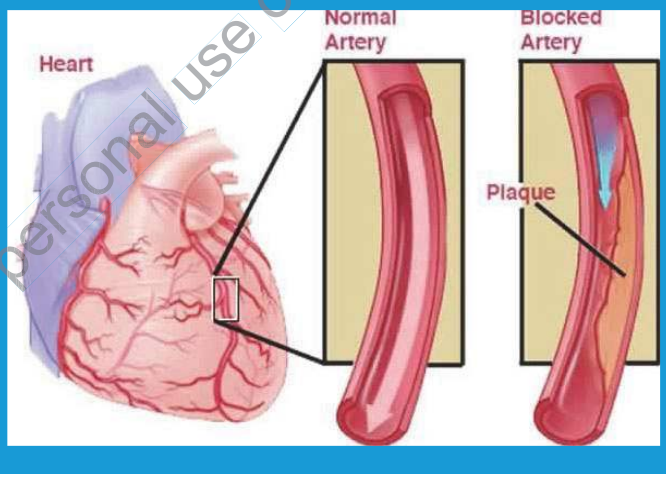
Myocardial

Infarction

1. STEMIs are associated with the build up of plaque which ruptures in the coronary arteries.

2. This stimulates platelets to stick together/aggregate and vessels near the rupture to constrict

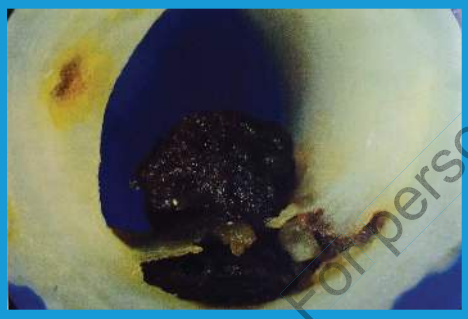
3. If the unstable area becomes totally blocked by a clot, also called a thrombus, a STEMI is occurring.



Acute Coronary Syndrome

RUPTURED PLAQUE

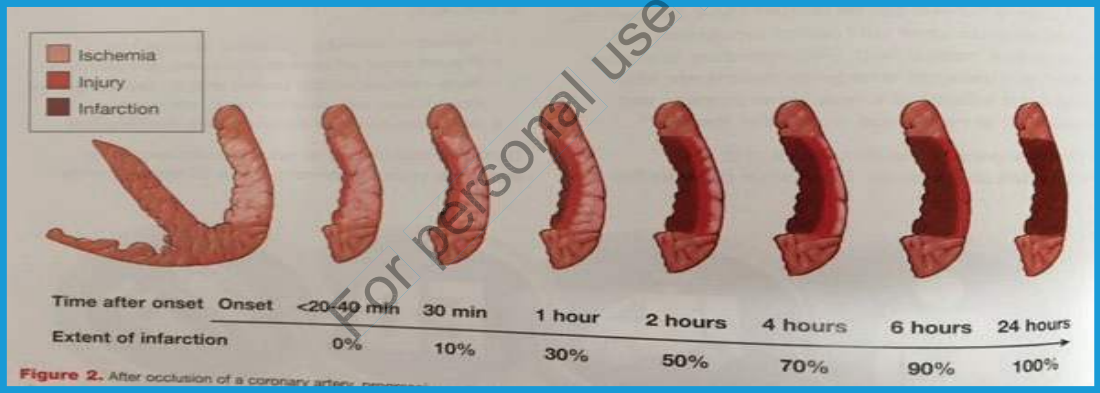
NSTEMI



STEMI

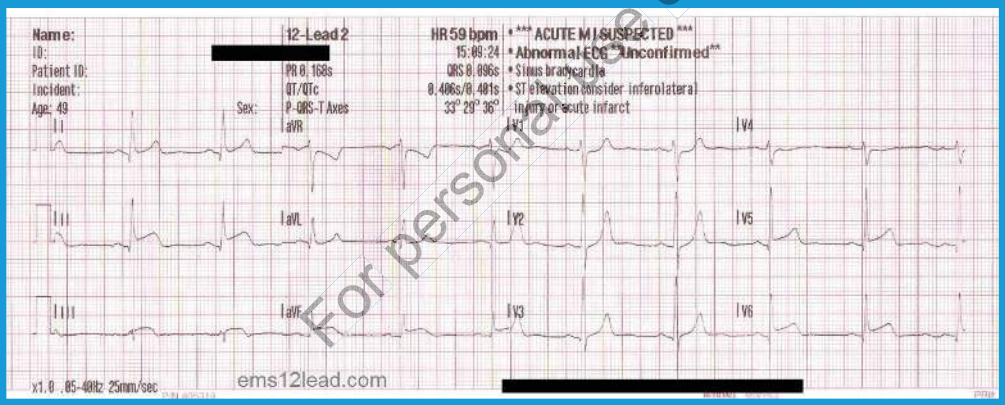
Amsterdam, et al., Circulation, 2014

"TIME IS MUSCLE"

