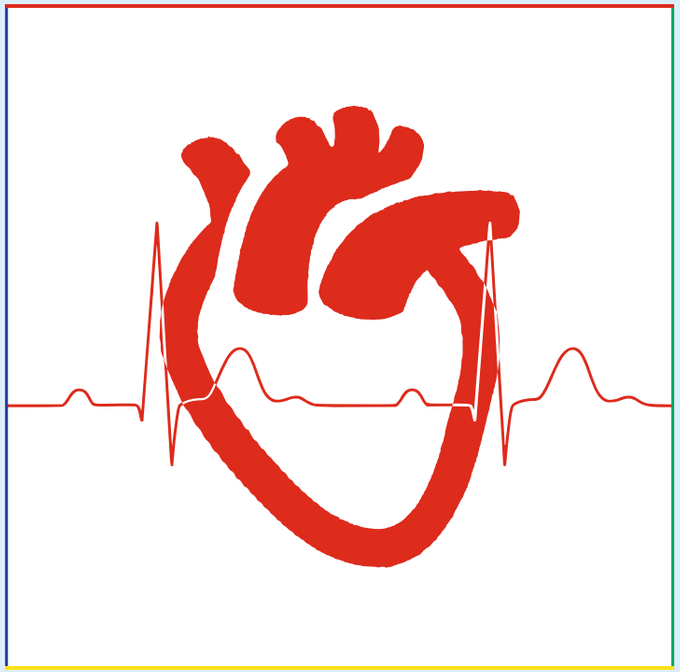
STANDARD TREATMENT GUIDELINES

CARDIOLOGY



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GOVERNMENT OF KERALA

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May 2026

STANDARD TREATMENT GUIDELINES
for
Management of Cardiac Conditions
in Secondary and Tertiary Care Centre

Foreword

At the outset, I appreciate the work done by the respective thematic teams and coordination done by the DME. The Standard Treatment Guidelines (STG) were prepared and published in 2021 in the thick of the Covid pandemic. On the last page of these volumes the road map was mentioned. The few points are mentioned here for the recall.

"The Department of Health has been taking a systematic approach of creating and enabling multiple initiatives with a focus on prevention along with improving health care services. Health care service delivery is one of the most important services and is always seen as a barometer to assess the Governance. While it is important to develop infrastructure, an essential prerequisite is to develop systems and processes to bring in standardization in management of patient care. ……The foundation is laid and we take up the challenge to work on the unfinished agenda."

It was mentioned in the road map to have institutional mechanism to ensure updation of Standard Treatment Guidelines. The next step that was suggested was to do analysis of Karunya Arogya Suraksha Padhati (KASP) and standard treatment guidelines to work on developing a Balance Score Card to give information regarding compliance from the Hospitals and to build a "feedback loop" to improve. These initiatives remained at concept level on the last page! But following detailed discussions with Dr Vishwanathan, Director Medical Education, some of the foundational things were prioritized and given an impetus to take it to finality. In this journey, many committed doctors from various Medical Colleges of respective specialties participated. The previous coordination team members and experts were also consulted and they also participated in discussions and these Standard Treatment Guidelines are prepared.

The standard treatment guidelines will be made available in the Kerala Health portal (health.kerala.gov.in). This will enable the resource book availability not only to people within the state but to all in the country and outside our borders as well. I am confident that it will be used by students and practicing doctors. We request inputs based on the research from the Specialists and Experts. The teams shall continue to update and make any required changes in the STG by doing periodic updates.

The most important thing we all need to internalize is to have a shared vision and

work as a team to reach to a state of 'excellence'. If we take a look at the preparation of the Directorate Medical Education Management Information System, documents of each Medical Colleges, it provides information regarding 'what we are, what we do and what we aspire to do', pandemic preparedness, AMR accreditation and many more such initiatives taken on scale, which are all outcomes of collective TEAM work. This has laid a foundation for involving all the stakeholders including undergraduate and postgraduate students. This should encourage the teams in Medical Colleges to believe in themselves and build future initiatives on such a sound platform.

I express my sincere thanks to Dr Vishwanathan for his patience and bearing with relentless follow ups! I also take this opportunity to thank each and every team and their members and everyone from Directorate Medical Education and Medical Colleges who supported these initiatives.

I would like to express my sincere gratitude to all those who have contributed to publish these Standard Treatment Guidelines.

I wish all the success to DME team to make Kerala MCH as a premier knowledge hub in Medical Science.

Dr Rajan Khobragade IAS

Additional Chief Secretary
Health & Family Welfare and
AYUSH Department
Govt of Kerala.

Message

Patient care today demands evidence-based, standardized, and contextually relevant clinical practice. In this regard, the publication of the **Second Edition of the Standard Treatment Guidelines** marks an important step forward in strengthening the quality, consistency, and accountability of healthcare delivery in Kerala.

The first edition laid a strong foundation for uniform clinical practice across specialties and super specialties. Since then, advances in medical knowledge, evolving treatment modalities, and the growing need for periodic updating have made it essential to revisit and refine these guidelines. The present edition reflects this commitment to continuous improvement and clinical excellence.

I am pleased to note that subject experts from various disciplines of Government Medical Colleges, private institutions and professional bodies have contributed as resource persons in the preparation of these guidelines. Their academic expertise, practical insight, and dedicated involvement have greatly enriched this edition. I deeply appreciate the sincere efforts of all the conveners, contributors, and coordinators whose collective commitment and teamwork made this publication possible.

These guidelines will serve as a valuable reference for clinicians, teachers, trainees, and healthcare institutions, helping to promote evidence-based decision-making and improve patient outcomes. I am confident that this edition will further support standardization of care and contribute to the advancement of medical education and clinical practice in the State.

I congratulate everyone involved in this commendable effort and commend this publication to all healthcare professionals.

Dr. K. V. Viswanathan
Director of Medical Education
Government of Kerala

Committee for Revision of Standard Treatment Guidelines Cardiology 2026

The first version of standard treatment guidelines was published in 2021. The revised version, brought out in 2026, gives guidance in the management of two common situations in Cardiology, acute coronary syndrome and heart failure. We have added a new chapter on arrhythmias, included as part of the section on acute coronary syndrome. The revised document has addition of several pictures and is updated and made more concise. I thank all members of the committee for the valuable contribution. I thank Dr. Karunadas CP for his efforts in preparing the heart failure section.

Convenor of STG in Cardiology

Dr Cibu Mathew, Professor of Cardiology, Medical College, Thiruvananthapuram

Members of the Expert committee who reviewed the revised guidelines.

1. Dr. N Sudhayakumar, Professor of Cardiology and former Director of Medical Education, Kerala
2. Dr. A George Koshy, Professor and former head of Cardiology, Medical College, Thiruvananthapuram
3. Dr. K Sivaprasad, Professor and former head of Cardiology, Medical College, Thiruvananthapuram
4. Dr. Mathew Iype, Professor and head of Cardiology, Medical College, Thiruvananthapuram
5. Dr. Karunadas CP, Professor and head of Cardiology, Medical College, Thrissur
6. Dr. Rajesh G, Professor and head of Cardiology, Medical College, Kozhikode
7. Dr. Abhilash SP, Professor of Cardiology, SCTIMST, Thiruvananthapuram
8. Dr. Paul Thomas, Head of cardiology, General hospital, Ernakulam
9. Dr. Praveen S, Associate Professor of Cardiology, Government Medical College, Thrissur.

External Expert

Dr. Abraham Oomman, President, Heart Failure Association of India and Senior consultant cardiologist, Apollo Hospital, Chennai.

Committee members for the development of first version of standard treatment guidelines (2021)

Convenor

Dr Cibu Mathew, Professor of Cardiology.

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7. Dr. Karunadas CP, Professor and head of Cardiology, Medical College, Thrissur

External Expert

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Section I

Evaluation and management of patients presenting with

Acute coronary syndrome in secondary and tertiary care hospitals

Section II

Evaluation and management of heart failure

Section - I

Evaluation and management of patients presenting with Acute coronary syndrome In secondary and tertiary care hospitals

1. Scope

Population

Adults more than 18 years of age

Key clinical issues covered:

Definition, clinical features, investigations, risk stratification and management of acute coronary syndromes

Clinical issues that will not be covered:

Management of non-cardiac causes of chest pain, methods of coronary intervention, cardiopulmonary resuscitation and management of heart failure. The following are only a guide and always the clinician has to exercise own judgment and clinical experience.

Health care setting:

Secondary and tertiary health care, as defined

Outcome:

Early diagnosis and management of acute coronary syndrome, early reperfusion in STEMI, prevention of cardiac arrest and mortality.

Abbreviations.

ACE-I	angiotensin converting enzyme inhibitor
ACLS	advanced cardiac life support
ACS	acute coronary syndrome
ARB	angiotensin receptor blocker
BB	beta blocker
CAD	coronary artery disease
CAG	coronary angiogram
CKMB	creatine phosphokinase MB fraction
CPR	cardiopulmonary resuscitation
CVA	cerebrovascular accident
CVD	cardiovascular disease
c-Tn	cardiac troponin
ECG	electrocardiogram
ED	emergency department.
GFR	glomerular filtration rate
LBBB	left bundle branch block
LMWH	low molecular weight heparin
MI	myocardial infarction
MR	mitral regurgitation
MRA	mineralocorticoid receptor antagonist
NSTEACS	non ST elevation acute coronary syndrome
NSTEMI	non ST Elevation Myocardial Infarction
NOMI	non occlusion myocardial infarction

OMI	Occlusion myocardial infarction
PCI	percutaneous coronary intervention
PTCA	percutanoeus transluminal angioplasty.
RBBB	right bundle branch block
RBS	random blood sugar.
STEACS	ST elevation acute coronary syndrome
STEMI	ST elevation myocardial infarction
UA	unstable angina
UFH	un-fractionated heparin
VSR	ventricular septal rupture.

2. Diagnosis, investigations and risk stratification of acute coronary syndrome (ACS)

2.1. Introduction

Chest pain due to myocardial ischemia can be life threatening and early diagnosis and appropriate management is needed to reduce mortality and morbidity. Detailed history, physical examination and investigations are useful in diagnosis and proper management. History of cardiac pain, typical electrocardiographic (ECG) changes and rise and fall of biomarkers like cardiac troponin or creatine phosphokinase MB fraction (CKMB) are the most important tools to diagnose acute coronary syndrome (ACS).

2.2. Definitions

2.2.1 Definition of the health care setting. For the purpose of this guideline, we define the level of health care setting for acute coronary syndrome (ACS) management as follows.

- Primary care setup is a hospital where a basic qualification doctor is available; ECG can be done but has no facility to manage ACS as inpatient.
- Secondary care setup is a hospital where ECG, cardiac Troponin and basic blood investigations are available and medical management including thrombolysis can be done but cardiac catheterisation lab (cathlab) based interventional management is not possible.
- Tertiary care setup is a hospital where there is facility to manage ACS both medically and by cathlab based interventional management.

2.2.2 Acute Coronary syndrome refers to clinical situations that are compatible with acute myocardial ischemia. They classically present with ischemic cardiac pain or angina, though atypical symptoms (like abdominal discomfort or dyspnoea) or even 'silent' ischemia (where there may not be a significant symptom) can occur. ECG changes of myocardial ischemia/infarction or biomarker elevation may or may not be present at presentation, but usually become evident over a period of time.

ACS consists of myocardial infarction (MI) and unstable angina (UA). Cardiac ischemia without development of myocardial infarction is called unstable angina. Presentation of UA

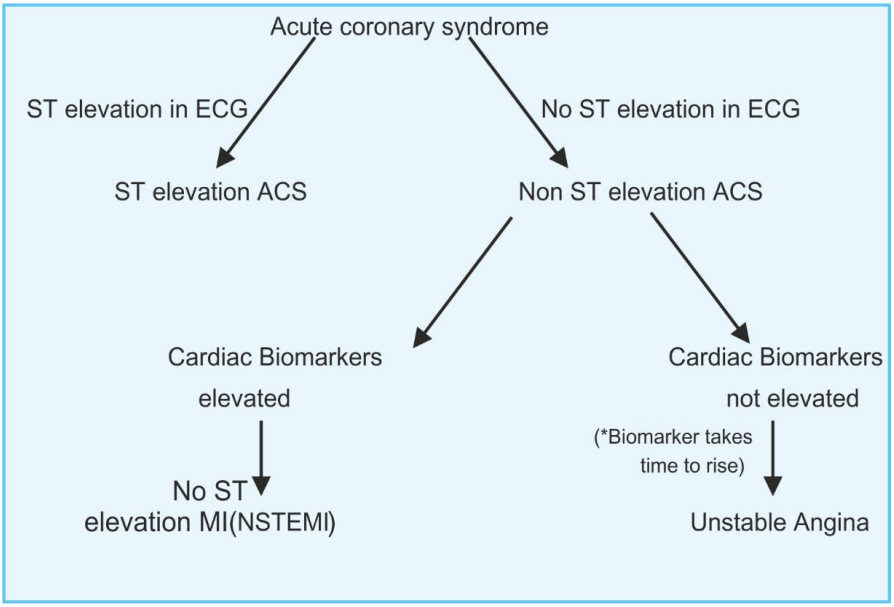
can be one among the following, angina at rest, a new onset effort angina, or rapid worsening of the preexisting effort angina. In UA, cardiac ischemic biomarkers will not be elevated to the range diagnostic of myocardial infarction (MI).

Acute myocardial infarction is defined as a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile (or upper reference limit of normal) and with at least one of the following:

- Symptoms of ischaemia.
- New or presumed new significant ST-segment–T wave changes (ST-T changes)
- Development of pathological Q waves in the ECG.
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
- identification of an intracoronary thrombus by angiography

In studies of disease prevalence by the World Health Organization, myocardial infarction was defined by a combination of two of three characteristics: typical symptoms, biomarker elevation and a typical ECG pattern. This definition seems to be suitable in context to applicability in a primary care center compared to the Universal definition of myocardial infarction.

Symptoms due to cardiac ischemia	Chronic Coronary syndrome	Transient, reversible, myocardial ischaemia, usually provoked by exertion, emotion or other stress, and may manifest as angina, other chest discomfort, or dyspnoea, or may be asymptomatic. Typically, the event lasts less than 20 minutes, is precipitated on effort, relieved by rest, with no hemodynamic changes, and no recent worsening in severity.
	Acute coronary Syndrome	Cardiac pain of ischemic nature at rest/ or new onset effort angina/ or effort angina with recent worsening/ or prolonged cardiac pain/ or cardiac ischemic symptoms with hemodynamic disturbance (like pulmonary congestion, hypotension) or with persistent ECG changes or cardiac biomarker positivity



ST elevations ACS vs non-ST elevation ACS

- **ST elevation ACS** have essentially ST segment elevation in the ECG, of ≥0.1 mV (or ≥ 1 mm in normal standardization) in two contiguous leads (except V_2 V_3, where the elevation should be ≥ 2mm in males > 40 years, ≥ 2.5mm in males < 40 years and ≥1.5 mm in females). ST segment elevation is measured at the J point. J point is the junction of QRS and ST segment.
- **Non-ST elevation ACS** is diagnosed in a patient having ACS but the ECG diagnostic criteria for ST elevation are not satisfied**.** It comprises of **unstable angina (UA) and Non-ST Elevation myocardial infarction (NSTEMI).** In the very early phase of ACS, biomarker elevation need not be present and hence it is impossible to distinguish between NSTEMI and UA. Hence, they are grouped as NSTEMI/UA initially and further classified into NSTEMI or UA once the disease evolves.

3. Assessment in suspected cases of ACS

3.1 History

Among unselected patients presenting with acute chest pain to the emergency department of tertiary care center, 5–10% have STEMI, 15–20% NSTEMI, 10% unstable angina, 15% other cardiac conditions and 50% have non-cardiac diseases. The background risk of the patient to develop atherosclerotic event and the description of the pain are very important in the early evaluation of acute chest pain.

Typical ischemic chest pain is characterized by a retrosternal sensation of pressure or heaviness ('angina') radiating classically to the left arm (less frequently to both arms or to the right arm), neck or jaw, which may be intermittent (usually lasting several minutes) or lasting for more than 30 minutes. Though right arm radiation is less frequent, radiation to right arm and lower jaw is more specific for cardiac pain (Fig 1). Cardiac pain lasting for more than 30 minutes is more likely to result in myocardial infarction than short duration pain and hence short episode of pain is provisionally considered as angina and longer duration as myocardial infarction, which may be reclassified based on further availability of investigations like ECG and cardiac markers. Additional symptoms such as sweating, nausea, abdominal pain, dyspnoea and syncope may be present. Exacerbation of symptoms by physical exertion and relief with rest favors diagnosis of cardiac ischemic pain. Relief of symptoms after nitrate administration is suggestive but not specific for cardiac pain.

Other presentations. Acute pulmonary oedema or cardiac arrest can be presenting symptom of ACS. In patients with longstanding diabetes mellitus, silent myocardial infarction (infarction without significant symptoms) can occur.

Atypical presentations of ACS include

- epigastric pain
- indigestion-like symptoms
- isolated dyspnoea
- fatigue
- syncope.

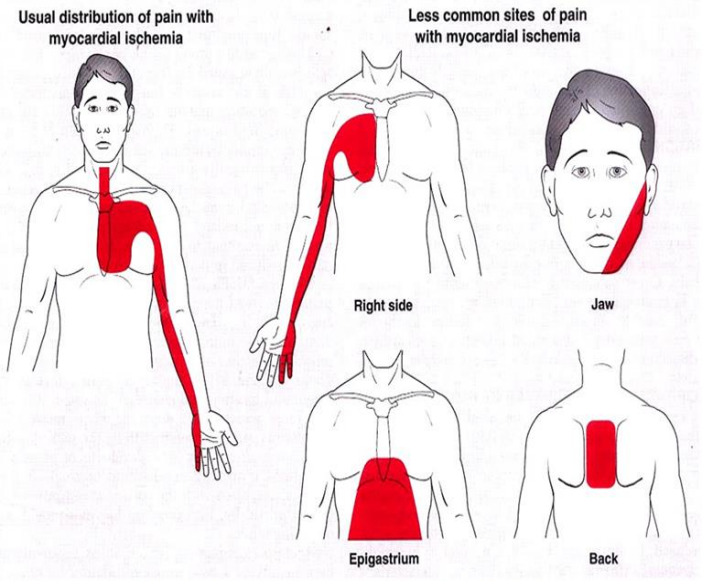


Figure 1. Sites of distribution of cardiac ischemic pain

Atypical type of ischemic symptoms is more often observed in the elderly, in diabetic patients, in women and in patients with chronic renal disease and dementia. Ischemia can present with syncope, dyspnoea or abdominal pain without chest pain. Hence in high-risk patients presenting with atypical symptoms, due care should be given to exclude ACS.

The following pain descriptions are not suggestive of myocardial ischemia.

- Pleuritic type pain, sharp or knife-like pain occurring on respiratory movements or cough
- Primary or sole location of discomfort in the middle or lower abdominal region
- Pain that can be localized with tip of one finger
- Pain reproduced with movement or palpation of the chest wall or arms
- Constant pain that persists for many days
- Very brief episodes of pain that last a few seconds or less
- Pain that radiates into the lower extremities

Causes of chest pain other than due to cardiac ischemia	
Gastro-oesophageal reflux disease	Associated with heartburn, regurgitation, and dysphagia and bad taste in the mouth. Relation with food and sometimes relief with upright posture is seen
Pericarditis	May be associated with fever and tachycardia. Pain can increase on movement. Pericardial rub may be heard. Global ST elevation (almost all leads showing ST elevation except in aVR and V1) in ECG with PR segment depression is suggestive of pericarditis. Echo cardiogram shows pericardial effusion.
Pleurisy	Pain increases on inspiration, may have associated cough, fever and evidence of respiratory tract infection. A pleural rub may be heard
Pulmonary embolism	Small pulmonary embolism can produce pleuritic chest pain. Massive embolism presents with hypotension or syncope and may be confused with ACS. Pulmonary embolism should be suspected in patients at high risk of deep venous thrombosis like those having prolonged limb immobilization.
Dissection of aorta	Acute chest or back pain and a difference in the pulse volume in the upper extremities suggest possibility of acute thoracic aortic dissection. In dissection of aorta the pain may be tearing in type and can have maximum intensity to start with, in contrast to myocardial ischemia where pain builds up.
Musculoskeletal	Musculoskeletal causes are very common and can be due to trauma, isolated musculoskeletal pain or costochondritis. Cervical spondylosis can produce chest pain and shoulder pain. Stinging character of pain, pain that is reproducible on palpation and

	movement/ respiration and localized muscle tension suggest musculoskeletal pain.
Acute cholecystitis/ pancreatitis	Abdominal examination and associated symptoms may give a clue. Cholecystitis can produce pain in right hypochondrium with radiation to right shoulder and minor ECG change like T inversion. In pancreatitis pain radiates to back and may be lesser on sitting and bending forward.

It is important to identify clinical circumstances that may precipitate or exacerbate ACS, such as anaemia, infection, fever and metabolic or thyroid disorders. Every patient suspected of having acute coronary syndrome should be asked for history of bleeding, for example melena. Identify conditions which puts patient at higher risk for bleeding like bleeding diathesis, use of oral anticoagulants or recent surgery or trauma. This is because ACS management essentially involves aggressive antithrombotic therapy.

3.2 Physical examination.

The first step in clinical evaluation is to identify any life-threatening situation with a view to preventing death. Rapidly evaluate for signs of high risk.

- **shock** - hypotension, feeble pulse, cold and clammy extremities, altered sensorium, reduced urine output
- **acute heart failure** – dyspnea or orthopnea, tachycardia and tachypnoea with bi-basal lung crepitation, 3rd heart sound and desaturation
- **arrhythmia** - very high or low heart rate especially with rhythm irregularity.
- Mechanical complications like **ventricular Septal Rupture / mitral regurgitation**

Shock, acute heart failure and arrhythmia need urgent intervention. In case of no features needing urgent intervention continue to do a quick but focused physical examination.

- A fast but focused physical examination is to be done when ACS is suspected. In stable patients a complete physical examination is to be done.
- Is the patient in distress, sweating, dyspnoeic, pale /or cold? Hypotension, acute pulmonary oedema, bradycardia or tachycardia may be the reason and are indications for immediate emergency care.
- Look for anemia which may the primary reason for cardiac ischemia.
- Pulse- rate, volume and pulse asymmetry are very important. Tachycardia, significant bradycardia and irregular rhythm are suggestive of arrhythmia.
- Both high blood pressure and hypotension are important. If there is pulse asymmetry and aortic dissection is suspected, blood pressure should be evaluated in lower limbs also.
- Respiratory rate and oxygen saturation by pulse oximetry may be measured in all patients whenever possible.

In the focused physical examination one has to look for anemia, evaluate the pulse (in all limbs) including rate, rhythm, volume and pulse symmetry, check blood pressure (BP) both systolic and diastolic (in all limbs when there is pulse asymmetry which will give a clue to dissection of aorta), respiratory rate, saturation by pulse oximetry, JVP and heart sounds including 3rd and 4th heart sound. Auscultate for systolic murmur (which can be due to mitral regurgitation/ ventricular septal rupture) and pericardial rub. Look for evidence of atherosclerotic cardiovascular disease like stroke, peripheral vascular disease and carotid bruit. A respiratory system evaluation for pneumothorax or pleural effusion and an abdominal examination for possible diseases like cholecystitis/pancreatitis is to be done in quick time. Vital signs can be used to determine subsequent evaluation and cause of chest pain. For example, hypoxemia increases suspicion for pulmonary or cardiac etiologies of chest pain, while fever raises suspicion for infection.

3.3 Investigations

3.3.1 Electrocardiogram

A 12 lead ECG is mandatory, if the history is suggestive of cardiac ischemia. It is preferable to obtain V_3R and V_4R recordings also and V7 V8 and V9 in select cases. **The resting 12-lead ECG may be obtained as early as possible and preferably within 10 minutes of the patient's arrival.** Initial ECG can be normal even in acute MI. A normal early ECG does not exclude ACS, if the history is suggestive of ACS. Repeat ECG may show ischemic changes. When clinical presentation is suggestive of ACS and initial ECG is normal, ECG may be repeated after ½ an hour and at 4-hour, if the patient is not having continuing symptom. ECG may be repeated more frequently at ½ hour interval, if the patient has symptoms of continuing cardiac ischemia.

- Look for ischemic changes like ST elevation, ST depression, T inversions, or pathological Q waves (width of Q wave equal to 1mm or more than 40 msec). New ECG changes like significant ST shift and T inversion and evolving ECG changes are highly suggestive of ACS. (Fig 2 & 3)
- Very tall T may occur before ST elevates and may be the first change in MI. T inversion of cardiac ischemia is typically symmetric and arrow shaped.

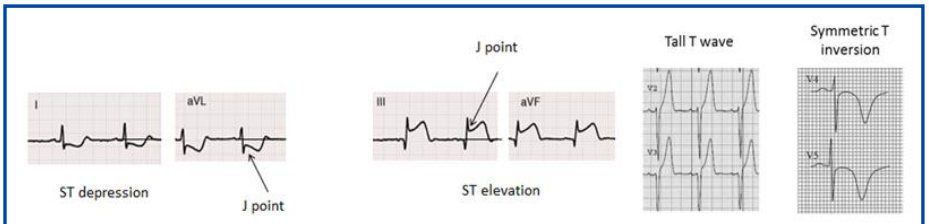


Figure 2. ECG changes in acute coronary syndrome

- Combination of ST elevation localized to an arterial territory (indicative changes) and ST depression in oppositely placed leads (reciprocal changes) is highly suggestive of MI. ST elevation can be used to localise the culprit artery.

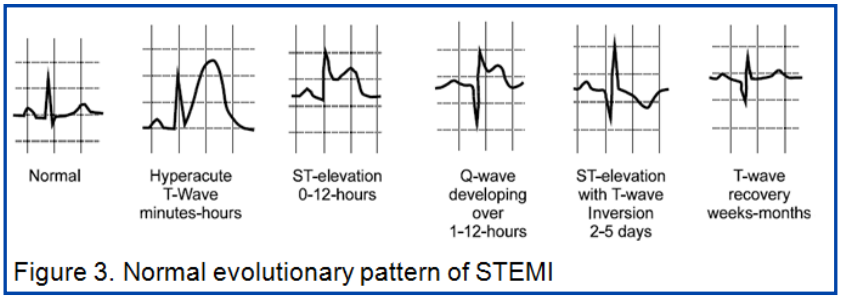


Figure 3. Normal evolutionary pattern of STEMI

STEMI - naming based on the leads showing ST elevation

Anterior wall	ST elevation in Chest leads
Inferior wall	ST elevation in 2, 3, aVF
Lateral wall	ST elevation in I, aVL, V5, V6
Posterior wall	ST elevation in V7, V8, V9; or may present as ST depression in V1-V4, with prominent R with and upright T in V1/V2
RV MI	ST elevation in right sided chest leads, V3R, V4R

- Right ventricular myocardial infarction (RVMI) is diagnosed when there is ST elevation in V_3R and V_4R in patients with inferior wall MI (Fig 4). When RV MI is present in a case of inferior wall MI, the lesion can be localized to proximal RCA. When there is associated RV MI, the outcome can be worse than a case of isolated inferior wall MI.

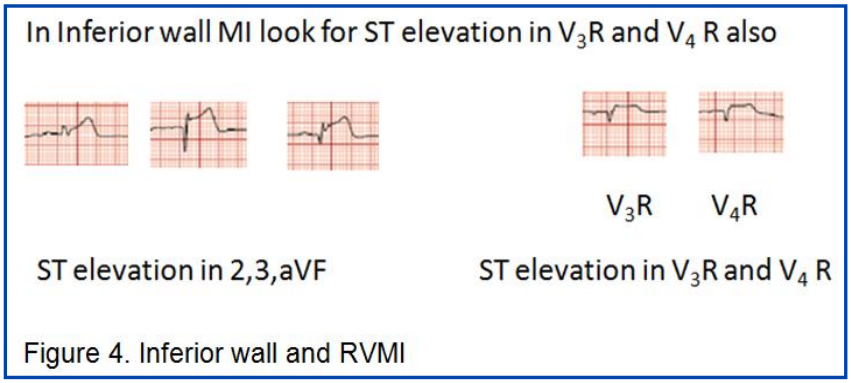
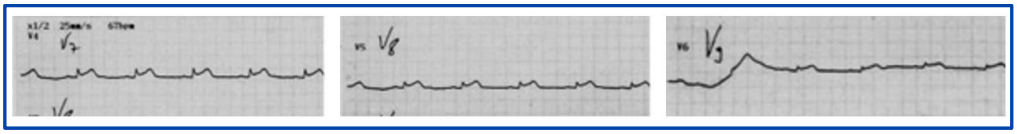


Figure 4. Inferior wall and RVMI

- Special leads V_7 V_8 V_9 may be recorded to look for ST elevation in posterior wall MI



- Asymmetric low amplitude (<3 mm) T inversions may occur at times in precordial and inferior leads in normal young people, especially in obese females and do not suggest ischemia. T inversion can occur in many non-cardiac conditions also.
- Rate and rhythm- look for presence of tachy or brady arrhythmias. Identify bundle branch block which will have wide QRS (≥120 millisec or ≥3 mm in normal paper speed). Atrioventricular block including PR interval prolongation are to be identified.
- Normal ECG does not exclude ACS in a patient with high likelihood presentation and serial ECG evaluation is very important to look at evolving changes.
- Apart from making diagnosis, ECG is useful for risk stratification. It also gives clues to the diagnosis of other conditions like pulmonary embolism (Right axis deviation; T inversion in V1 to V3; S1, Q3, T3; RBBB), or pericarditis (diffuse concave upward ST elevation in most leads, no reciprocal ST depression, PR segment depression).
- Q (q) wave is the first deflection of QRS which is negative. Leads I, aVL, V5 and V6 normally show a physiological q wave which is narrow. Q wave seen in evolved myocardial infarction (pathological Q wave) is wider (≥ 40 m.sec - 1mm in standard ECG) and deeper (more than 2 mm. or > 25 % or R wave height) as shown in (Fig 5)

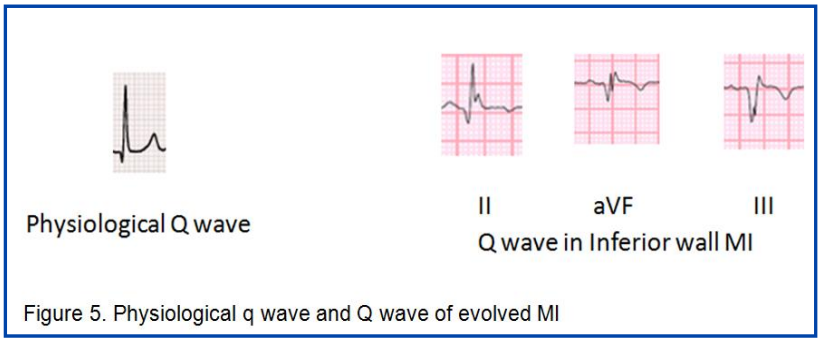


Figure 5. Physiological q wave and Q wave of evolved MI

- Examples of some arrhythmias are given below. Atrial fibrillation (Fig. 6) will have absence of P wave, presence of fibrillary waves and irregularly irregular RR interval.

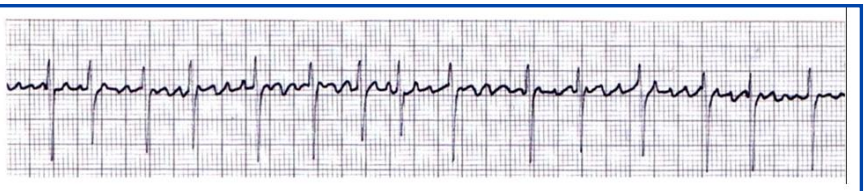


Figure 6. Atrial fibrillation. Irregular rhythm, absence of P wave and presence of fibrillary wave.

- In complete heart block (Fig. 7) there will be dissociation of P and QRS waves and regularly occurring P at a faster rate than regularly occurring QRS. Compete heart block is more common in inferior wall MI.

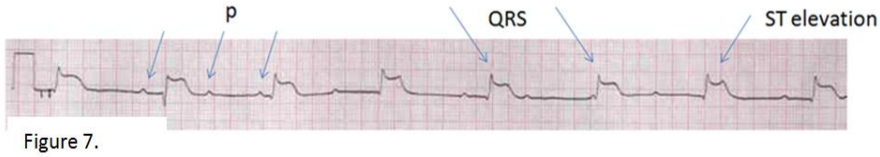


Figure 7.

Complete heart block and Inferior wall MI (ST segment elevation in lead II). Note the Atrio ventricular dissociation (QRS is totally independent of P). P rate is more than QRS rate

- Ventricular tachycardia is regular tachycardia with wide QRS (≥120 msec or 3 mm in regular ECG) and presence of atrioventricular dissociation (P and QRS occurring independently). (Fig 8.)

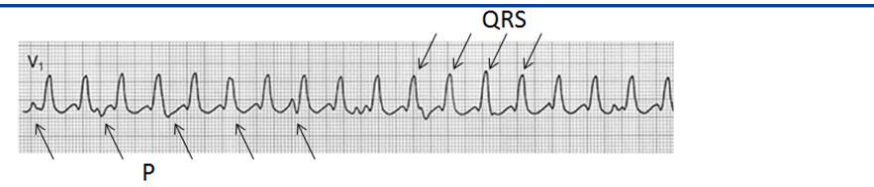


Figure 8. Ventricular tachycardia. Wide QRS tachycardia with regular P, regular QRS, P-QRS dissociation and faster QRS rate than P rate

- Presence of RBBB in anterior wall STEMI (Fig 8) is highly suggestive of block in the proximal left coronary vessel and hence indicates poor outcome. ST elevation in aVR and V1 are also suggestive of proximal left coronary occlusion.

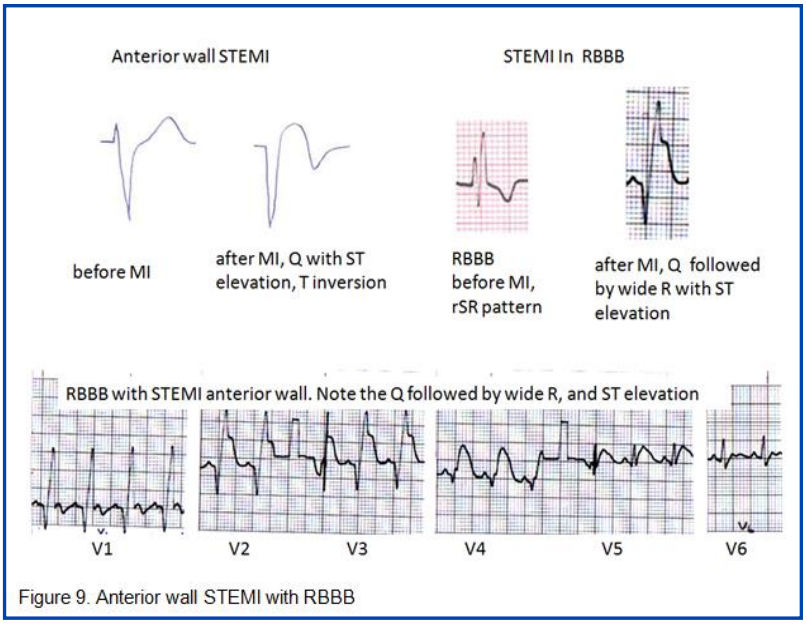


Figure 9. Anterior wall STEMI with RBBB

Concept of occlusion myocardial infarction (OMI)

Recently there is increasing discussion on the concept of occlusion myocardial infarction. As per the present treatment paradigm, STEMI is considered to be due to complete occlusion of coronary artery resulting in transmural (full thickness of myocardium) infarction and NSTEMI due to partial obstruction to coronary flow resulting in non-transmural infarction. However, NSTEMI also can have total occlusion of the coronary artery. 56% of coronary occlusion do not present with STEMI, 25-33% of NSTEMI have acute coronary occlusion and may benefit from early revascularization. Apart from the ST elevation in the ECG, certain ECG patterns suggest occlusion MI (OMI) and are at a higher risk for events. Hence it is proposed to look beyond ST elevation alone and identify occlusion myocardial infarction (OMI) patterns in ECG and offer early reperfusion. American college of cardiology (ACC) use the term STEMI equivalents for some of these ECG patterns (2022 ECDP on Evaluation and Disposition of Acute Chest Pain in ED). As per the latest guidelines by ACC/AHA for ACS (2025), the acute management strategy is still based on STEMI/NSTEMI classification. Recent Australian ACS guidelines have used the term ACOMI (acute coronary occlusion myocardial infarction).