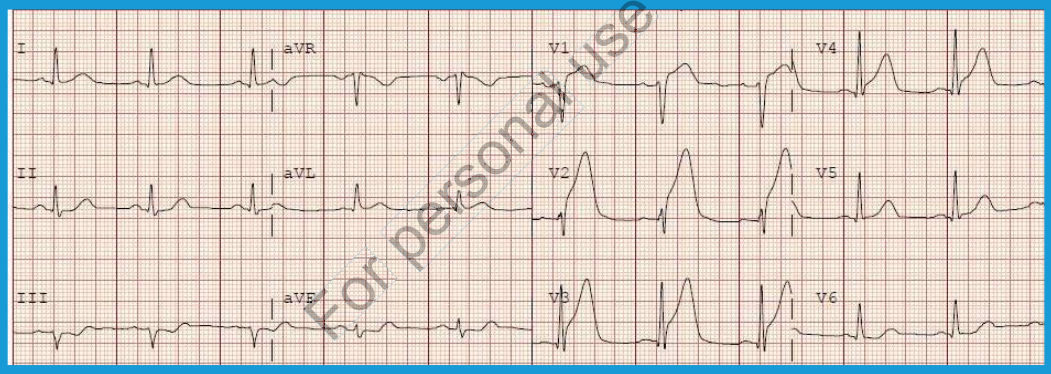


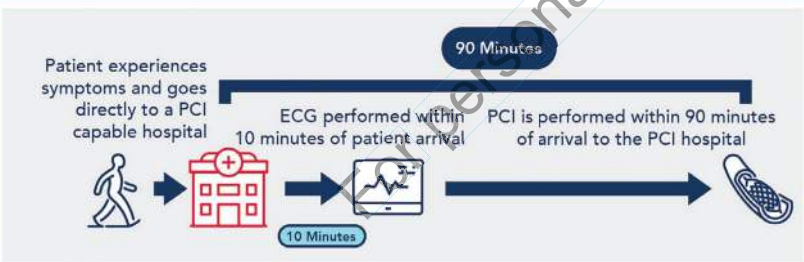
AHA STEMI Provider Manual

INFERIOR STEMI



ANTERIOR STEMI

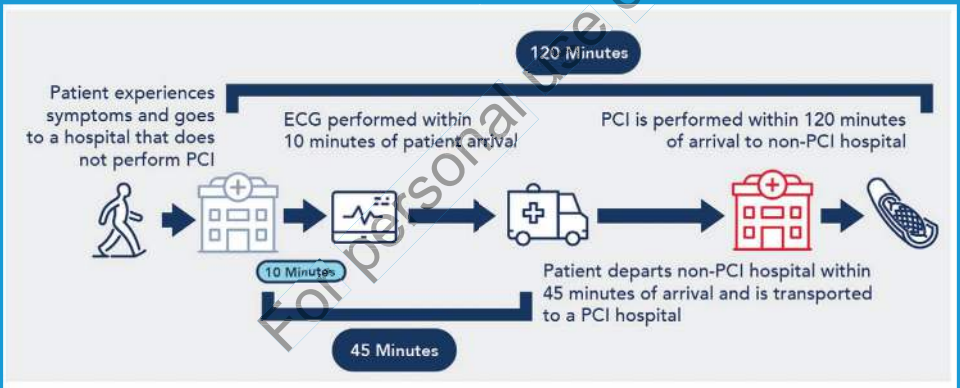




STEMI Time Benchmarks

Tamis-Holland, et al., 2024 JSCAI

REGIONAL TRANSFER



Tamis-Holland, et al., 2024 JSCAI

PROBLEMS WITH FIBRINOLYTIC THERAPY

- Failure to open infarct artery ~40%
- Intracranial hemorrhage 1-2%
- Contraindications up to 40%
- Lytic outcomes consistently inferior to timely PCI

REVASCULARIZATION FOR CORONARY ARTERY DISEASE
CATH LAB

Cath Lab Interventions

- Stenting or balloon angioplasty
- Assisted PCI
  - Robotic assisted PCI
  - IABP
  - Impella
  - Lithotripsy

REVASCULARIZATION FOR CORONARY ARTERY DISEASE OPERATING ROOM

- Coronary Artery Bypass Grafting (CABG)
- Beating Heart CABG
- Impella assisted CABG

CARDIOGENIC SHOCK

CARDIOGENIC SHOCK

- Cardiogenic shock is a low cardiac output state resulting in life-threatening end organ hypoperfusion and hypoxia. van Diepen et al., Circulation, 2017
- Short-term mortality ranges from 30%-40% with 1-year mortality approaching or exceeding 50%

Sinha, et al., JACC, 2025

AMI-CS VS HF-CS

- AMI-CS
  - Includes patients with CS due to AMI in the presence or absence of ST-segment elevation on 12 lead ECG (STEMI or NSTEMI).
- HF-CS
  - CS related to primary myocardial dysfunction ascribed to either ischemic or non-ischemic etiologies of cardiomyopathy.
    - De novo – new onset of HF
    - Acute on chronic – acute decompensation of chronic or progressive HF with dilated cardiomyopathy.

Sinha, et al., JACC, 2025

TABLE 1 Common Etiologies of Cardiogenic Shock

Left ventricular failure
- Acute myocardial infarction
- Hypertrophic obstructive cardiomyopathy
- Myocarditis
- Myocardial contusion
- Peripartum cardiomyopathy
- Post-cardiotomy
- Progressive cardiomyopathy
- Septic cardiomyopathy
- Stress cardiomyopathy (takotsubo)
- Ventricular outflow obstruction

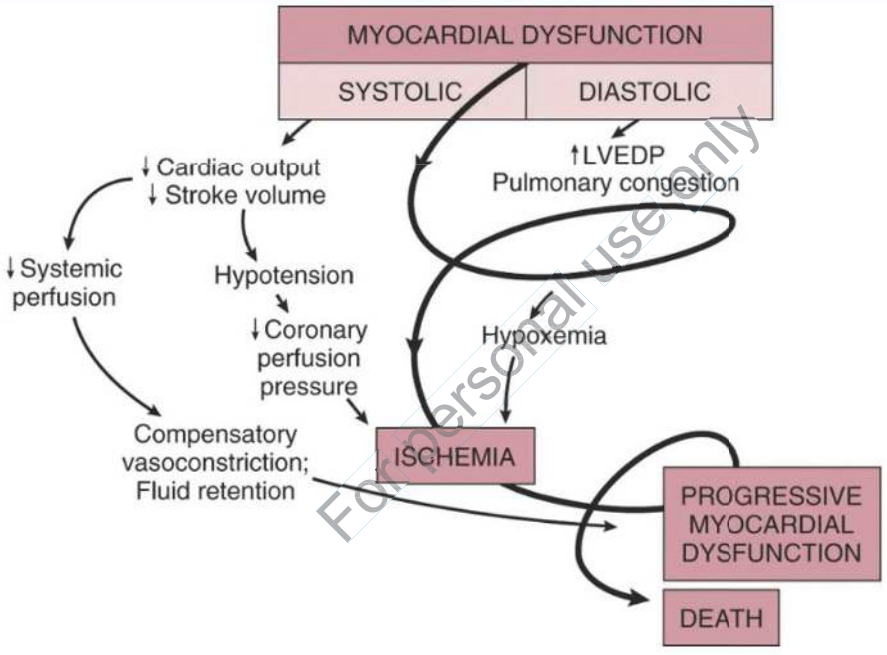
Right ventricular failure
- Acute myocardial infarction
- Myocarditis
- Post-cardiotomy
- Progressive cardiomyopathy
- Pulmonary embolism
- Septic cardiomyopathy
- Worsening pulmonary hypertension

Arrhythmia
- Atrial fibrillation or flutter
- Ventricular tachycardia or fibrillation
- Bradycardia or heart block

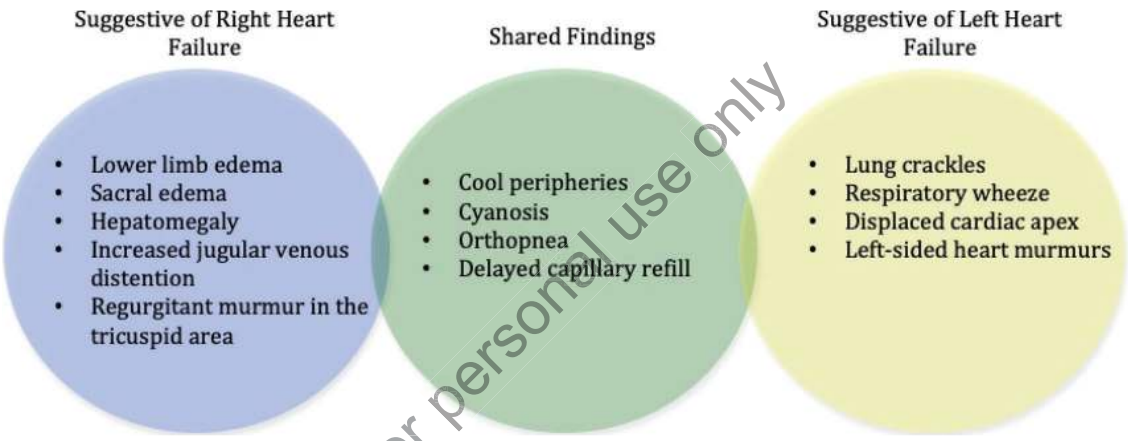
Pericardial disease
- Tamponade
- Progressive pericardial constriction

Chemotherapeutic, toxic, metabolic
- Calcium-channel antagonists
- Adrenergic receptor antagonists
- Thyroid disorders

Valvular or mechanical dysfunction
- Aortic regurgitation—acute bacterial endocarditis
- Mechanical valve dysfunction or thrombosis
- Mitral regurgitation—myocardial ischemia or infarction
- Progressive mitral stenosis
- Progressive aortic stenosis
- Ventricular septal defect or free wall rupture



Hochman et al. Circulation. 2003



PHYSICAL FINDINGS OF VENTRICULAR INVOLVEMENT

Vahdatpour et al. JAHA. 2019; 8:e011991

		Volume Status: Dry	Volume Status: Wet
Peripheral Perfusion	Warm	**Vasodilatory shock** (not CS) Increased cardiac index, low SVRI, low/ normal PCWP	**Mixed CS** Low cardiac index, low / normal SVRI, Elevated PCWP
	Cold	**Euvolemic CS** Low Cardiac index, high SVRI, low/ normal PCWP	**Classic CS** Low cardiac index, High SVRI, Elevated PCWP **C**

van Diepen et al. Circulation. 2017; 136: e232-e268

TRIALS

Lack of appropriately powered randomized trials have prevented consensus in CS-MI management. Baran et al., 2020.

Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock? SHOCK Trial, 1999

- For patients with AMI-CS earlier revascularization led to increased rates of survival

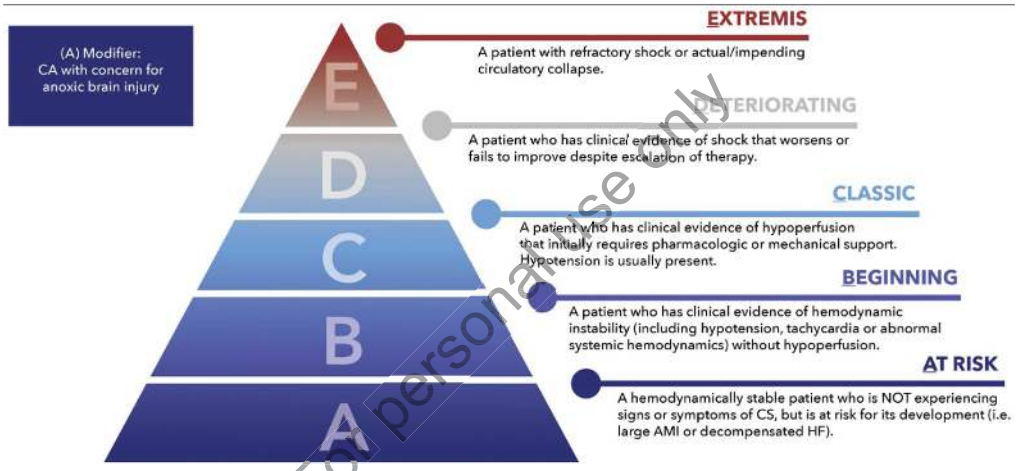
DanGer Shock Trial 2024

- Use of early microaxial flow pump in select patients with STEMI related shock improved 180-day survival vs standard of care.

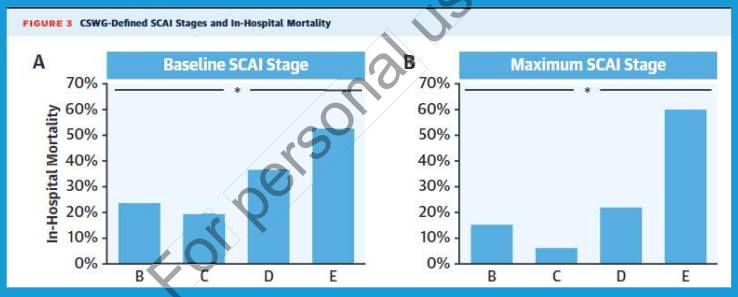
Clinical Trial/Guideline	CS Criteria
SHOCK Trial (1999)[7]	• SBP <90 mm Hg for >30 min or vasopressor support to maintain SBP >90 mm Hg • Evidence of end-organ damage (UO <30 mL/h or cool extremities) • Hemodynamic criteria: CI <2.2 and PCWP >15 mm Hg
IABP-SHOCK II (2012)[8]	• MAP <70 mm Hg or SBP <100 mm Hg despite adequate fluid resuscitation (at least 1 L of crystalloids or 500 mL of colloids) • Evidence of end-organ damage (AMS, mottled skin, UO <0.5 mL/kg for 1 h, or serum lactate >2 mmol/L)
EHS-PCI (2012)[9]	• SBP <90 mm Hg for 30 min or inotropes use to maintain SBP >90 mm Hg • Evidence of end-organ damage and increased filling pressures
ESC-HF Guidelines (2016)[6]	• SBP <90 mm Hg with appropriate fluid resuscitation with clinical and laboratory evidence of end-organ damage • Clinical: cold extremities, oliguria, AMS, narrow pulse pressure Laboratory: metabolic acidosis, elevated serum lactate, elevated serum creatinine
KAMIR-NIH (2018)[5]	• SBP <90 mm Hg for >30 min or supportive intervention to maintain SBP >90 mm Hg • Evidence of end-organ damage (AMS, UO <30 mL/h, or cool extremities)