High risk ECG patterns in acute coronary syndrome

There are some high-risk patterns of ECG in acute coronary syndrome which may suggest occlusion of coronary arteries. In such situations aggressive management and early coronary angiogram and invasive management may be considered. 2022 ACC Expert Consensus Decision Pathway on the Evaluation and Disposition of Acute Chest Pain in the Emergency Department mentions the following patterns as STEMI equivalents

- Posterior MI, presenting as ST depression in V1-V4
- LBBB/ventricular paced rhythm with Smith-modified Sgarbossa Criteria
- De Winter Sign
- Hyperacute T waves

ECG findings consistent with acute/subacute myocardial ischemia and associated with high risk are the following

- aVR ST-segment elevation
- ST-segment depression multiple leads
- Wellen's pattern (seen after reperfusion and not at the time of acute occlusion)

Other Pattern in ECG associated with occlusion of coronary arteries in ACS

- Aslanger Pattern

Examples of some ECG signs/patterns discussed below are important to recognize high risk patients with ACS/ are likely to have acute coronary occlusion

1. de Winter sign

This is an ECG pattern which signifies occlusion of the proximal left anterior descending coronary artery (LAD). Instead of the classical ST-segment elevation, there will be a 1- to 3-mm upsloping ST-segment depression at the J point in leads V1 to V6 that then continues into tall, positive symmetrical T waves. The pattern is described as remaining static for longer periods in contrast to the hyperacute tall T waves of STEMI, which rapidly evolve to ST elevation

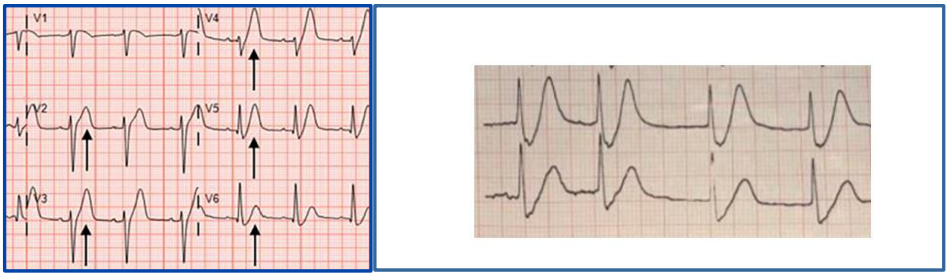


Figure 10. 1- to 3-mm upsloping ST-segment depression at the J point in leads V1 to V6 that continues into tall, positive symmetrical T waves (arrow). Left anterior descending artery obstruction can result in this pattern

2. Wellens pattern

There are two types of Wellens pattern of ACS in the ECG (type A and type B). Wellens pattern in chest leads signifies LAD occlusion with specificity of 99% in type A and 97% in type B. The ECG may appear normal during an episode of chest discomfort and classic ECG abnormalities are subsequently observed when the patient is pain-free.

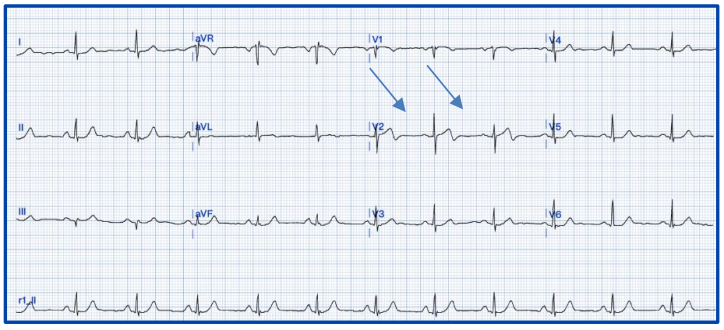


Figure 11. Type A Wellens pattern is less common compared to type B and shows biphasic T waves in leads V2 and V3.

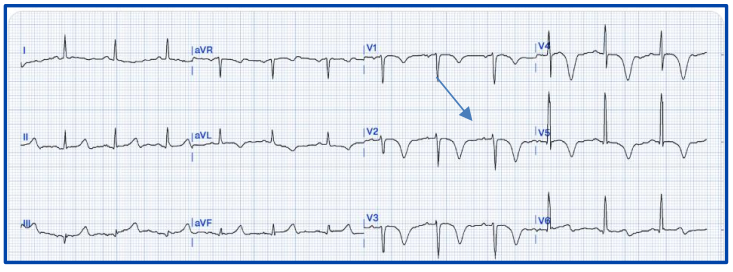


Figure 12. Type B (more common) Wellens pattern shows deep T-wave inversions in V2 and V3.

3. Hyperacute T waves are seen in the earliest phase of STEMI.

Even if the ST elevation is not enough satisfying the criteria for STEMI, the tall broad-based T waves in the anterior leads with reciprocal changes in inferior leads in a patient with cardiac pain is to be considered a STEMI equivalent. Hyperacute T waves may present in inferior leads also.

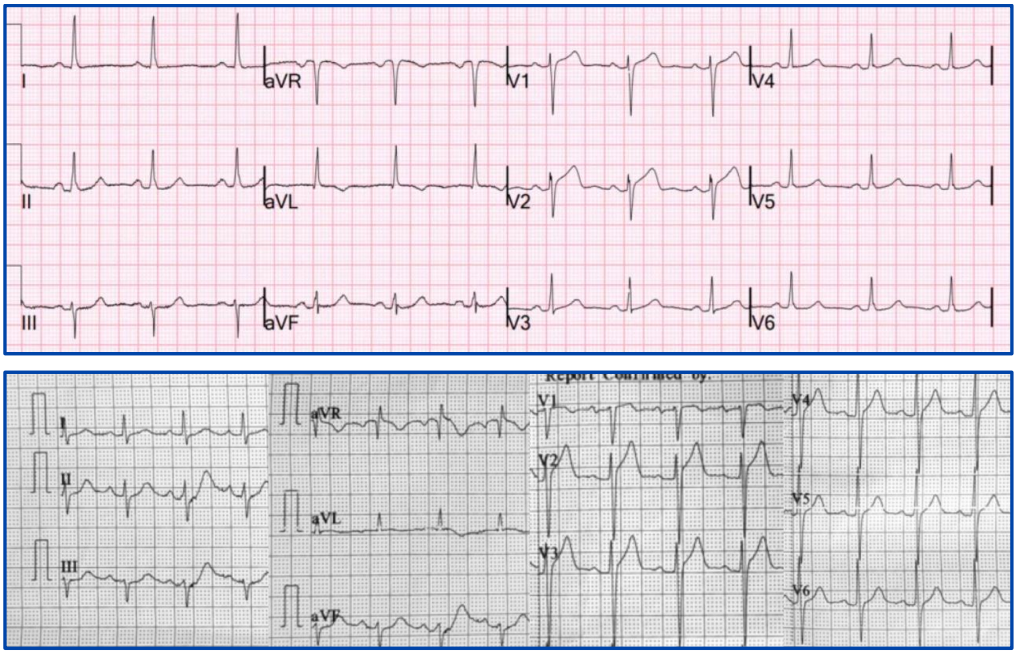


Figure 13. Both the ECGs show hyperacute T waves in anterior chest leads seen V2, V3,V4, with minimal ST depression in leads, 2,3,aVF.

4. Aslanger pattern

This pattern does not qualify for classical definition of STEMI as 2 adjacent leads do not show ST elevation. The components of this pattern are, (1) ST Elevation in III but not in any other inferior lead, (2) ST depression in any of leads V4 to 6 with a positive (at least terminally positive) T-wave and (3) ST in lead V1 higher than ST in V2. This pattern usually suggests multivessel disease with LCX or RCA occlusion.

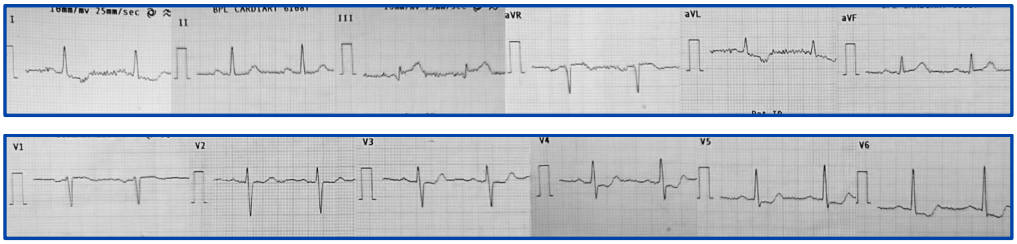


Figure 14. ST segment elevation in lead 3 and aVR and ST segment depression in V4- V6, ST V1 elevated compared to V2.

5. Posterior OMI pattern.

This pattern has ST depression in V1- V4, in the absence of any conduction abnormalities like RBBB, which can produce ST depression in those leads. This is considered a STEMI equivalent. Special leads V7, V8 and V9 are useful to show ST elevation in posterior leads.

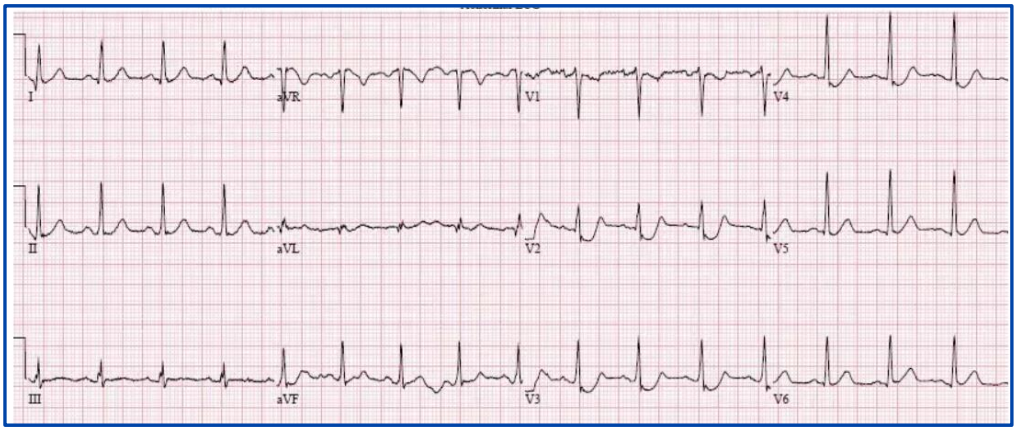


Figure 15. Posterior infarction pattern. ST depression in V1-4, with upright T in V2-V4

6. Minimal ST elevation with reciprocal ST shift in opposite leads.

Sometimes the ST segment elevation can be very subtle. In lateral wall MI, there can be subtle ST elevation in leads I and aVL or in case of inferior wall MI, subtle ST elevation in leads, 2,3, aVF (the magnitude of ST elevation being less than 1mm). The clue for STEMI is the reciprocal change, the ST segment depression in oppositely placed leads; in leads 2,3, aVF in lateral wall MI and in leads I and aVL in inferior wall

MI. The ECG will evolve over a period of time. High index of suspicion is needed for early diagnosis.

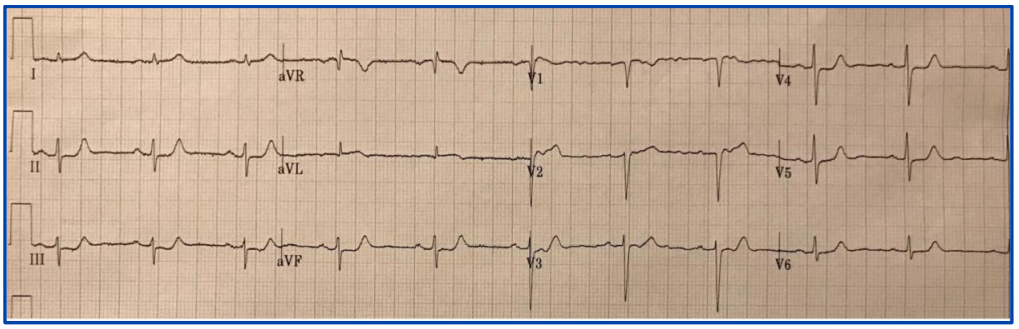


Figure 16. Lateral wall MI subtle ST elevation in lead aVL and ST depression in lead II, III, aVF

7. When there is LBBB, diagnosis of myocardial infarction is difficult.

This is because there will be secondary ST shift because of LBBB itself. In LBBB or in cases of paced rhythm when ischemia/MI is suspected, presence of Sgarbossa or modified (Smith) Sgarbossa criteria can help in diagnosis of STEMI.

Sgarbossa Criteria

In cases of left bundle branch block or ventricular paced rhythm presenting with symptoms of ACS, Sgarbossa Criteria showing >3 points is suggestive of STEMI.

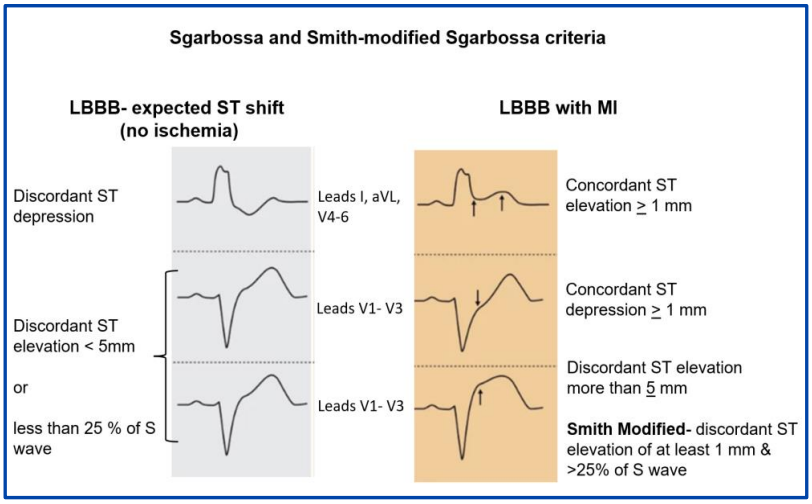
1. Concordant ST-segment elevation >1 mm in leads with a positive QRS complex (5 points)
2. Concordant ST-segment depression >1 mm in leads V1-V3 (3 points)
3. Discordant ST-segment elevation >5 mm in leads with a negative QRS complex (2 points)

Smith-modified Sgarbossa Criteria.

Here the third criteria as given above is modified for better accuracy. Criteria is positive if any of the following is present

Concordant ST-segment elevation of 1 mm in leads with a positive QRS complex

1. Concordant ST-segment depression of 1 mm in V1-V3
2. ST-segment elevation at the J-point, is at least 1 mm and has an amplitude of at least 25% of the preceding S-wave



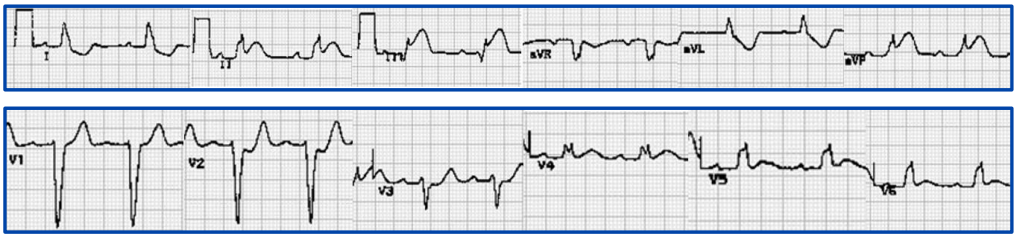


Figure 18 . LBBB with concordant ST elevation in leads II, III, aVF, discordant ST depression in V2, V3

Causes of ST segment elevation other than myocardial infarction.

ST segment elevation can occur in situations other than myocardial infarction. The points in favor of STEMI are

- Classical cardiac pain of ischemic nature
- Serial evolution of ECG in a classical pattern of STEMI including convex upward ST elevation ending with T inversion, development of Q wave.
- Reciprocal changes (ST depression in opposite placed leads)
- ST elevation in an arterial distribution.

Global ST segment elevation except in aVR and V1, PR segment depression and tachycardia are ECG changes suggestive of pericarditis. In myocardial infarction, the ST elevation is confined to arterial territory with reciprocal ST depression in oppositely placed leads. In the evolution, in myocardial infarction, T starts inverting when the ST is still elevated, where as in pericarditis, ST shift usually normalizes before the development of T inversion.

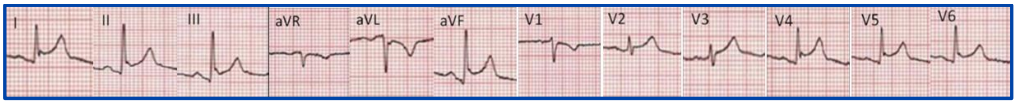


Figure 19. Pericarditis. Global ST segment elevation except in aVR and V1 and PR segment depression in lead II, III and absence of reciprocal ST depression.

Bradycardia, concave upward ST elevation with J point elevation, classically seen in mid-precordial leads (can be seen in inferior leads also), tall T waves and absence of reciprocal changes are suggestive of early repolarization syndrome. An rSR pattern in right precordial leads with down slopping ST elevation starting at the top of R wave and ending in a T inversion is suggestive of type 1 Brugada pattern. The examples of these are given below.

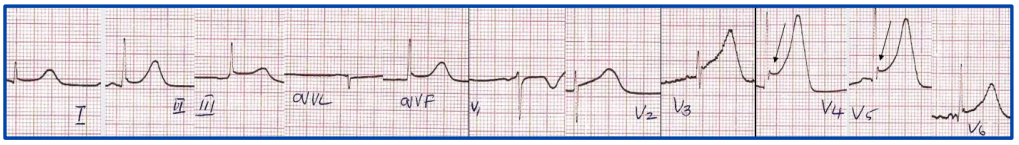


Figure 20. Early repolarization pattern. J point elevation (arrow) and concave upward ST elevation, maximum in mid precordial leads and tall T waves, no reciprocal changes (no ST depression in inferior leads).

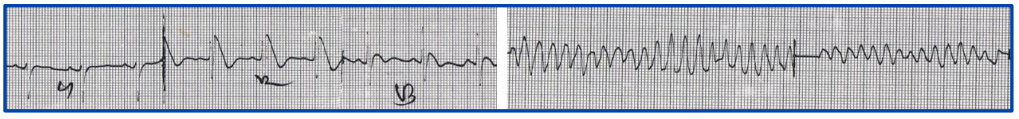


Figure 21. Brugada syndrome. rsR pattern in V1 or V2 with down-sloping ST segment elevation ending in T inversion. This patient developed cardiac arrest (VF), successfully resuscitated.

3.3.2 Biomarkers

Measurement of a biomarker of cardiomyocyte injury is to be done in all patients with suspected ACS whenever facility permits. Cardiac troponins are more sensitive and specific markers of cardiomyocyte injury than creatine kinase (CK), creatine kinase MB isoenzyme (CK-MB) and myoglobin and hence cardiac troponin I or T is preferred and should be available in all secondary or tertiary care hospitals. Elevation of cardiac troponin above the 99th percentile or the upper reference limit indicates MI. Troponin starts rising by 4-6 hours (Fig. 10) after chest pain. If the first value is negative, troponin can be repeated after six hours in cases of continuing pain or after 24 hours to rule out ACS.

Cardiac troponin may take hours to become elevated (biomarker blind period). Therefore, very early treatment like thrombolysis is not based on troponin elevation.