CARDIOGENIC SHOCK DEFINITIONS

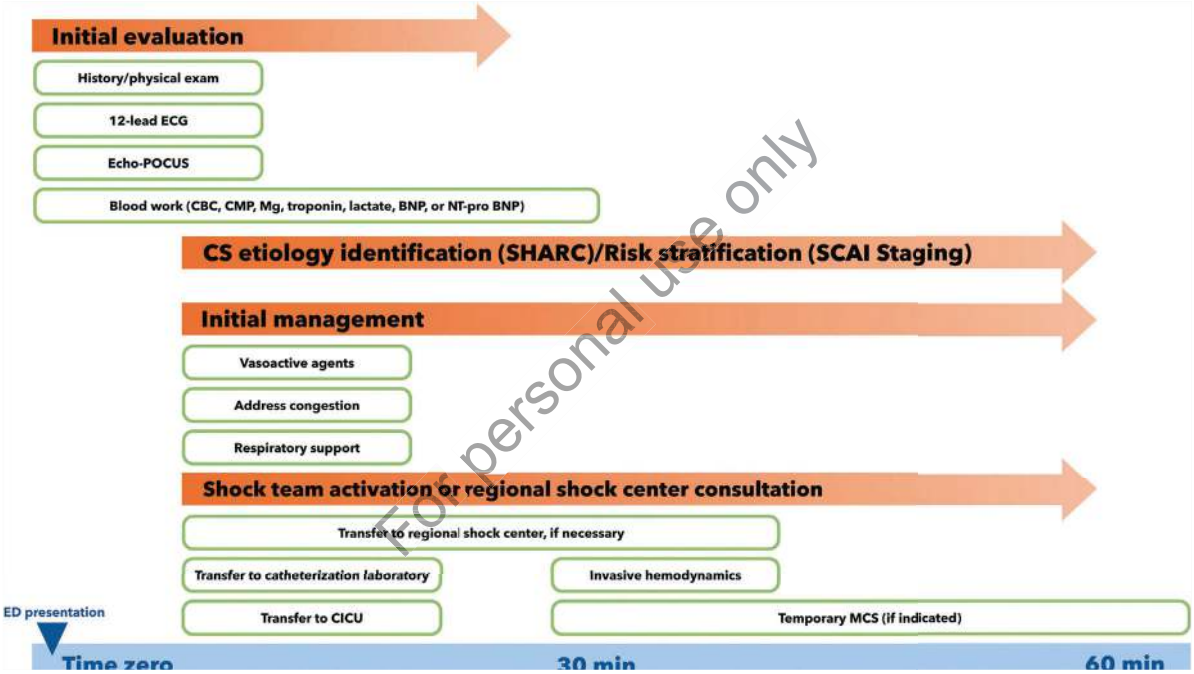
Vahdatpour et al. JAHA. 2019; 8:e011991

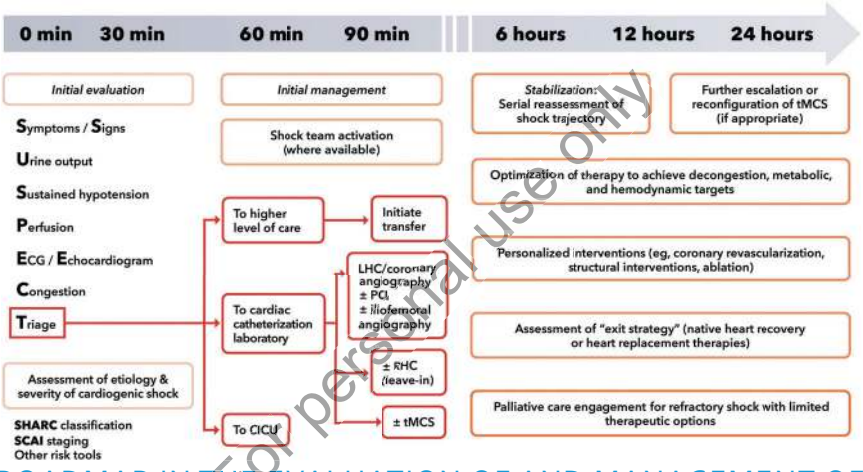


B= BAD



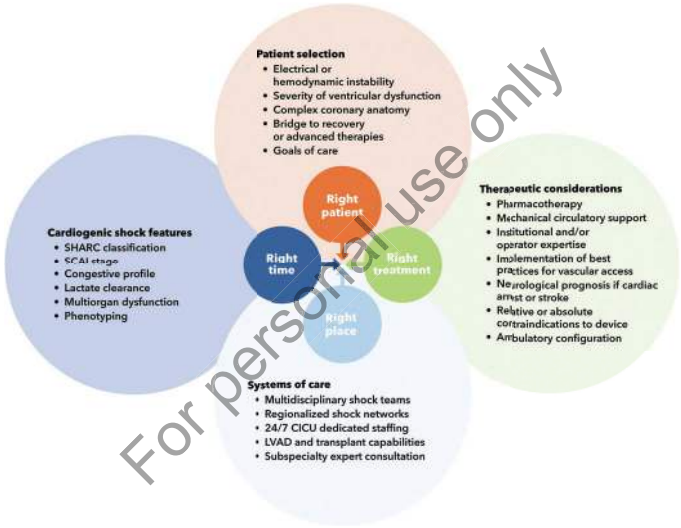
Kapur et al. JACC, 2022





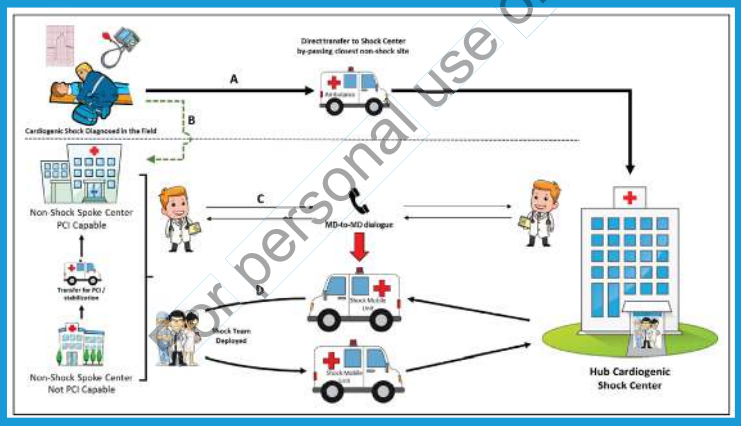
ROADMAP IN THE EVALUATION OF AND MANAGEMENT OF CARDIOGENIC SHOCK

Sinha, et al., JACC, 2025



Sinha, et al., JACC, 2025

REGIONAL SYSTEM OF CARE MODEL



van Diepen et al., Circulation, 2017

MULTIPHASE TEAM-BASED APPROACH

Recognize/Rescue

↓

Optimize

↓

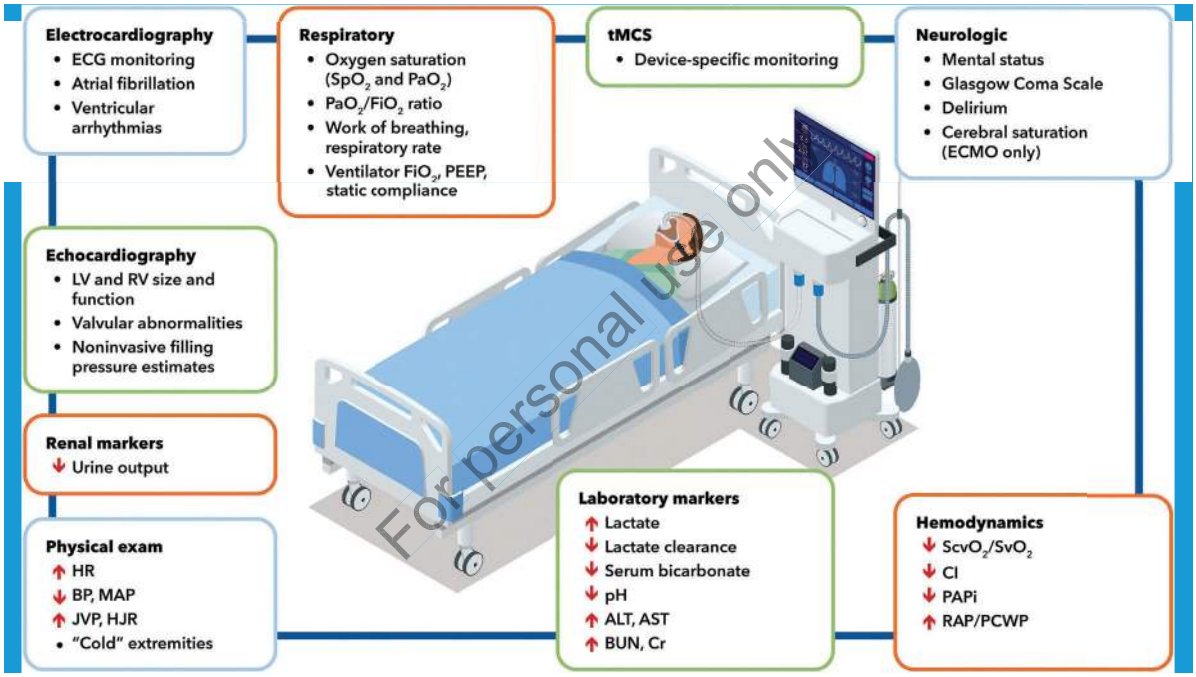
Stabilize

↓

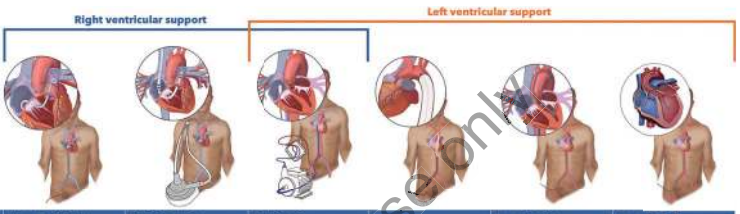
De-Escalation/exit

Sinha, et al., JACC, 2025

CARE AND MONITORING



MCSD



	Impella RP Flex	RA-PA pVAD	VA-ECMO	IABP	Impella CP	Impella 5.5
Max flow	3.0 - 4.0 L/min	4.0 - 5.0 L/min	5.0 - 7.0 L/min	0.5 - 1.0 L/min	3.0 - 4.3 L/min	5.0 - 5.0 L/min
Max pump speed	33,000 rpm	7,500 rpm	6,400 rpm	NA	46,000 rpm	33,000 rpm
Mechanism	Axial flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-AO)	Balloon inflation-deflation (AO)	Axial flow continuous pump (LV-to-AO)	Axialflow continuous pump (LV-to-AO)
Sheath size	23 F venous peel-away	29 or 31 F venous (inflow)	15-24 F arterial 19-25 F venous	7-8 F arterial	14 F arterial peel-away	23 F arterial peel-away
Typical insertion/ placement	Internal jugular vein	Internal jugular vein	Femoral vein (drain) Femoral artery (return)	Femoral artery or Axillary artery	Femoral artery or Axillary artery	Axillary artery
Direct LV unloading	-	-	-	-	+++	+++
Direct RV unloading	+	+	+	-	-	-
Afterload	-	-	↑↑↑	↓↓	↓	↓
Coronary perfusion	-	-	↑↑	↑↑	↑↑	↑↑