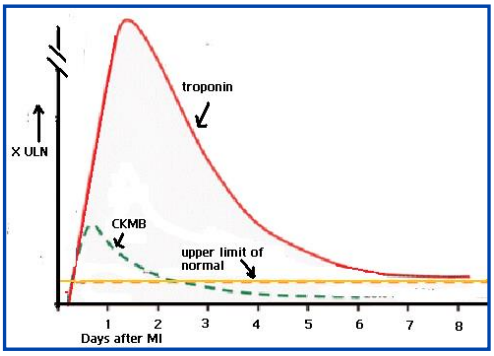


Figure 22. Pattern of rise and fall of troponin & CKMB in acute MI.

CK-MB shows a more rapid decline and normalize by 24-48 hours after MI and may provide added value for the timing of myocardial injury and the detection of early re-infarction compared to troponin. CK-MB is useful in renal disease where troponin can be elevated.

Cardiac troponins can remain elevated for 7-10 days after myocardial infarction. **High sensitivity troponin** (hs-Trop) can accurately measure smaller quantities of troponin and hence is useful in early rule in and rule out of MI. High sensitivity cardiac troponin is preferred over conventional troponin. Rule out and rule in algorithms for MI using hs-Trop are available. **However, this is not to be used to decide reperfusion strategy in STEMI.** Early rule in and rule out algorithms are used for suspected NSTEMI when the patient presents very early after chest pain. When initial very early hs-Trop is not elevated in patients

presenting within 3 hours of onset of cardiac pain, a repeat test is done either at 1 hour, 2 hour of first test or sometimes at 3 hour. This is called 0h/1h, 0h/2h or 0h/3h protocol where 0h test is the test done immediately at presentation to emergency department. The repeat test is useful when the presentation is very early after the onset of chest pain, i.e., <3 hours.

Which hs Trop to do and what values?

Both hs- Trop I and hs-Trop T can be used. However, the upper limit of normal value can vary with the method/manufacturer and hence is to be modified accordingly. The value also varies according to the population. The testing has to be standardized by the lab on the population being tested to get a good accuracy. Upper limit of normal (ULN) is the 99th percentile of value in healthy controls.

Rule in and rule out based on high sensitivity cardiac troponin T

As per the European society of cardiology recommendations, A value of hs-cTnT <5 ng/L at 0 hour or a 0-hour value <12 ng/L and change at 0 to 1-hour <3 ng/L rules out myocardial infarction. For 'rule in' of myocardial infarction, a 0-hour value of ≥52 ng/L or change of 0 to1-hour of ≥5 ng/L is used. Any hs-cTnT value or change not meeting the rule-out or rule-in criteria will qualify for observation category. When using 2-hour criteria, a value of 0 hour value of <14ng/L and change of value at 0 to 2-hour of less than 4ng/L may be used as rule out criteria. For rule in, a 2 hour value of ≥52 ng/L or change of 0 to 2-hour of ≥10 ng/L may be used.

Hs-Trop T	**Rule out**	**observe**	**Rule in**
0/1 hour testing	<5 ng/L at 0 hour **Or** 0 hour <12ng/L and change from 0 hour to 1 hour < 3ng/L	**All other patterns**	>52 ng/L at 0 hour **Or** change from 0 hour to 1 hour >5 ng/L

0/2 hour testing	<5 ng/L at 0 hour **Or** 0 hour <14ng/L and change from 0 hour to 1 hour < 4ng/L	**All other patterns**	>52 ng/L at 0 hour **Or** change from 0 hour to 1 hour >10 ng/L

Rule in and rule out criteria using troponin I

For hs-Trop I, different vendors have different testing methodology and hence for hs-Trop I we don't have a uniform value. A 0 hour value of <3ng/L may be taken as rule out and >60 ng/L may be taken as rule in. For 0/1 hour testing, a change less than 3 ng/L at 1 hour can rule out with high sensitivity and a change ≥8 ng/L can rule in with good specificity. Again, the repeat test and change in value is important when the patient presents very early with chest pain, within 3 hours. The rule out value has very high sensitivity.

Single value Hs-Trop I		**0/1 hour testing Hs-Trop I**		
Rule out	**Rule in**	**Rule out**	**observe**	**Rule in**
0 hour <3 ng/L	0-hour value of >60 ng/L	0 hour <4 ng/L Or 0 hour <5 ng/L change at 1 hour <3ng/L	**All other pattern**	0 hour value of >60 ng/L **Or** **Change at 1 hour >8ng/L**

Troponin and clinical probability can be combined for decision of rule in or rule out of ACS

Time of Hs-Trop	Rule in ACS	Rule out ACS	Observe
0 hour / 1 hour Or 0 hour/2 hour	Clinical probability high and + Hs- Trop value more than the Upper limit of normal at 0 hour or at 1 hours (or 0 hour/2 hour)	Pain free, low clinical probability + values of Hs –Trop less than ULN at both 0 hour and 1 hour (or 0 hour/2 hour)	Intermediate clinical probability + Rise of troponins in serial testing but not reaching the diagnostic value.

Causes of troponin elevation other than MI. Elevated C-tn do not always mean ACS. Elevated levels of cardiac troponins can occur several conditions other than ACS and hence troponin elevation should be always analysed on the background of clinical presentation.

Causes of Troponin elevation other than ACS.
• Renal dysfunction. • Myocardial injury other than due to ACS, as in the case of myocarditis, myocardial contusion, DC shock given for cardioversion or defibrillation • Left ventricular strain from congestive heart failure • Hypertensive crisis • Right ventricular strain from pulmonary embolism or other causes of acute pulmonary hypertension • Severe sepsis

- Hypotension or shock
- Sustained tachy or bradyarrhythmia
- Severe anemia
- Myocardial toxicity due to drugs(chemotherapy), poisons
- Stress cardiomyopathy
- Acute stroke
- Extreme exercise
- Rhabdomyolysis

From the clinical presentation the likelihood of CAD can be classified as low, intermediate or high.

Feature	**High likelihood**	**Intermediate likelihood**	**Low likelihood**
History	Chest or left arm pain or discomfort is the chief symptom and similar to prior documented angina, occurring in known CAD including MI + Any of the following	Chest or left arm pain or discomfort as chief symptom in Age >70, Male sex, Diabetes mellitus + Absence of high likelihood features and presence of any of the following	Probable ischemic symptoms in absence of any of the intermediate likelihood characteristic + Absence of high or intermediate likelihood features but may have any of the following
Examination	Transient MR murmur	Extra cardiac vascular disease	Chest pain reproduced by palpation

	Pulmonary edema or rales		
ECG	New or presumably new, transient ST segment elevation(≥0.1mV) or T inversion in multiple precordial leads	Fixed Q waves ST depression 0.05 to 0.1 mV or T inversion >0.1mV	T flattening or inversion <0.1mV in leads with dominant R waves or normal ECG
Cardiac markers	Elevated Cardiac Troponin I or T, or CKMB	Normal or borderline change	Normal

3.3.3 Lab investigations

Blood tests to be done in all cases at presentation

- Hemogram
- serum creatinine and blood urea
- blood sugar.

Other lab tests

- Liver function test, urinalysis, serum electrolytes, lipid profile, fasting blood sugar/post prandial blood sugar, PT-INR etc. may be done as per the clinical situation. For those needing coronary angiography (CAG), screening for markers of viral infection HBs Ag, HCV and HIV may also be done.
- **Blood Gas evaluation** may be done if available in cases of dyspnoea and shock.

3.3.4 Other investigations

Echo cardiogram

Early evaluation with 2D echocardiography, Doppler and Color Doppler may be done in cases of clinical ventricular dysfunction or hemodynamic disturbance. Echo may be done electively in stable cases. Estimation of left ventricular (LV) function is useful to prognosticate. Echo identifies ventricular wall motion abnormality, hypertrophy, abnormality of the valves, pericardial effusion or thrombus, dissection of aorta and may give clue to concomitant diseases. Mechanical complications like acute mitral regurgitation, ventricular septal rupture or free wall rupture can be diagnosed with Echo. In patients with dyspnoea, presence of lung comets or multiple B lines on the chest ultrasonography is highly suggestive of pulmonary oedema. (see chapter on heart failure). Ultrasound measurement of IVC is extremely useful in assessing hydration status. In dehydrated patients the likelihood of renal dysfunction, hypotension, which can be aggravated by many of the drugs like nitrate or ACE-I and poor coronary perfusion is likely.

Chest X-ray is not routinely needed in the acute setting of every ACS but will be useful in patients with dyspnea / desaturation. It is also useful to detect pneumonia, pneumothorax, rib fractures or other thoracic disorders which can present as chest pain. **CT and pulmonary angiogram** is useful in suspected case of pulmonary embolism and dissection of aorta.

4. Early risk stratification

Patients with ACS must be evaluated rapidly in order to identify those at risk of life-threatening arrhythmias and therefore needing close surveillance. All patients with ACS need monitoring. Those having continuing pain and high risk ACS need continuous ECG and monitoring of the vitals in an ICU. There are several scoring systems to assess the risk of ACS like TIMI risk scores and Killips classification.

4.1 The **TIMI risk score** in **NSTEMI/UA** uses seven variables in an additive way. A low TIMI score <3 usually indicates a low risk and a TIMI score $\geq$ 3 indicates intermediate or high risk. Decision on medical management or early invasive management can be done based on the risk.

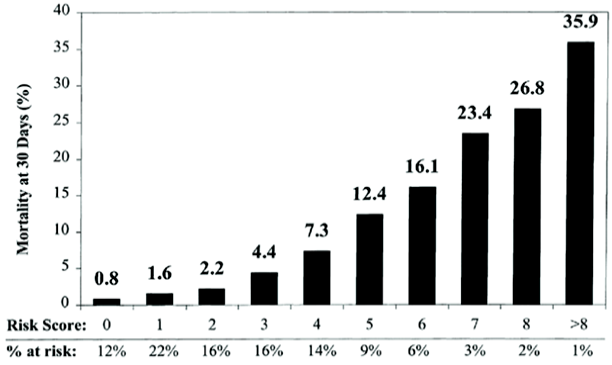
HISTORY	**Points**
Age ≥65 years	1
Three or more risk factors for coronary artery disease (CAD) (family history of CAD, hypertension, hypercholesterolemia, diabetes mellitus, tobacco use)	1
Known CAD (coronary stenosis >50%)	1
Aspirin use in the past 7 days	1
PRESENTATION	
Severe angina (≥2 episodes in 24 hours)	1
ST deviation ≥0.5 mm	1
Elevated cardiac marker level	1
RISK SCORE	Total 0-7

Risk of Cardiac events by 14 days		
Risk score	Death or MI	Death/ MI/ revascularization
0-1	3%	5%
2	3%	8%
3	5%	13%
4	7%	20%
5	12%	26%
6-7	19%	41%

4.2 TIMI score for STEMI. Since early reperfusion therapy is to be done in STEMI presenting in window period, risk scoring is not that important compared to NSTEMI in deciding various strategies in early management. TIMI scoring for STEMI has 8 variables and Killips class is one among them.

The table gives the variables assessed and the chart gives the risk for the TIMI score.

HISTORY	Points
Age	
65-74	2
≥75	3
DM/HTN or angina	1
EXAMINATION	
SBP<100	3
HR>100	2
Killip class II-IV	2
Weight <67	1
PRESENTATION	
Anterior STEMI or LBBB	1
Time to treatment >4 hours	1
Risk score	0-14



(Only 1% of patients with STEMI are at high risk with >8 score and 50% have score of 2 or less with a low risk)

Killip class is a very simple tool for risk stratifying. 'Killip class is based on clinical features at admission, and has independent prognostic value, with Class I at very low risk and Class IV at very high risk.

Killip class in STEMI

Class 1No signs of heart failure

Class II S3, elevated JVP, rales less than half of posterior lung fields

Class III Overt pulmonary edema

Class IV Cardiogenic shock

4.3 GRACE (Global registry of acute coronary events) Risk Score for ACS

Grace Score version 2.0 is widely used system for risk stratification in ACS. The link provided (https://www.mdcalc.com/calc/1099/grace-acs-risk-mortality-calculator) can be used to access the GRACE 2.0

The Variables used in GRACE 2.0 are

- Age
- Heart rate
- Systolic blood pressure

- Serum creatinine (renal function)
- Killip class (clinical heart failure)
- Cardiac arrest at admission
- ST-segment deviation
- Elevated cardiac biomarkers (troponin/enzyme presence)

Risk Category	GRACE Score Range	Clinical Risk
Low Risk	≤ 108	relatively low mortality
Intermediate	109–140	moderate risk
High Risk	> 140	higher mortality risk

The following are very high risk features in any ACS

- cardiogenic shock
- acute heart failure
- life-threatening arrhythmias or cardiac arrest
- recurrent or ongoing chest pain refractory to medical treatment
- mechanical complications of MI
- recurrent dynamic ST-T wave changes, particularly with intermittent ST-elevation

5. Management of ACS (STEMI and NSTEM/UA)

Once acute coronary syndrome is diagnosed, early, rapid and aggressive management is to be instituted. Immediate recognition and management of any life threatening emergency like hypotension / shock / pulmonary oedema / arrhythmia should be done. The specific management consists of administration of antiplatelets, antithrombotic like heparin, opening of the occluded vessel by reperfusion strategies, high dose statins, hypertension/diabetes management and management of complications like arrhythmia and heart failure.

Essential points in immediate care

All patients with diagnosed ACS should receive the following initially.

- **Dual antiplatelets**

  Aspirin 300 mg, to be chewed +

  One P2 Y12 inhibitor (like **Ticagrelor or Prasugrel or clopidogrel** - loading dose)
- **High dose atorvastatin** (40 or 80 mg) or rosuvastatin (20 mg to 40 mg).
- Secure an IV line as soon as diagnosis is made
- **Oxygen,** if saturation is <90% or if features of desaturation/dyspnea present.

All **STEMI** patients presenting within 12 hours to be considered for **reperfusion therapy.**

- **Primary percutaneous coronary intervention (PCI)** (reperfusion method of choice)
- **Thrombolysis** if delay of more than 120 minutes, from STEMI diagnosis to primary PCI
- **STEMI** patients presenting within 12 -24 hours PCI is the preferred reperfusion strategy
- STEMI presenting beyond 24 hours routine primary PCI not indicated. PCI may be considered if ongoing ischemia, acute heart failure, life threatening arrhythmia

NSTEMI/UA needs risk stratification. Thrombolysis is for **only STEMI** and is **not to be given** in UA/NSTEMI

- High risk NSTEMI/UA need early invasive management (PCI)

Antithrombotics, like heparin to be given in UA/NSTEMI and medially managed STEMI including thrombolysis in STEMI

Other medications based on need

- **Nitrates**- Sublingual nitrate/IV nitrate may be given to patients with pain if the BP is >100mm of Hg systolic. Caution / avoid in RV infarction.
- **Beta-blockers.** Metoprolol may be given in STEMI, especially if the patient has tachycardia and high blood pressure with no features of heart failure/AV block. In all UA/NSTEMI, early initiation of metoprolol may be done if there is no contraindication.
- **ACE-I and aldosterone receptor blocker** are indicated in STEMI and LV dysfunction and for hypertension management
- **Treatment of complications and comorbidities like diabetes, hypertension**

5.1 General measures

- With a quick evaluation identify presence of any life-threatening situations like shock, significant tachyarrhythmia or bradyarrhythmia and pulmonary edema. If any of the above is present, appropriate management for that may be given.
- All cases of STEMI and high-risk UA/NSTEMI should have an IV line secured. In case of hypotension, volume supplementation by IV infusion and pressor support may be initiated. Many patients with hypotension will be hypovolemic and unless volume is corrected, the BP will not stabilize.

Hypotension in ACS

- Hypovolemia- significant fluid loss can occur due to sweating and vomiting- volume correction needed. Clinical evaluation and IVC measurement by ultrasound will help.
- Massive myocardial damage- early reperfusion and inotropes
- Arrhythmia like atrial fibrillation or ventricular tachycardia- DC shock may be needed.

- Bezold-Jarisch reflex- seen in inferior wall MI associated with significant bradycardia and hypotension - atropine and IV fluids to be given
- Drug induced. Many drugs like nitrate, betablocker, and streptokinase can produce hypotension. Adjust the drug dose
- Mechanical complications like ventricular septal rupture/ pericardial tamponade

- **Oxygen administration** is to be considered when the patient is dyspnoeic and having desaturation. Administer oxygen, at 2-4 L/min, to maintain oxygen saturation > 90%. Routine administration of oxygen in stable patients with Spo2 >90% is not needed.
- In patients whose ischemic symptoms are not relieved by nitrates and beta-blockers, small dose of morphine (dose of 2-3 mg i.v. with Inj. promethazine 12.5 mg.) is useful. Morphine can be repeated to a maximum dose of 10 mg depending on the symptom and BP. Alternatively inj. fentanyl up to 100 mg can be useful for relief of pain. There is a concern that morphine may reduce the effectiveness of anti-platelets and hence is to be used in select patients having significant pain or pulmonary oedema.

5.2 Specific treatment of ACS.

5.2.1 Antiplatelets. All cases of ACS, should receive aspirin 300 mg (to chew) immediately, followed by 75-100/150 mg daily unless there is a contraindication for aspirin use. In case of aspirin use along with ticargrelor, the dose of aspirin should be less than 100 mg/day. Second antiplatelet agent, a P2Y12 inhibitor, ticagrelor or prasugrel or clopidogrel is to be given in addition to aspirin. Ticagrelor or prasugrel are prefered over clopidogrel in patients being managed with percutaneous coronary intervention. Clopidogrel is prefered in patients receiving thrombolysis.

Clopidogrel.

- Dose of clopidogrel is 300 mg given orally as a loading in case of thrombolysis in patients with <75 years of age or when non invasive (medical) management is planned.
- The loading dose for clopidogrel is 600 mg orally for angioplasty.
- Clopidogrel maintanance dose is 75mg orally daily.

- No loading dose in patients > 75 years of age.

Ticagrelor

- Dose of ticagrelor is 180 mg loading followed by 90 mg twice a day as maintanance.

Prasugrel

- Dose of prasugrel is 60 mg loading followed by 10mg daily adminstration. A lower dose of 5mg per day is considered when weight of patient is < 60 kg or age > 75 years (caution- risk of bleeding complication is higher in this group).
- Prasugrel is not to be given in patients with previous CVA, planning for early CABG, and should be careful in patients with low body weight and old age due to risk of bleeding.

In case clopidogrel is given earlier and a change to ticagrelor or prasugrel are considered, loading dose of those drugs are to be given.

Choice of antiplatelet

STEMI- thrombolysis	Aspirin + clopidogrel
STEMI – for PCI NSTEMI – for PCI	Aspirin + Ticagrelor or Aspirin + Prasugrel {Aspirin + clopidogrel (second choice compared to above)}
STEMI/NSTEMI, with no reperfusion strategy like PCI/lysis	Aspirin + Ticagrelor or Aspirin + clopidogrel
Post lysis pharmaco invasive therapy→ angioplasty	May change from clopidogrel to either ticagrelor or prasugrel with loading dose of the drug
Planning for CABG	Aspirin + Ticagrelor or Aspirin + clopidogrel

GP IIb/IIIa inhibitors are potent platelet inhibitors (eg. Tirofiban) They are not to be routinely given in ACS. In case of large thrombus burden while doing angioplasty or for cases of no flow/slow flow during angioplasty, it may be used as intracoronary administration followed by IV infusion for 24 hours in select cases.

5.2.2 Lipid lowering drug

Statin. High dose statin like atorvastatin 40-80 mg or rosuvastatin 20-40 mg is to be given at admission for all ACS patients and is to be continued daily in high internsity dose in all patients unless there is a contraindication. The dose of statin is not dependent on lipid level in the ACS setting and hence fasting lipd profile is not needed to initiate high dose statin.

Non statin drugs for lipid lowering.

In those with high levels of LDL-c, or in those presenting with ACS inspite of being on statin and LDL-c >70mg/dl, tab ezetimibe at 10mg per day may be considered in addition to statin. Other nonstatin drug which may be considered in patients having high LDL-c inspite of being on statin is bempedoic acid 180 mg daily or PCSK9 inhibitors like alirocumab, evolocumab or inclisiran. PCSK9 inhibitors are parenteral drugs and are coslty and are considered selectively.

5.2.3 Nitrates are useful in reducing pain and ischemic symptms by reducing preload (venodilation) and by coronary artery dilation. it also reduces the pulmonary congestion. It is useful in reducing the BP in hypertensive states. It may be given orally/sublingually or intravenously till the pain subsides and if the BP is >100mm of Hg systolic or till the BP is controlled in patients with high BP.

- Sublingual administration of 5 mg of isosorbide dintrate (or nitroglycerine) followed by repeat dose at 15 minutes interval may be given before i.v. line is established.
- Intravenous adminstration should be under careful blood pressure monitoring. The Starting dose of nitroglycerine (NTG) is 10 mcg /min. by i.v. infusion and the dose should be titrated upwards by 10mcg/min every 15-30 minutes, until symptoms are relieved, unless side effects (notably headache or hypotension) occur.
- In hypertensive patients NTG is given till the mean arterial blood pressure is reduced by at least 25%.

- **Caution.** Nitrates can cause significant hypotension in cases of right ventricular MI and in patients using sildenafil or other phospho diesterase - 5 (PDE-5) inhibitor and hence may be avoided. Right ventricular MI is to be expected in all cases of inferior wall MI especially when there is hypotension.

5.2.4 STEMI patients - Reperfusion therapy

Reperfusion therapy is to be considered urgently in all STEMI patients and are of two types.

- Percutaneous coronary Intervention (PCI).
- Thrombolysis or fibrinolysis

Window period-Reperfusion is indicated if a STEMI patient presents within the window period of 12 hours after the onset of index cardiac pain. Repefusion therapy should be given as early as possible. Routine primary PCI after 24 hours has not shown benefit. In cases of continuing ischemia, significant arrhythmia, shock or LV failure PCI may be done after 24 hours in select cases.

Which method of reperfusion?. Reperfusion by angioplasty is prefered over thrombolysis if the procedure can be done within 120 minutes of first diagnosis of STEMI. If angioplasty is not possible within in 120 minutes from diagnosis of STEMI (various reasons for delay are delay in decision making, financial issues and delay in transportation to another centre), thrombolysis is the reperfusion strategy.

- Decide on the mode of reperfusion strategy as early as possibe.
- In all STEMI patients presenting in window period of 12 hours from onset of pain, primary angioplasty is the prefered method of reperfusion if it can be done in 120 minutes from the point of STEMI diagnosis.
- Once decided to do PCI, wire crossing of the occluded vessel may be done within 60 minutes. In a patient refered from another centre for primary or rescue angioplasty, do a quick assessement and if PCI option is considered appropriate, shift to cathlab for PCI with minimum possible delay.
- If primary angioplasty is not **possible in time frame within 120 minutes of STEMI** diagnosis (eg. nonavailability of cathlab for timely intervention due to various reasons) or if there are **contraindications for PCI** or if the patient and relatives **do**

not want invasive management with angioplasty, thrombolysis may be administered without delay (if there is no contraindication to lysis).

- In patients with STEMI, reperfusion with PCI may be considered after 12 hours up to 24 hours.
- Routine PCI after 24 hours of STEMI is not recommended. In cases of continuing ischemia, significant arrhythmia, shock or LV failure, PCI may be done after 24 hours in select cases.
- **Early CAG** within 2- 24 hours **after thrombolysis** is to be considered in all feasible cases**. PCI** of infarct related artery may be considered if significant lesion is present in culprit vessel after thrombolysis especially in high risk cases.
- In stable patients, non culprit vessel revascularisation may be done in the same sitting, if the lesion is non complexIn shock, routine PCI of noninfarct related vessel is not to be done.
- PCI by radial access is preferred.

Time to reperfusion. Time to reperfusion should be minimised to the lowest in all cases of STEMI. Steps to reduce this are

- Early diagnosis. ECG to be done as early as possible, preferably in 10 minutes.
- Quick decision making on reperfusion strategy after discussing the options with patient and relatives.
- Consider a shift directly to cathlab from ED to prevent time delay, when primary angioplasty is decided.
- Thrombolytic therapy may be initiated in ED itself, when thrombolysis is the reperfusion strategy

Percutaneous coronary intervention. Different terminologies are used in angioplasty in STEMI

- **Primary PCI** is the term used when angioplasty is the first method of reperfusion in acute STEMI.
- **Rescue PCI.** Whenever there is evidence of failed thrombolysis rescue angioplasty is to be considered after discussing with the patient and relatives. Failed thrombolysis

is diagnosed when after lytic therapy, there is continuing ischemia, hemodynamic or electriclal instabillity and absence of ST resolution of at least 50% in the ECG taken at 60-90 minutes after starting lysis.

- **Pharmaco invasive PCI.** Routine PCI 2-24 hours after successful thrombolysis is a systematic pharmacoinvasive PCI. Rescue PCI in case of failed thormbolysis is also a pharmaco invasive strategy.

Thrombolytic therapy. It is indicated in STEMI patients presenting within 12 hours of onset of pain, when there are no contraindications, when primary PCI is not the choice of reperfusion because of delay in PCI (as discussed above), financial reasons or other contraindications or preferences. **(In NSTEMI/UA thrombolysis is contraindicated.)** There are absolute and relative contraindications to thrombolysis in STEMI.

Absolute contrindication for thrombolysis	**Relative contraindication for thrombolysis**
• Previous intracranial bleed • Ischemic stroke in 3 months except acute ischemis stroke where thrombolysis is treatment. • Malignant intracranial neoplasm • Cerebral vascular lesion like AV malformation • Active bleeding or bleeding diathesis • Aortic dissection • Significant closed head trauma in 3 months • Refractory hypertension of >180 mm Hg. systolic or >110 distolic. • Intracranial or intraspinal surgery within 2 months	• Oral anticoagulant therapy • Prior ischemic stroke >3 months • Pregnancy or within one week postpartum • Traumatic or prolonged CPR >10 minutes • Advanced liver disease or active peptic ulcer • Noncompressible vascular punctures in last 24 hours • Recent internal bleeding within 4 weeks • Chronic severe poorly controlled hypertension

Thrombolytic agents. Fibrin specific agents like tenecteplase or reteplase are prefered over streptokinase. Streptokinase is not fibrin-specific, requires to be given as an infusion over 30 minutes to one hour and may be associated with hypersensitivity reactions. Previous streptokinase administration can result in antibody formation and repeat administration is to be avoided especially within 6 months of previous use. Tenecteplase has the advantage of being fibrin-specific, can be given as a bolus dose, and has a lower incidence of hypersensitivity reactions. Reperfusion is significantly better with tenecteplase compared to streptokinase. Advantage of streptokinase is that is is very cheap compared to tenecteplase. Doses of individual lytic agents are given below. Those getting fibrin specific lytic agents should be given heparin or enoxaparin also(dose given below) to be started immediately with thrombolysis. Fondaparinux is an antithrombotic option after Streptokinase (dose 2.5 mg i.v. followed by 2.5mg/day/s.c)

Fibrinolytic agent	Dose
Streptokinase	1.5 million units as an infusion over 30-60 minutes
Reteplase	10 units + 10 units iv bolus given 10 minuts apart
Tenecteplase	Single IV bolus 30 mg if <60 kg body weight 35 mg if 60 to <70 kg body weight 40 mg if 70 to <80 kg body weight 45 mg if 80 to <90 kg body weight 50 mg if > 90 kg body weight Half the dose in > 75 years of age (co-therapy with parenteral anticoaglation with enoxaparin or heparin infusion is to be given, till PCI is performed or if PCI not done for the duration of the hospital stay for minimum of 48 hours and maximum 8 days - dose detailed below)

5.2.5 PCI in NSTEMI/UA. PCI is indicated in higher risk cases of NSTEMI/UA and may be considered as urgent/immediate or elective as per the risk at presentation. In very low risk cases of NSTEMI/UA, early medical management may be done. Use TIMI score/ GRACE score or look at high risk features discussed in risk stratification. The ACC guidelines give directions for invasive management for NSTEMI. The following is a modified approach suitable for our system.

Very high risk	High risk	Intermediate and low risk
Cardiogenic shock Heart failure Conitnuing ischemia Hemodynamic or electric instability	Grace Score >140 TIMI Score ≥3 Dynamic ST shift Steeply rising Troponin values	Grace score <140 TIMI Score <3 No ongoing pain, heart failure or electrical instability
Urgent CAG and revascularisation	Early CAG and revascularisation within hospital admission	Further risk assessment post discharge by stress test and PCI as needed. Optional- early in hospital CAG and reascularisation

5.2.6 Antithrombotics (heparin, enoxaparin or fondaparinux)

- Antithrombotics are indicated in all cases of STEMI with thrombolysis and in all cases of NSTEMI/UA until PCI is done. If PCI is not done, heparin may be continued till hospital discharge usually by 5th day, or for maximum 8 days. Continuation of antithrombotics is to be considered when there is persistant atrial fibrillation (AF) or left ventricular (LV) clot.
- **Dose of antithrombotic is** given in the table. Most commonly used low molecular weight heparin (LMWH) is enoxaparin.
- LMWH should not be given in patients with eGFR <15mL/min and here UFH is preferred.

Antithrombotic agent	Dose
Unfractionated heparin	For medical management, including post lysis, i.v. bolus of 60–70 IU/kg up to a maximum of 4000-5000 IU, followed by an infusion of 12 -16IU/kg/h up to a maximum of 1000 IU/h, aPTT target 50–75 sec or 1.5– 2.5 times the upper limit of normal. For PCI, initial bolus of 70-100 IU/kg in case of no GPIIb/IIIa administration and 60-70 IU/kg in case of GPIIb/IIIa co-therapy, under ACT monitoring
enoxaparin	Post thrombolysis, loading 30mg i.v. followed 15 minutes later by 1 mg/kg s/c twice daily. For >75 years of age, avoid bolus and 0.75 mg/kg per dose s/c twice a day In NSTEMI/UA, loading dose not to be given. Daily dose same. **When the eGFR is 15-30 mL/mt** reduce to 1mg/kg once a day
fondaparinux	2.5 mg iv bolus followd by 2.5 mg s/c daily for maximum 8 days. Not to be given in PCI.

5.2.7 Beta-blockers (BB) reduce myocardial oxygen consumption by lowering heart rate, BP and myocardial contractility and produce 13% relative risk reduction of mortality in the first week following MI. BB are more useful in patients with tachycardia and hypertension. Oral metoprolol may be considered in all cases of UA/NSTEMI in the absence of contraindications. I.V. metoprolol is selectively used in acute phase of STEMI, especially if there is tachycardia and hypertension. After acute phase is over, BB may be started by 24 hours, if not already started. BB (carvedilol, metoprolol succinate or bisoprolol) is indicated

in patients with reduced LV systolic function (see the chapter on management of heart failure).

Contraindication for initiating betablocker

- signs of heart failure- (in heart failure start at low dose, once congestive symptoms are controlled)
- systolic BP <100 mmHg
- heart rate <60 beats/min
- PR interval > 0.24 sec, second- or third-degree heart block
- active asthma or COPD- relative contraindication

If there is a contraindication to start early betablocker therapy, reassessment may be done at 24 hours and if the condition has resolved (eg. AV block), drug may be considered

Dose of oral metoprolol succinate is 25-50 mg 12 hourly, titrated up to 100 mg every 12 hour based on BP and HR. In select cases with high BP and tachycardia, if there is no evidence of heart failure, i.v. metoprolol may be given at a dose of 5mg, to be followed by two more doses of 5mg at 5-minute interval, to a maximum of 15 mg, provided heart rate > 60/mt and BP > 100 mm Hg systolic. Intravenous metoprolol is to be followed by oral metoprolol 50 mg within 6 hours. When there is LV dysfunction, preferred drugs are carvedilol, bisoprolol or metoprolol succinate to be started at low dose and stepped up gradually.

5.2.8 Other medications

- ACE-I or ARB may be started by 24 hours if there is no hypotension or elevated serum creatinine. ACE-I or ARB are indicated in symptomatic or asymptomatic LV dysfunction. They are also indicated in hypertension/ diabetes/ large area of infarction as in AWMI.

Drugs like enalapril, ramilpril or valsartan may be started based on the blood pressure monitoring and uptitrated.

- Aldosterone receptor blockers are indicated in LV dysfunction both in STEMI or NSTEMI. The usual dose is 25 mg/day of spironolactone or eplerenone.

- Calcium channel blockers like diltiazem and verapamil are to be avoided in cases of STEMI, and when there are features of heart failure. In NSTEMI/UA with no features of heart failure and not tolerating BB due to wheeze, diltiazem or verapamil may be considered as they reduce heart rate and have antianginal action. Amlodipine may be used for hypertension control as add on therapy in the continuing treatment of MI.

5.3 Referral from secondary care to tertiary care for PCI In STEMI/NSTEMI.

- Primary angioplasty (angioplasty as the primary mode of reperfusion in STEMI) is prefered over thrombolysis if the angioplasty can be done within 120 minutes of STEMI diagnosis. If the **time limit can be met**, patient may be immediatley referred to a tertiary care center, preferably after contacting the center to which patient is being refered to. If time delay for PCI is anticipated, for STEMI patients, thrombolysis should be done before referral (if no contraindication). Referral also should consider the clincial situation and risk of transportation and should be done after discussing with the patient/relatives regarding various issues in transportation.
- Angioplasty after thrombolysis may be considered in some situations. They are
    - **Rescue PCI.** Whenever there is evidence of failed thrombolysis rescue angioplasty may be considered after discussing with the patient and relatives. Failed thrombolysis is diagnosed when the patient after lytic therapy has continuing ischemia, hemodynamic or electriclal instabillity and absence of ST resolution by at least 50% in the ECG taken at 60-90 minutes after starting lysis.
    - **Early CAG after fibrinolysis followed by PCI of culprit vessel.** In cases of stable patients after lysis, a CAG may be considered within 2 to 24 hours after thrombolysis.

When considering referral for angioplasty, the centre to which patient is being refered to should be contacted and the feasibilty of angioplasty in 120 minutes from diagnosis of STEMI assessed and if angioplasty in time limit is not possible, thrombolysis may be initiated in the secondary care centre. When decided to give thrombolytic therapy, it should be initiated as early as possible, preferably in 10 minutes.

Details to be included in the referral letter when sending to tertiary care centre. All referred patients may be given a letter with a provisional clinical diagnosis along with ECG and other investigation reports. Drugs given and time of administration are to be mentioned. A copy of the check list can be useful during referral.

5.4. Monitoring and follow up.

All cases of ACS especially those with high risk should be monitored for a period of 24 - 48 hours after ischemia onset or till they are stable, preferably in the ICU. All patients who had PCI should be followed up with s. creatinine estimation. Care of arterial access site and observation for any bleeding or access site complications are important. Early inhospital rehabilitation and lifestyle modification are to be instituted. Advise regading healthy lifestyle and smoking cessation should be given.

<table>
<tr><th colspan="2">Acute coronary syndrome</th></tr>
<tr><td colspan="2">• General Care
• Identification and treatment of life-threatening complications
(Acute pulmonary edema, arrhythmia, cardiogenic shock)</td></tr>
<tr><th>STEMI</th><th>NSTEMI/UA (No ST elevation ACS)</th></tr>
<tr><td>Reperfusion in a time bound manner.
(time gap between first medical contact to reperfusion to be as short as possible)</td><td><u>No thrombolysis</u></td></tr>
</table>

<table>
<tr><td>If in window period
• Primary angioplasty (preferred) or
• Thrombolysis
(see text)</td><td>Angioplasty, based on the risk status
(see text)</td></tr>
<tr><td colspan="2">Other in hospital management
•Parenteral Anticoagulant (Heparin or enoxaparin)
•Dual antiplatelets- at diagnosis
•High dose statins- at diagnosis
•BB blocker- early initiation, within first 24 hours when no contraindication. See text
•RAAS blocker/MRA (especially LV dysfunction/diabetes/hypertension), early initiation. See text
•Treatment of hypertension and diabetes</td></tr>
<tr><td colspan="2">Longer term management
•Dual antiplatelets
•High dose statins
•BB blocker
•RAAS blocker/MRA (especially LV dysfunction/diabetes/hypertension)
•Treatment of hypertension and diabetes
•Treatment of heart failure/arrhythmia</td></tr>
</table>

Healthy lifestyle

smoking cessation, avoidance of alcohol, dietary advises, physical activity

Rehabilitation

6. Arrhythmias in Acute Coronary Syndrome

In patients with ACS and MI, the optimal heart rate is in the range of 60 beats/min in sinus rhythm. Tachycardia increases myocardial oxygen demand. All forms of tachycardia and bradycardia can depress cardiac output in patients with MI. The atrial contraction contributes to 20-25% of cardiac output normally. Atrial contribution to ventricular filling is affected in AF (no atrial contraction) and in complete AV block (CHB) and ventricular tachycardia (VT) due to atrioventricular dissociation. Hence in these arrhythmias, the cardiac output comes down, left atrial (LA) pressure increases (resulting in pulmonary congestion) and coronary perfusion becomes compromised further.