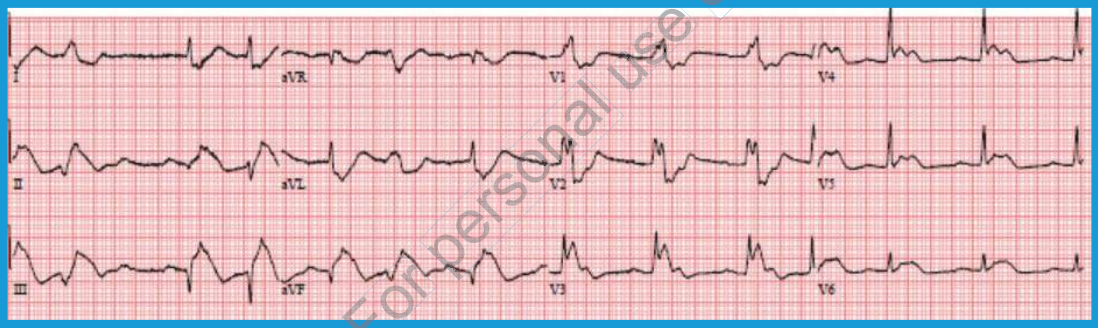
tMCS

CASE STUDY

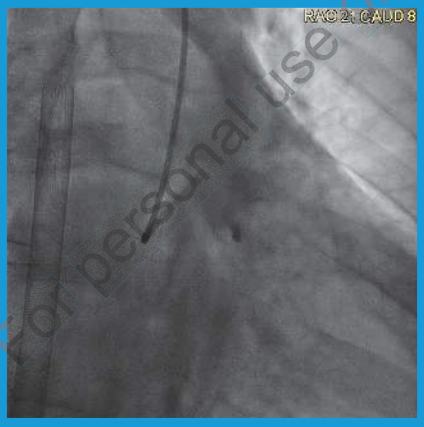
CASE STUDY

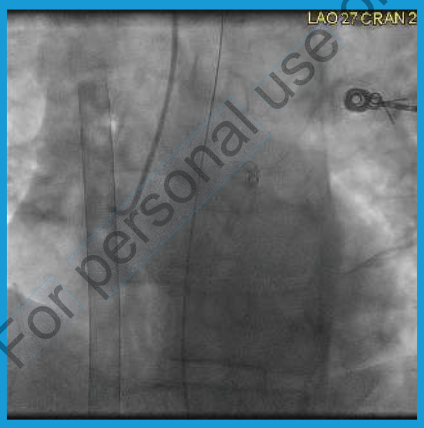
- 51 y.o. male who was found outside by friends after complaining of chest pain and shortness of breath 2 hours prior. 911 was called.
- EMS noted ST elevation on his 12-lead ECG, Med Flight was called, and he was taken to UH. Due to concerns for hypothermia and carbon monoxide exposure, he was first evaluated in the ED.

ED ECG @ 0555



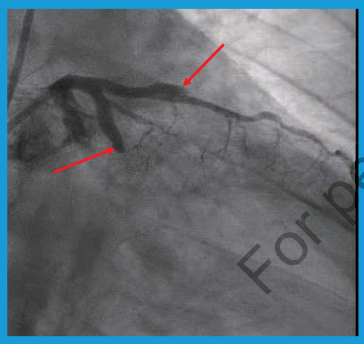
- Pt. decompensated in the ED- bradycardia→asystole. ECPR (extracorporeal cardiopulmonary resuscitation) was initiated in the ED.
- Pan CT Scan
- Cath Lab –
  - 100% proximal LAD, 3 drug-eluting stents
  - 100% proximal circumflex, 3 drug-eluting stents
  - 100% proximal RCA with bridging collaterals (chronic).
  - Impella Percutaneous Left Ventricular Assist Device inserted.