6.1 Brady arrhythmias

Sinus bradycardia. Sinus bradycardia is sinus rhythm with rate less than 60 bpm (beats per minute). Sinus bradycardia occurs more commonly in inferior wall STEMI compared to AWMI. It can occur as a result of sinus node ischemia or increased vagal activity. Mild sinus bradycardia (rate >40 bpm) with no hypotension does not need active management. When sinus bradycardia is associated with hypotension, or when bradycardia is significant (<40 bpm) and symptomatic, intravenous injection atropine 1.2 mg IV to a maximum dose of 3.6 mg IV and IV fluids are the treatment of choice. If not responding to atropine, heart rate may improve with isoprenaline infusion at a dose of 1mcg/mt. Potential complications of atropine will be tachycardia, worsening of ischemia and in low doses of atropine (eg: 0.6mg IV), paradoxical worsening of bradycardia due to Vago mimetic effects. If not responding to

these medications, temporary pacing is indicated in symptomatic or hemodynamically significant sinus bradycardia along with early reperfusion strategies.

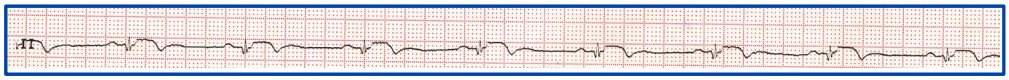


Figure 23: Example of sinus bradycardia in inferior wall STEMI. rate 50 bpm. Regular P wave followed by QRS

Atrioventricular (AV) Block. AV node is supplied by right coronary artery in 75-80% of persons. AV block is more common in inferior wall MI compared to anterior wall MI.

First degree AV block means prolongation of PR interval (more than 200 msec) with every P followed by QRS. No active management is needed for first degree AV block as there is no bradycardia due to first degree AV block alone. However, AV blocking agents like beta blockers are to be avoided and close monitoring of ECG is needed to detect worsening of AV conduction.

Second degree AV block can be Mobitz type 1 or type 2. In type 1 there is progressive prolongation of PR interval followed by failure of AV conduction. In Type 2, the PR interval is constant and there will be intermittent failure of conduction of atrial activity to ventricle. Mobitz type 2 AV block is not common in acute myocardial infarction. In addition to Mobitz type 1 and type 2 AV blocks, second degree AV block can be 2:1 AV block in which only alternate P waves are conducted. (See Figure). When two or more P waves are not conducted consecutively, it is called high degree AV block.

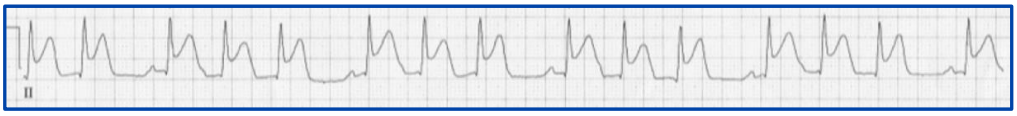


Figure 24: second degree type 1 AV block in inferior wall MI. Progressive prolongation of PR interval followed by failure of conduction.

Type 1 second degree AV block does not need active management for the arrhythmia per se. However, close ECG monitoring is needed. Second degree type 2 AV block can have worse prognosis than type 1 AV block and needs close monitoring. Generally, no specific

management for arrhythmia is needed. However, if the patient develops bradycardia related symptoms, management to increase heart rate will be needed, including atropine/pacing.

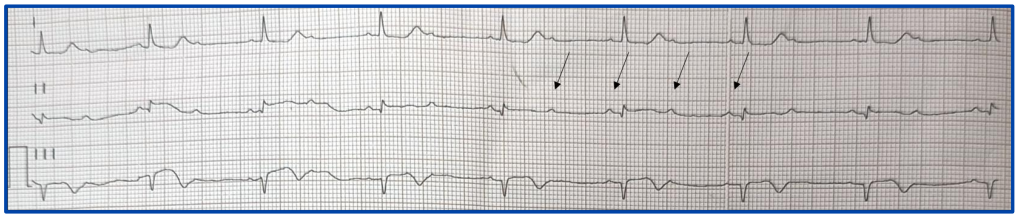


Figure25: 2nd Degree AV block with 2:1 conduction, in inferior wall MI. Arrows show P waves. Alternate P waves are followed by QRS. ST elevation is present in II and III

Complete atrioventricular block or complete heart block (CHB)

Complete Atrioventricular block can occur early or late after MI. When complete heart block develops in less than 6 hours of onset of ischemia, inj. atropine 1.2 mg IV is likely to abolish the AV block or cause acceleration of the escape rhythm. Early onset AVB is likely to be transient and related to increases in vagal tone. AV block occurring later in the course of MI may require cardiac pacing more often. CHB in inferior wall MI may not indicate a large MI whereas CHB is associated with larger infarct and worse outcome in AWMI. (see figure 7)

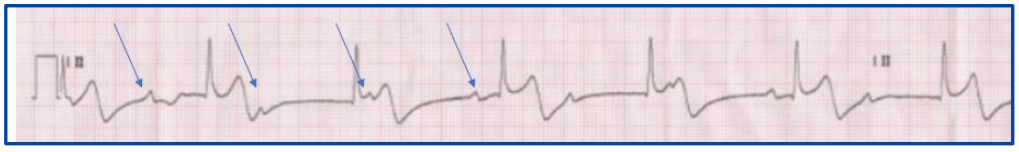


Figure 26: Inferior wall MI and complete heart block. Regular P wave, Regular QRS, AV dissociation, P faster than QRS.

CHB in Inferior wall MI	CHB in Anterior wall MI
CHB is more common in inferior wall MI	CHB is less common

Better prognosis in IWMI than AWMI	Poor prognosis, extensive necrosis, High mortality
Escape rhythm usually stable and narrow	Infranodal escape rhythm, wide QRS, escape rhythm unstable
Usually develops gradually	May develop rapidly

Temporary pacemaker is indicated in medically refractory, symptomatic or hemodynamically significant bradycardia due to sinus node dysfunction or AV block. Temporary pacing can be done rapidly by transcutaneous pacing using adhesive pads. This is especially useful in places with no cathlab facility or expertise for transvenous pacing.

Permanent pacemaker may be considered in acute MI after a waiting period of 5-14 days in IWMI, whereas AWMI may require permanent pace maker even early as recovery of AV conduction is less common in the latter.

General treatment approach to bradyarrhythmia in acute MI

- Adequate hydration
- Revascularisation as early as possible
- Inj. atropine when bradycardia is significant (rate <40 bpm or if there is hemodynamic disturbance)
- Correction of electrolyte abnormalities and maintenance of oxygenation.
- Temporary pacemaker when atropine is not effective.
- Permanent pacemaker if CHB persists for longer period (5-14 days)

Bundle branch block and fascicular block.

Bundle branch block and fascicular block may provide prognostic value during MI and can cause diagnostic challenges. Left anterior hemiblock is common and does not have additional prognostic value by itself. Left posterior hemiblock development is associated with poorer

prognosis as it occurs in larger infarcts (left posterolateral fascicle is thicker and has dual blood supply). Right bundle branch block in anterior wall MI suggests localization of obstruction to proximal left anterior descending artery (LAD) and has worse prognosis. Left bundle branch block development can confuse the diagnosis of acute MI as the LBBB itself may produce ST shifting. New development of Left bundle branch block in the setting of ACS can be considered as STEMI equivalent and is an indication for angiogram and revascularisation. Bi-fascicular blocks (RBBB+ LAHB/ RBBB+LPHB) carry higher likelihood of complications and needs meticulous monitoring.

6.2 Tachyarrhythmias

Sinus tachycardia (sinus rhythm with rate more than 100/mt)

The commonest arrhythmia in acute coronary syndrome is sinus tachycardia. Sinus tachycardia is more common in anterior wall MI than inferior wall MI. When a patient is having sinus tachycardia, the treating doctor has to rule out the common causes which can produce or contribute to tachycardia. It is important to correct the basic cause than blindly treat sinus tachycardia by drugs. Causes of sinus tachycardia in ACS can be

1. Hypovolemia (almost all patients with ACS/MI will be hypovolemic due to sweating, poor intake of fluids and sometimes vomiting)
2. Heart failure
3. Continuing ischemia, cardiac pain and reactive tachycardia
4. Drugs. Vasodilatory drugs can produce compensatory tachycardia.
5. Pericarditis
6. Other causes like fever/stress/anxiety/urinary retention/hyperthyroidism

A patient with tachycardia has higher myocardial oxygen demand. Heart rate reduction may be achieved by beta blockers, if there is no specific cause for sinus tachycardia. The commonly used beta blocker is metoprolol orally or as injection IV. The dose of metoprolol has been discussed already. Avoid betablocker in heart failure or hypotension.

Ventricular arrhythmias

Ventricular premature complexes (VPC).

Ventricular premature complexes are common in ACS. They can occur due to ischemia as well as a result of reperfusion. Heart failure, electrolyte abnormalities like hypokalemia or hypomagnesemia and hypoxia can increase the incidence of VPCs. Apart from early reperfusion therapy, correction of electrolytes, metabolic abnormalities, and early administration of betablocker is indicated. No active management with antiarrhythmic drugs like amiodarone is needed for VPCs.

Ventricular tachycardia.

Ventricular tachycardia (VT) is defined as 3 or more ventricular complexes in succession at a rate of > 100 bpm. VT can be sustained or non-sustained. When VT is associated with hemodynamic disturbance or lasts longer than 30 seconds, it is considered sustained VT. Approximately 6% of patients hospitalized with acute MI will develop significant sustained VT or VF within 48-hours of Admission. VT can be early or late.

- **Early VT**. VT occurring within 48 hours of hospital admission is due to Ischemia and/or reperfusion and has a better prognosis. Early VT/VF have increased 30-day mortality but no increase in long-term arrhythmic risk. Early VT is usually polymorphic.
- **Late onset VT**. Late onset VT is when VT occurs later than 48 hours post-MI. Patients with late VT experience worse outcomes and are more likely to have concomitant left ventricular dysfunction and severe heart failure. They are generally older and have other comorbidities. Late onset VT can be monomorphic.

Treatment of ventricular tachycardia.

Non-sustained Ventricular Tachycardia (NSVT) does not need specific antiarrhythmic therapy. The Management includes

- Beta blockers, if no contraindication
- Early revascularization

- Treatment of HF
- Correction of metabolic abnormalities especially hypokalemia.

Preferred treatment of sustained ventricular tachycardia in ACS is electrical cardioversion. Stable VT can be initially treated with antiarrhythmics like amiodarone and if not successful, treated with electrical cardioversion. Generally, VT converts with synchronized lower energy of 25 to 50 J. Pulse less VT should be managed as per ACLS protocol with higher energy asynchronized DC shock of 150 J or above. The two antiarrhythmics commonly used in hemodynamically stable VT or in recurrent VT are amiodarone and lignocaine. Recurrent non-sustained monomorphic VT should not be treated by DC shock. DC shock just converts the arrhythmia and does not prevent recurrence. All patients with VT should receive beta blocker unless contraindicated.

- Inj. Amiodarone. Amiodarone is given as a bolus dose of 150 mg over 10 min followed by continuous infusion at a rate of 1mg/minute over 6 hours (360 mg) followed by 0.5mg/minute thereafter (540 mg over 18 hours). In a center without infusion pump, easy calculation of administration is to give 150 mg (one ampoule) as slow bolus, followed by 300 mg (2 ampoules) in 5% dextrose over 6 hours as infusion; followed by 600 mg (4 ampoules) in 5% dextrose as infusion over 18 hours. Amiodarone infusion may take a longer time for conversion to sinus rhythm than lignocaine.
- Inj. Lignocaine. Inj. lignocaine bolus dose of 1 mg/kg over 2-3 mts, and a repeat bolus dose of 0.5 mg/kg up to 3mg/kg if needed if arrhythmia is not terminated, followed by continuous infusion of 1-4 mg/min for 24 hours.

An **electrical storm** (ES) is defined as the presence of at least three episodes of sustained ventricular tachycardia or ventricular fibrillation within 24 h.

Management of recurrent VT/VF and electrical storm in ACS

Cardioversion/Defibrillation

↓

Complete revascularisation and treatment of ischemia

Betablockers

Correction of electrolyte abnormality/ acid base imbalance/ hypoxia

Antiarrhythmics

Overdrive pacing (pacing at a faster rate than VT by a temporary pacemaker)

Stellate ganglion block.

Deep sedation

↓

Catheter ablation with EP study

Left ventricular assist devices/mechanical circulatory support

Accelerated idioventricular rhythm

Accelerated idioventricular rhythm (AIVR) is a common arrhythmia observed early after reperfusion. Up to one third of patients presenting with STEMI will have AIVR. AIVR is defined as 3 or more consecutive ventricular origin complexes at a rate of 60 - 100/minute. Abnormal automaticity in the subendocardial Purkinje fibers and sympathetic stimulatory effects in infarcted tissue undergoing reperfusion along with a slow sinus rate is the mechanism of production of AIVR. AIVR is often benign and self-limited and no treatment is needed.

When AIVR occurs within 6 hours after start of thrombolysis, it is considered early. Early AIVR and frequent (> 30 episodes/hour) and repetitive (occurring during > 3 consecutive hours) have high specificity but relative low sensitivity as a predictor of reperfusion. Very early AIVR (within 2 h of thrombolytic therapy) has a very good positive predictive value for successful reperfusion with a sensitivity of 38% and a specificity of 96%

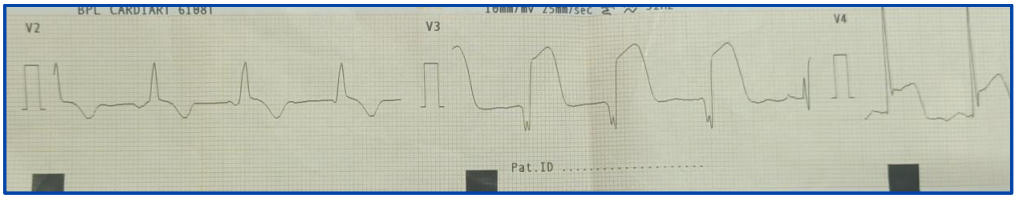


Figure 27 : Accelerated idioventricular rhythm in a case of anterior wall MI, regular, Wide QRS complexes without preceding P waves with a heart rate less than 100 bpm

Arrhythmia markers of reperfusion

- AIVR
- Frequent premature ventricular complexes (> two-fold increase in frequency within 90 minutes after the start of thrombolysis)
- Late diastolic VPC, VPC occurring after p wave in diastole
- Sinus bradycardia (with hypotension)
- Episodes of non-sustained ventricular tachycardia

Atrial fibrillation

Atrial fibrillation is the most frequent supraventricular arrhythmia in patients with ACS apart from sinus tachycardia. AF may be pre-existing one, or of new onset during the ACS. New onset atrial fibrillation occurring within 6 hours, especially after IWMI, is typically the result of atrial ischemia/infarction. Late onset of AF after 6 hours can be due to persistent ischemia or elevated filling pressures (LA pressure) and atrial stretch. Atrial fibrillation in AWMI usually suggest large ventricular infarction and LV dysfunction. Risk factors associated with developing atrial fibrillation include advanced age, increased extent of myocardial damage, depressed left ventricular function, and multi-vessel coronary disease. Occurrence of AF is an independent predictor of both in-hospital and long-term mortality.

AF is also associated with higher incidence of stroke, bleeding and recurrent ischemic events. Even silent or stable AF has negative effect on prognosis.

Management of AF

In hemodynamically stable patients, (no hypotension/heart failure/continuing cardiac pain), the primary focus is on the ventricular rate control and anticoagulation. The drug preferred for rate control is beta blockers (BB). Metoprolol orally IV can be given. Bisoprolol or nebivolol are other drugs which can be given orally. Non-dihydropyridine calcium channel blockers (CCB) like diltiazem or verapamil are not to be used in STEMI and LV dysfunction. CCB can be used in NSTEMI with good LV function when BB are contraindicated. Other drugs useful for rate control especially in presence of LV dysfunction are amiodarone and digoxin, but will take more time to act. In cases of hypotension, digoxin is preferred over amiodarone or beta-blockers.

Cardioversion with synchronized DC shock (150 J) is indicated in all hemodynamically unstable patients with AF. DC shock may also be considered in AF with uncontrolled ventricular rate persisting despite pharmacotherapy.

Anticoagulation

All patients having atrial fibrillation should receive anticoagulation. In the acute phase, UFH or LMWH are to be given. In cases of AF lasting more than 48 hours, oral anticoagulation is to be continued based on CHA2DS2VA score. When long term anticoagulation is considered, concomitant use of antiplatelet therapy may be modified. P2Y12 inhibitor of choice is clopidogrel. Dual antiplatelet therapy is converted to single antiplatelet therapy by 1 week to 4 weeks' time.

In patients with higher bleeding risk, a very short term - one week- dual antiplatelet therapy of aspirin and clopidogrel along with anticoagulant followed by single antiplatelet clopidogrel and anticoagulant is preferred.

In patients with higher thrombotic/ischemic risk and lower bleeding risk, dual antiplatelet therapy of aspirin and clopidogrel along with anticoagulant of 1 month duration followed by clopidogrel (single antiplatelet) and anticoagulant continuation is recommended.

Role of prophylactic antiarrhythmic therapy

Prophylactic anti arrhythmic management is generally not advisable. In cases of frequent VPC or runs of NSVT, the management will be early reperfusion, early use of beta blockers, correction of hypomagnesaemia/ /hypokalemia as well as statin therapy.

7. Check list for early management of ACS

Name ……age……sex……weight……time of presentation……

Clinical evaluation

Symptoms	Chest pain Classical cardiac pain not classical but suggestive noncardiac pain		Time of onset Duration	Other symptoms Dyspnoea Palpitaion Syncope
Other history	Diabetes Hypertension CKD CVA Wheeze Allergy Menstrual history	**Drug history** Antiplatelets Anticoagulants Phodphodiesterase inhibitor Betablocker Previous thrombolysis,	Any history of bleeding/active bleeding Contraindication to lytic therapy	Previous interventions (eg) PCI TAVR

		if yes the Agent		
Physical exam.	Pallor	**Pulse:** Rate/Rhythm/peripheral pusles **BP** All limbs (especially if pulse asymmetry)	JVP Hydration status	Features of heart failure 3rd heart sound Respiratoy rate Crepitations Pulse oximetry

Investigations

ECG	STEMI/STEMI equivalent Repeat ECG/ECG monitoring Serial changes Possibility of ST elevation other than MI?		Rate Rhythm PR interval	Axis RBBB LBBB LAHB/LPHB
Blood tests	C. Trop T/ I (Repeat if the first sample is very early)	CBC HB Platelet count PT/INR, aPTT	RBS S.Creatinine S. potassium eGFR	Lipid panel Viral markers if invasive management
Echo Cardiogram	EF and other measures of LV function RV function		IVC Lung comets/B lines	Clot Pericardial effusion MR/VSR

	RWMA		

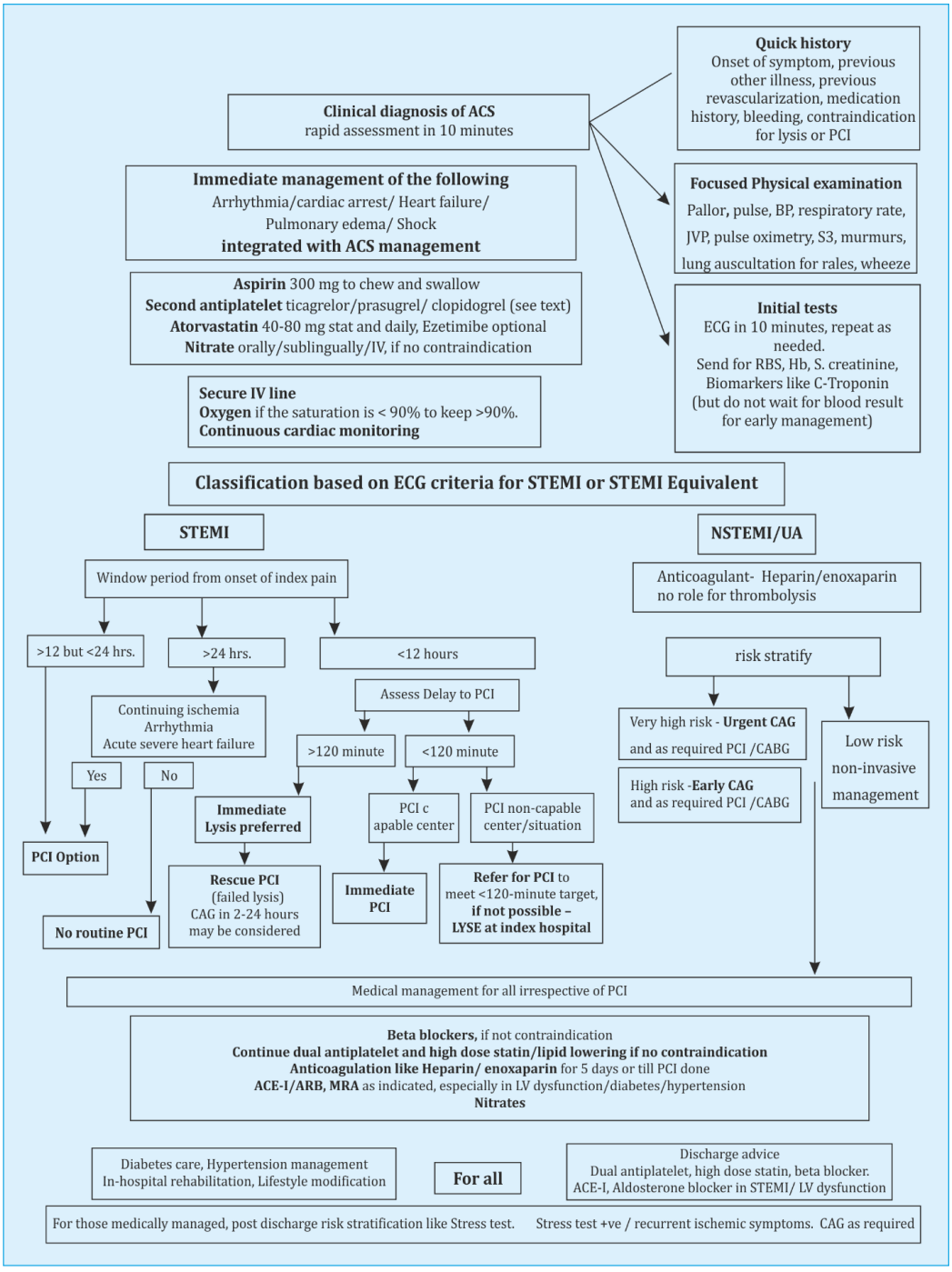
Provisional diagnosis STEMI NSTEMI/UA	**STEMI** Window period Late presentation? Symptoms Hemodynamics Arrhythmia Plan reperfusion strategy	**NSTEMI** **Risk stratify** for early invasive management TIMI score Grace score	**Very high risk features?** Acute heart failure Shock arrhythmia Cardiac arrest Recurrent pain Dynamic ST shift Mechanical complication
Alternate diagnosis? Think of pericarditis/myocarditis, stress cardiomyopathy, electrolyte abnormality		**Type of MI.** primary atherosclerotic or secondary to anemia etc	

Treatment

IV line Oxygen when needed Opiods when needed	Dual antiplatelets with appropriate loading dose anticoagulants	STEMI reperfusion Expected time to PCI from STEMI diagnosis >120 min/<120 min. Select PCI/fibrinolysis	NSTEMI High risk? Early invasive coronary managment needed?

		Is fibrinolysis success? Need for rescue PCI? post fibrinolysis- CAG in 2-24 hours?		Low risk - Medical
High dose statins + ezetemibe	Beta blocker Esepcially if hypertension and tachycardia	ACE-I/ARB LV dysfunction Diabetes Hypertension Anterior wall MI	MRA LV dysfunction	Other drugs nitrates

8. Management algorithm for ACS



9. Facilities to be available in hospitals for ACS care.

- Every health care center should have a functioning ECG machine available 24 hours a day.
- Finger pulse oximeter to assess saturation to be available in all hospitals
- Blood investigations like hemogram, S. Creatinine, RBS in all hospitals.
- High sensitivity troponin evaluation in tertiary care. Hs Troponin or regular troponin in secondary care center.
- A handheld echo machine/portable echo machine is desirable in the emergency department/casualty or ICU in tertiary care centre. Every emergency care physician should be trained in basic echocardiogram to assess LV function, valve dysfunction and regional wall motion abnormalities, to look for pericardial effusion and to look for lung comets of pulmonary oedema (see chapter on heart failure).
- Facility to do angioplasty, including cathlab (tertiary care) and Thrombolyse (tertiary care and secondary care).
- Facility for continuous monitoring of ECG and vitals and availability of defibrillator in secondary and tertiary care centre.
- Every center should initiate treatment of acute coronary syndrome as early as possible. Primary care centre should administer dual antiplatelets and high dose statin and start general care. Secondary and tertiary care centre should administer early reperfusion.
- Whenever there can be delay in angioplasty (as discussed in text), STEMI patients should receive thrombolytic therapy in a time bound manner (in secondary and tertiary care centre). All doctors with MD in internal medicine are qualified to give early ACS management including thrombolytic therapy.
- When clarification is needed regarding ECG change or arrhythmia, opinion can be sought from experts, over phone and by sending images through digital platforms like WhatsApp. All primary care, secondary care and also tertiary care centers should identify a predetermined expert or centre for this and seek the expert opinion rather than physically referring the patient and delaying treatment.

- It is useful to have risk calculators/ drug dose calculator/drip calculator in the smart phone of the healthcare provider and several algorithms/apps available in the internet may be downloaded to help in decision making

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Section - II

Evaluation and management of heart failure

Scope

Population

Adults more than 18 years of age; not applicable to paediatric population

Key clinical issues covered:

Evaluation of heart failure

Management of Heart Failure with reduced Ejection Fraction

Heart Failure with Preserved Ejection Fraction

Acute decompensated heart failure

Clinical issues not covered:

Detailed description of drugs and interventions

Health care setting:

Secondary and tertiary health care, patient presenting in the outpatient department or emergency department

Outcome:

Early diagnosis of heart failure with appropriate management and reduction in mortality and morbidity.

Abbreviations

ACEI	Angiotensin Converting Enzyme Inhibitor
ADHF	Acute Decompensated Heart Failure
ARB	Angiotensin receptor blocker
ARNI	Angiotensin Receptor Blocker Neprilysin inhibitor
BB	Beta Blocker
CAD	Coronary Artery Disease
CBC	Complete Blood Count
COPD	Chronic Obstructive Pulmonary Diseases
CVD	Cardiovascular Disease
DCM	Dilated Cardiomyopathy
ECG	Electrocardiogram
EF	Ejection fraction
HCM	Hypertrophic Cardiomyopathy
HF	Heart Failure
HFrEF	Heart Failure with Reduced Ejection Fraction
HFmrEF	Heart failure with mildly reduced ejection fraction
HFimpEF	Heart failure with improved ejection fraction
HFpEF	Heart Failure with Preserved Ejection Fraction

PPCM	Peripartum Cardiomyopathy
IHD	Ischemic Heart Disease
LVD	Left Ventricular Dysfunction
MI	Myocardial Infarction
NP	Natriuretic Peptide
OMT	Optimal Medical Therapy
PND	Paroxysmal Nocturnal Dyspnoea
SGLT2	Sodium – glucose 2 co transporter

Chapter I. Evaluation of Heart Failure

1. Introduction

Patients with heart failure (HF) present with various symptoms and signs. The predominant symptom of heart failure is dyspnoea. Dyspnoea can occur in other conditions, the commonest being chronic obstructive pulmonary disease (COPD). Similarly the major sign of HF, oedema, can be caused by many other conditions especially renal failure. It is important to sort out the symptoms and signs so as to diagnose HF early and to start appropriate treatment as the short term and long term mortality of HF are high.

2. Definition

HF as a clinical syndrome with symptoms and/or signs caused by a structural and/or functional cardiac abnormality and corroborated by elevated natriuretic peptide levels and/or objective evidence of pulmonary or systemic congestion.

3. Classification

HFrEF (HF with reduced Ejection Fraction)	HFmrEF (HF with mildly reduced Ejection Fraction)	HFimpEF (HF with improved Ejection Fraction)	HFpEF (HF with preserved Ejection Fraction)
Symptoms ± signs of heart failure LVEF ≤ 40%	Symptoms ± signs of heart failure LVEF 41- 49%	Symptoms ± signs of heart failure LVEF improved to >40%on follow-up with a previous LVEF ≤40%	Symptoms ± signs of heart failure LVEF ≥ 50 % Objective evidence of cardiac structural and/or functional abnormalities consistent with the presence of LV diastolic dysfunction/ raised LV filling pressures including raised natriuretic peptides

4. Staging

Stage A	Stage B	Stage C	Stage D
At risk for HF	Pre-HF	Symptomatic HF	Advanced HF

5. Diagnosis

The symptoms and signs could be the result of systemic congestion, pulmonary congestion and low cardiac output. These can be summarised as given in the table below

	Systemic Congestion	**Pulmonary congestion**	**Low cardiac output**
Symptoms	Weight gain Leg edema Early satiety Abdominal distension Bloating	Reduced exercise tolerance Dyspnea at rest Orthopnea Paroxysmal nocturnal dyspnea	Fatigue Decreased urine output Altered mental status Nausea/ vomiting
Signs	Edema Elevated jugular venous pressure Hepatomegaly	Tachypnea Tachycardia Basal lung crepitations Low oxygen saturation	Cool extremities Low volume pulse Hypotension Altered mental status Worsening renal function

5. a. Symptoms

Dyspnea, one of the typical symptoms of HF, can also be due to respiratory system diseases. The pointers to a cardiac cause are new onset of dyspnea with no previous history of wheezing or other respiratory symptoms and the presence of paroxysmal nocturnal dyspnea (PND). Patients having PND wake up from sleep 1-2 hours after going to bed with intense breathlessness which makes them sit up or even go out of the room to get air. The dyspnea subsides in about half an hour after which the patient will have a comfortable sleep with no recurrence in the same night. It may be associated with cough with pink frothy sputum. COPD patients also can have nocturnal dyspnea. Here the exacerbation occurs in the early morning hours (3 am - 4 am). HF can also present with less typical symptoms like nocturnal dry cough, wheezing, bloated feeling, loss of appetite, other gastrointestinal symptoms and confusion (especially in the elderly). When eliciting history, any underlying cause for HF and any precipitating factor leading to acute worsening must be investigated. Underlying causes include coronary artery disease, hypertension, valvular heart diseases and the use of cardio toxic medications or radiation. Precipitating factors include excess fluid intake, anemia, infection, thyrotoxicosis, intake of drugs retaining salt and water (e.g. NSAIDs) and stoppage of drugs given as treatment for HF. The symptom status should be expressed as NYHA class so that any improvement or deterioration of symptoms can be recorded objectively.

NYHA Class I	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnoea or angina
NYHA Class II	Slight limitation of physical activity. Comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea, or angina
NYHA Class III	Marked limitation of physical activity. Comfortable at rest. Less than ordinary activity causes fatigue, palpitation, dyspnoea or angina
NYHA Class IV	Unable to carry on any physical activity without discomfort. Symptoms of heart failure may be present at rest.

5. b. Signs

The following signs can be present

- cold extremities (warm extremities point to COPD and thyrotoxicosis)
- peripheral edema (ankle, sacral, scrotal)
- tachypnea
- tachycardia
- irregular pulse
- narrow pulse pressure
- elevated jugular venous pressure
- cardiomegaly
- third heart sound (gallop rhythm if there is tachycardia)
- murmur
- hepatomegaly
- ascites
- pulmonary crepitations
- signs of pleural effusion
- cachexia

5. c. Investigations

- **Blood**

  Complete blood count, blood sugar, blood urea, serum creatinine, sodium, potassium, magnesium, thyroid function tests and liver function tests are indicated in all HF patients.

  Anemia is a common precipitating factor and needs further evaluation including peripheral smear, serum iron, ferritin and transferrin saturations. Peripheral smear

showing microcytic hypochromic anemia indicates iron deficiency while macrocytic anemia indicates vitamin B12 deficiency. Elevated WBC count can be a clue to infection. HF as well as the drugs used to treat HF can lead to renal function derangement. Hence renal function tests are to be done prior to initiation of therapy and while continuing treatment. HF therapy can lead to electrolyte imbalance and frequent monitoring is needed. As both hyper and hypothyroidism can lead to or precipitate HF, thyroid function tests should be done.

- **Natriuretic Peptides(NP)**

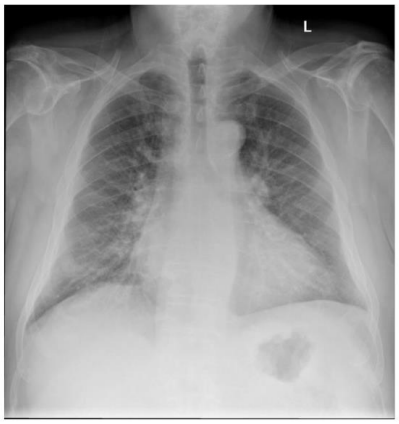
  Plasma concentrations of natriuretic peptides can be used to rule out HF if the values are below the cut off limits. Elevated concentrations support a diagnosis of HF and are useful for prognostication. But there are many other causes of elevated NP levels like AF, increasing age, and acute or chronic kidney disease which can result in false positive values. On the other hand NP concentrations may be disproportionately low in obese patients. The upper limits of normal in the non-acute setting are 35 pg/mL for BNP and 125 pg/mL for NT-pro BNP

- **Chest X-ray**

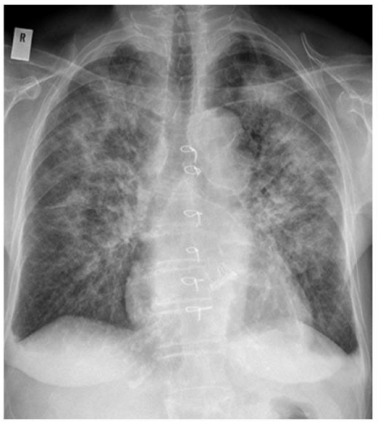
  Features of HF include cardiomegaly, prominence of upper lobe vessels, interstitial edema and pleural effusion. X-ray can give a clue about underlying valvular heart disease. But it may be normal especially in recent onset HF. It is also useful to rule out respiratory diseases like COPD, pneumonia or pneumothorax.

Larry Elliot's classification of pulmonary venous hypertension (simplified)			
Grade	X-ray feature	Acute disease, LA pressure in mm HG	Chronic disease, LA pressure in mm HG
Grade 1	Redistribution of blood	13-17	13-17

	flow with upper lobe vessels more prominent than lower lobe vessels		
Grade 2	Interstitial edema Peribronchial cuffing Kerley lines Interlobular effusion Pleural effusion	18-25	18-30
Grade 3	Alveolar odema/ Bilateral diffuse patchy Cotton wool opacities with bat wing appearance	>25	>30



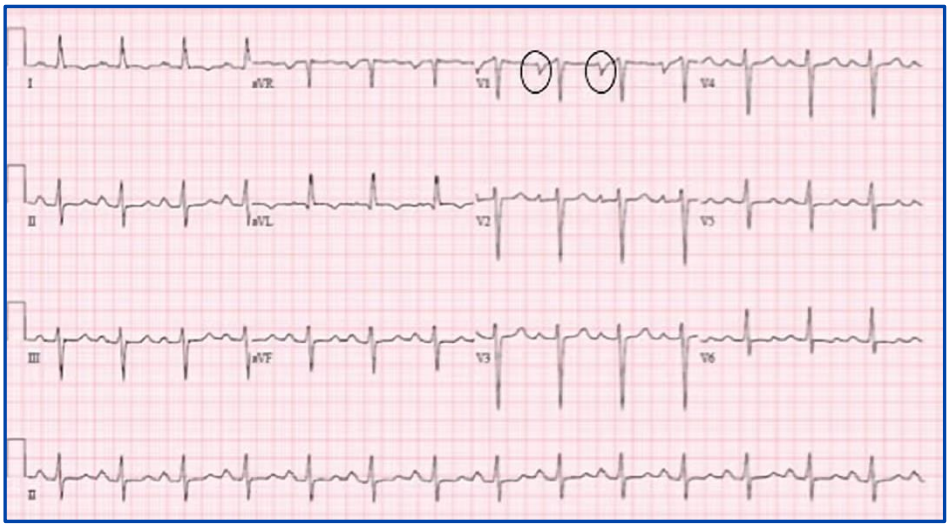
Xray showing Cardiomegaly and Prominent upper lobe vessels



Xray showing alveolar edema, bilateral opacities

- **ECG**

  There is no specific abnormality in ECG to diagnose HF. An abnormal ECG increases the likelihood of HF. Abnormalities on the ECG may provide information on etiology (e.g. myocardial infarction). ECG can help in diagnosing chamber enlargement, arrhythmias and electrolyte abnormalities.