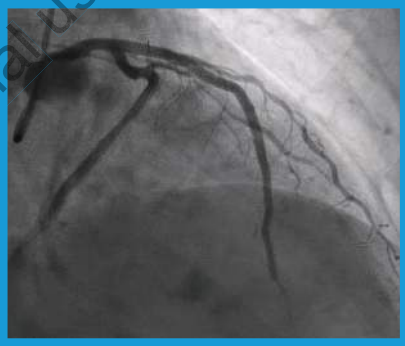


LAD AND CIRCUMFLEX

Pre-Intervention

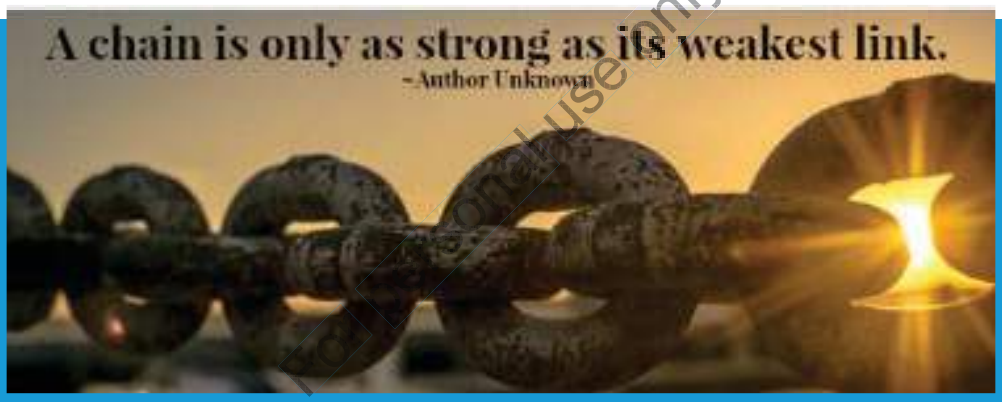


Post-Intervention



HOSPITAL COURSE

- 11/28 LVEF assessed at 10%
- 12/2- Impella 5.5 Insertion
- 12/4- ECMO Decannulation and CVVH started due to poor renal function
- 12/6- Extubated
- 12/20 – Listed for heart and kidney transplant
- 12/26- Sepsis, removed from transplant listing
- 12/31 – Re-activated on transplant list
- 1/3- Orthotopic heart transplant, Impella removal
- 1/4 - Kidney transplant
- 1/13 – Cardiac tamponade with 1.3 liters of blood aspirated and drain placement.
- 1/19 – Drain removed
- 1/20 – Discharged to self-care



KEY TAKEAWAYS

- Early identification and guideline directed treatment of ACS are critical to patient outcomes.
- Systems of care approach utilized for ACS should be implemented in the care of cardiogenic shock patients.
- Cardiogenic shock is a complex disease process and is the most extreme form or cardiac compromise with a 30-day mortality of 30-40%.
- Recognition and treatment of cardiogenic shock at early stages is critical to prevent further deterioration. Stage B = "Bad."
- Systems of care approach should be implemented for cardiogenic shock patients, much like the systems in place for STEMI.

REFERENCES

- American Heart Association, Heart attack symptoms in women, Heart Attack Symptoms in Women | American Heart Association, retrieved 3/26/2025.
- American Heart Association,
- Anesthesia Key, Pulmonary Artery Catheter, retrieved 5/31/2024 from https://aneskey.com/the-pulmonary-artery-catheter/
- Atti, V., Narayanan, M., Patel, B., Balla, S., Siddique, A., Lundgren, S., Velagapudi, Poonam. (2022, 03, 08) A comprehensive review of mechanical circulatory support devices. *Heart International*. 2022;16(1):37-48
- Bakhshi H, Gibson CM. MINOCA: Myocardial infarction no obstructive coronary artery disease. Am Heart J Plus. 2023 Jul 29;33:100312. doi: 10.1016/j.ahjo.2023.100312. PMID: 38510552; PMCID: PMC10945965.
- Baran, D.A., Long, A. &Jentzer, J.C. The Stages of CS: Clinical and Translational Update. Curr Heart Fail Rep 17, 33-340 (2020). http://doi.org/10.1007/s11897-020-00496-6
- Berg, David D.; Bohula, Erin A.; Morrow, David A.. Epidemiology and causes of cardiogenic shock. Current Opinion in Critical Care 27(4):p 401-408, August 2021. | DOI: 10.1097/MCC.0000000000000845

- Berg, David D.; Bohula, Erin A.; Morrow, David A.. Epidemiology and causes of cardiogenic shock. Current Opinion in Critical Care 27(4):p 401-408, August 2021. | DOI: 10.1097/MCC.0000000000000845
- Kapur, N, Kanwar, M, Sinha, S. et al. Criteria for Defining Stages of Cardiogenic Shock Severity. J Am Coll Cardiol. 2022 Jul, 80 (3) 185–198. https://doi.org/10.1016/j.jacc.2022.04.049
- Mickael Lescroart, M., Pequignot, B., Janah, D., Levy, B. The medical treatment of cardiogenic shock. *Journal of Intensive Medicine*, 2023; 3(2): 114-123
- Sinha, S., Morrow, D., Kapur, N. et al. (2025) Concise clinical guidance: An ACC expert consensus statement on the evaluation and management of cardiogenic shock: A report of the American College of Cardiology solution set oversight committee. JACC. null2025, 0 (0) https://doi.org/10.1016/j.jacc.2025.02.018
- Vahdatpour, C., Collins, D., Goldberg, S. (2019). Cardiogenic shock. *Journal of the American Heart Association,* 8(8). https://www.ahajournals.org/doi/full/10.1161/JAHA.119.011991
- van Diepen, S., Katz, J., Albert, N., Henry, T., Jacobs, A., Kapur, N., Kilic, A., Menson, V., Ohman, E., Sweitzer, N., Holger, T., Washam, J., Cohen, M. (2017). Contemporary management of cardiogenic shock: A scientific statement from the American Heart Association. Circulation, 136:e232-e268.
- Waksman, R., Pahuja, M., vanDiepen, S. et al. (2023). Standardized definitions for cardiogenic shock research and mechanical circulatory support devices: scientific expert panel from the Shock Academic Research Consortium (SHARC). Circulation, 148:113-1126.