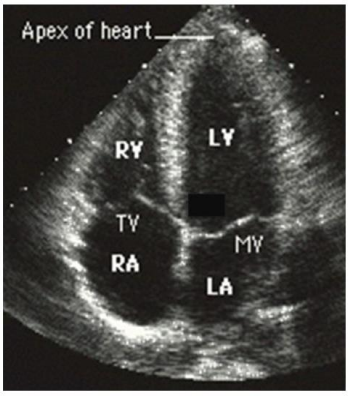


ECG showing sinus tachycardia and prominent negative P waves in V1 (circled)indicating left atrial overload which are common in HFrEF

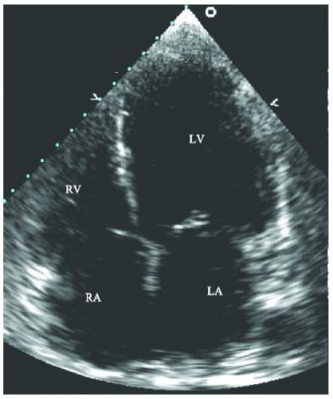
- **Echocardiogram**

  Following parameters are to assessed

  - LA size
  - LV size especially LV end systolic diameter
  - Ejection fraction to assess the systolic function
  - Wall motion abnormality- can be regional or global
  - Valvular disease
  - E/A reversal on mitral Doppler for diastolic dysfunction
  - Left ventricular hypertrophy
  - Pulmonary artery pressure
  - Right ventricular function
  - Inferior venecava – more than 21 mm in diameter and not collapsing by 50% on sniffing indicates fluid overload



Echo in normal person



Echo in HF showing dilated chambers

6. Advanced Investigations

- Trans esophageal Echocardiogram (TEE) –Consider in cases of poor trans thoracic window, prosthetic valve malfunction and suspected infective endocarditis
- Coronary angiography(CAG)- In patients with previous history of IHD, having angina pectoris or ECG and Echo evidence of CAD (after stabilizing HF)
- SPECT-(Single Photon Emission Computerized Tomography)-to look for viability of myocardium prior to revascularization
- Cardiac Magnetic Resonance Imaging (CMR)-to rule out suspected inflammatory / infiltrative conditions or cardiomyopathy

7. High risk indicators

- NYHA Class III/IV
- Hypotension
- EF <35%
- Severe pulmonary artery hypertension
- Renal failure
- Electrolyte imbalance
- RV Dysfunction

Chapter II. Management of Heart Failure with reduced Ejection Fraction

1. Introduction

The aims of treatment are to control the HF, stabilise the cardiac status, correct the precipitating factors, prevent progression of the disease and if possible correct the underlying cause. The main stay of treatment is medical in majority of patients so that drug therapy should be optimal with ***periodic stepping up or stepping down of the dose of medications***. Other modes of therapy like Cath lab based intervention, devices or surgery may be required in selected patients only. Classification based on the left ventricular function (LV ejection fraction) into HF with reduced EF (HFrEF), HF with mildly reduced ejection fraction (HFmrEF) or HF with preserved EF (HFpEF) is required prior to deciding the mode of treatment. (Refer Chapter I Section 3 classification). LVEF in the range of 40–49% (HFmrEF)is grouped along with HFrEF for treatment purpose.

2. Treatment- General Measures

- Assess all the drugs patient is taking and make necessary changes. Drugs which can produce fluid retention like non-steroid anti-inflammatory drugs, steroids and pioglitazone should be stopped
- Drugs for managing diabetes, hypertension, COPD or CAD should be continued
- In patients having Atrial Fibrillation oral anticoagulants should be given depending on CHADS-VAS score
- Salt restriction – less than 4 gm per day
- Fluid restriction- 1.2-1.5 litres per day if patient has features of fluid retention
- NYHA Class I and II Patients can be managed as outpatients
- NYHA Class III and IV are to be managed as in-patients
- In in-patients, propped up position as needed and oxygen inhalation if O2 saturation<90%
- Record heart rate, BP, respiratory rate and oxygen saturation by pulse oxymeter twice daily

- Weight recording daily- plan to lose at least 0.5 kg per day with proper treatment
- Blood investigations to be routinely monitored during hospital stay include Hb, serum levels of creatinine, sodium and potassium

3. Drug treatment for heart failure

This includes the use of diuretics, inotropes as well as the four pillars of HF therapy viz. (1) ACEI/ARB/ARNI (2) Beta blockers (3) SGLT2 inhibitors (4) MRAs. Out of these, all drugs except beta blocker can be started if patient's blood pressure permits even if patient has pulmonary odema. The general principles while using the drugs considered as four pillars of HF treatment are the following

- Early initiation and rapid up titration
- Titrating up to maximum recommended dose or maximum tolerated dose
- Monitoring for side effects and making change of dose or even the drug
- Modifying therapy based on the clinical status of patient

❖ **Diuretics**

Oral absorption of drugs may be decreased due to gut wall edema in patients with gross edema. In such patients, Inj. Furosemide 40 -80mg i.v. 8thhourly or 12th hourly or Furosemide i.v. infusion of 5-10 mg/ hr for 24 hours can be given. Monitor electrolytes and correct any deficit urgently. By 48 hours in most patients, parenteral diuretic can be converted to oral therapy. Tab. Furosemide 40 mg twice daily or Torsemide 10-20 mg daily can be given

❖ **ACEI/ARB/ARNI**(angiotensin converting enzyme inhibitor/ angiotensin receptor blocker/angiotensin receptor neprilysin inhibitor)

These drugs are effective in reducing hospitalizations for worsening heart failure as well cardiovascular and all-cause mortality. ARNI is superior to ACEI and must be considered as first line. If ARNI cannot be started, consider ACEI and if intolerant to ACEI start ARB. ARNI is an angiotensin receptor blocker (valsartan) combined with neprilysin inhibitor (Sacubitril). Natriuretic peptides naturally promote diuresis and are degraded by neprilysin. Neprilysin inhibitor raises

levels of natriuretic peptides and induces diuresis. When introducing ARNI, if the patient is already on ACEI, stop ACEI and start ARNI only after 36 hours. If the patient is on ARB, it can be directly switched over to ARNI without waiting period. Strict monitoring of blood pressure, serum electrolytes and renal function should be done after initiation and titration. Contraindications are renal dysfunction with creatinine >2.5 mg/dl, hyperkalemia with potassium more than 5.5 meq/L and pregnancy. (See Table I, II and III for doses)

- **Beta blockers**

  Beta blockers have shown to improve symptoms, reduce the risk of HF hospitalization, and reduce mortality. BB should be initiated in clinically stable, euvolemic patients with normal BP and heart rate at a low dose and gradually up titrated to the maximum tolerated dose. In patients admitted with acute decompensated HF, beta blockers should be cautiously initiated in hospital, once the patient is hemodynamically stabilized. (See Table IV for doses). Contraindications are atrioventricular block, significant bradycardia with heart rate less than 50 beats per minute and extensive bronchospasm.

- **Mineralocorticoid receptor antagonists (MRAs)**

  MRAs improve HF outcomes by blocking the effects of aldosterone, a hormone that can worsen heart failure. Watch for hyperkalemia and consider changing Spironolactone to Eplerenone if patient complains of painful gynecomastia. Finerenone is a third-generation non-steroidal mineralocorticoid receptor antagonist (MRA).It selectively binds to mineralocorticoid receptors, reducing the side effect especially in patients with CKD and type II Diabetes and is particularly useful in patients with mildly reduced EF. (See Table V for doses)

- **SGLT2 inhibitors**

  Lead to osmotic diuresis and natriuresis and improve heart failure by reducing preload and afterload as well as by reducing blood pressure. SGLT inhibitors reduce the risk of HF hospitalization and CV mortality, irrespective of the presence of diabetes. It is not approved for use in patients with type 1 diabetes due to increased risk of diabetic ketoacidosis. They can cause urogenital

infections and if recurrent infections occur, they must be discontinued. (See Table VI for doses)

- **Acetazolamide**

  Is a carbonic anhydrase inhibitor that reduces proximal tubular sodium reabsorption. This can improve the efficiency of loop diuretics, potentially leading to more and faster decongestion in patients with acute decompensated heart failure with volume overload. 500 mg IV once daily for 3 days can be given along with loop diuretics. 250 mg once daily can be given in patients with body weight less than 60 kg. This drug is particularly beneficial in patients with alkalosis as well as hypochloremia.

- **I.V Inotropes**

  In extremely dyspneic patients a short period- 24 to 48 hours- of inotropic support can be helpful. I.V Dobutamine can be given as infusion at a dose of 5 microgram/kg/min and can be increased up to 20 microgram/kg/min

- **Vasodilators**

  A combination of nitrates and hydralazine can be used for vasodilatation in patients who cannot be given ARNI/ACEI or ARB. (See Table VII for doses)

- **Vericiguat**

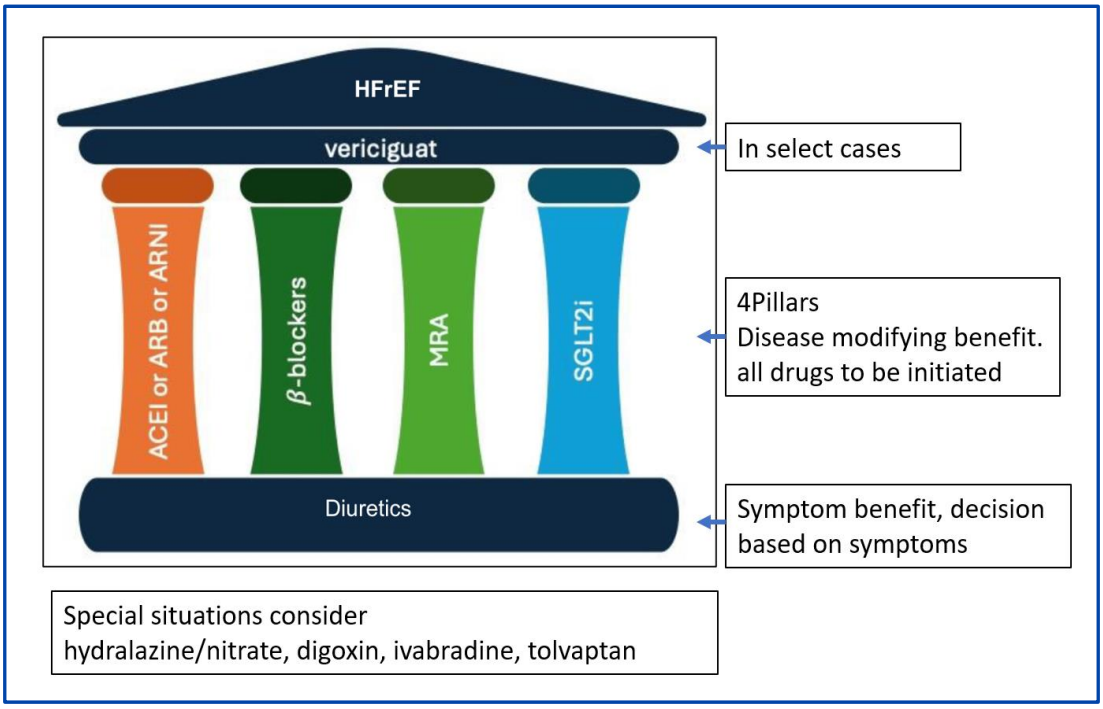
  Vericiguat is an oral soluble guanyl cyclase stimulator which binds and stimulates soluble guanyl cyclase and increases cyclic GMP production in cells. In the myocardium it reduces fibrosis, remodelling and hypertrophy. At the vascular level, it reduces constriction, inflammation and remodelling. At the renal level it increases renal perfusion and reduces salt and water retention. All these effects are beneficial for the failing heart. It can be started at a dose of 2.5 mg daily and increased every 2 weeks to reach the target dose of 10mg or the maximum tolerated dose.

4. Optimal care vs. basic care

Even though all the drugs mentioned above are to be used in all patients as optimal care, practically it may not be possible due to various reasons like intolerance of patients to certain drugs and financial constraints. In such cases as basic care the following drugs can be given-

ACEI + MRA + BB + SGLT2 inhibitors. Use of ARNI may be limited by the higher cost of the drug.

Pictorial summary of Drug treatment of HFrEF showing the 4 pillars



5. Managing patient who is improving clinically from symptoms

- Change to oral diuretics from parenteral
- Step up ARNI/ACEI/ARB
- If not started on BB, start BB
- Advise ambulation- walk slowly for short distance in the ward- gradually increase
- On discharge ensure that all heart failure patients are **initiated on all "4 pillars "of heart therapy and titrated to maximum permitted dose**
- Schedule review at 2 weeks with the following investigations- Hb, Serum levels of creatinine, sodium and potassium

6. Managing a patient who is not clinically improving

- Delay initiation of beta blockers, if the patient is having significant congestive features
- Check for compliance to drugs, salt and fluid restriction
- Add a second diuretic if sodium and potassium are normal. Add metolazone 2.5 mg once daily. Can be stepped up if needed with monitoring of electrolytes
- Digoxin 0.125 mg once daily may be started (Watch for side effects)
- Ivabradine can be used to slow heart rate if the patient is in sinus rhythm in whom BB cannot be started or are not tolerated or when the heart rate cannot be brought down below 70/min with BB alone. Ivabradine should not be used in atrial fibrillation. Starting dose is 5 mg twice daily and can be stepped up to 7.5 mg twice daily if needed to control heart rate
- Tolvaptan: To be considered in cases with hyponatremia and fluid overload for a short-term use. Better to use for in-patients only with careful monitoring of electrolytes.

7. Resistant Heart Failure

If patient has not improved by 5 days, consider referral to a specialized centre for special investigations and advanced modes of management

8. Follow UP

- **First review- 2 weeks after discharge**

  a) See investigation results- Hb, S. creatinine, sodium and potassium and take corrective steps if abnormal.

  b) Reduce diuretics if no fluid overload

  c) Step up ARNI/ACEI/ARB if BP is more than 100 mm of Hg

  d) Step up Beta blocker if heart rate is high and BP is more than 100 mm of Hg

e) Consider H Influenza and pneumococcal vaccination in cases with repeated hospitalization

- **Second review- 1 month after first review**

a) Do investigations as in previous step and correct if needed

b) Earlier review if patient had issues like creatinine or potassium derangement in the first review

c) Try to step up ARNI/ACEI/ARB, BB and MRA depending on BP and heart rate

- **Long term follow up**

a) Reduce diuretics to minimum possible dose or stop

b) Continue the four pillars, ARNI/ARB/ACEI, BB, SGLT2 Inhibitors and MRAs at maximum tolerated/ maximum recommended dose

c) Continue the 4 pillars of treatment in HF with improved ejection fraction also (see chapter 1 for definition)

9. Problems during follow up

a) Dry cough: Can be due to ARNI/ACEI – change to ARB if sleep is disturbed

b) Cramps: Electrolyte imbalance due to diuretics. Correct by oral supplementation and by reducing diuretics

c) Painful gynecomastia can be due to spironolactone or digoxin. Try changing to Eplerenone

d) Anemia: Absolute or functional iron deficiency (ID) is seen in around 50% patients with HF. Evaluate for the type and cause and correct appropriately. Iron deficiency is defined as ferritin < 100 mcg/L or ferritin 100-300 mcg/L with transferrin saturation of ≤ 20%. In patients with HF meeting these criteria even without anemia, intravenous iron repletion improves exercise capacity and quality of life. In patients with symptomatic HF and iron deficiency, with or without anemia, intravenous iron replacement (preferably ferric carboxymaltose) is to be considered for symptom improvement. Another way of correction of Iron deficit is to give Iron Sucrose as

drip infusion of 200 mg of ISC (in 0.9% sodium chloride solution) weekly for 3 or 5 weeks, depending on the total required dose.

e) Hypotension – May need dose reduction or temporary discontinuation of ARNI/ACEI/ARB, BB or MRAs

f) Renal impairment – If creatinine levels increase less than 30 % from baseline, ARNI/ACEI/ARB can be continued with close monitoring of creatinine values. If creatinine values increase by more than 30%, ARNI/ACEI/ARB may need to be adjusted or discontinued. If creatinine levels increase by more than 50%, ARNI/ACEI/ARB should be discontinued.

10. Doses of drugs for Initiation and titration (Tables I-VII)

Table I. ARNI

Drug	Starting Dose	Target dose
Sacubitril/Valsartan	24/26 mg twice daily orally (1/2 tablet twice daily can be used if BP is low)	97/103 mg twice daily

Table II. ACE Inhibitors

Drug	Starting Dose	Target dose
Enalapril	2.5 mg twice daily orally	20 mg twice daily
Ramipril	1.25 mg once daily orally	10 mg once daily
Lisinopril	2.5 mg oncedaily orally	20mg once daily

Table III. ARBs

Drug	Starting dose	Target dose
Losartan	25mg once daily orally	100mg once daily
Valsartan	40mg twice daily orally	160mg twice daily

Table IV- Beta Blockers

Drug	Starting dose	Target dose
Carvedilol	3.125 mg twice daily orally	25mg twice daily
Metoprolol succinate (CR/XL) (sustained release only to be used)	12.5 mg once daily orally	100-200 mg once daily
Bisoprolol	1.25mg once daily	10mg once daily

Table V. MRA

Drug	Starting Dose	Target dose
Spironolactone	12.5-25 mg daily	25-50 mg daily
Eplerenone	25 mg daily	50 mg daily
Finerenone	10 mg daily	20-40 mg daily

Table VI. SGLT2 Inhibitors

Drug	Starting Dose	Target dose
Dapagliflozin	10 mg daily orally	10 mg daily orally
Empagliflozin	10 mg daily orally	10 mg daily orally

Table VII. Vasodilators

Drug	Starting Dose	Target dose
Fixed-dose combination isosorbide dinitrate/hydralazine	20 mg/37.5 mg (one tab) thrice daily	20 mg/37.5 mg (two tab) thrice daily

11. Pregnancy

Heart failure may be pre-existing or can occur de novo during pregnancy. Pre-existing HF can worsen during pregnancy. HF occurring for the first time during pregnancy can be due to unmasking of previously undiagnosed conditions like cardiomyopathies and valve diseases, Takotsubo syndrome due to emotional stress or Peripartum cardiomyopathy (PPCM). PPCM is heart failure with EF <45% occurring towards the end of pregnancy (usually in the last one month) or in the months following delivery without any other identifiable cause. Many of the drugs used to treat HF are contraindicated in pregnancy. They include ACE-Is, ARBs, ARNI, MRAs, Ivabradine, and SGLT2 inhibitors. If a patient is on these drugs and is planning pregnancy, these drugs should be stopped prior to conception after evaluation in a specialist centre. Loop diuretics, Beta-1-selective blockers like Metoprolol, hydralazine, oral nitrates and digoxin are safe in pregnancy and are used for heart failure treatment. In cases of AF or other indications like prosthetic valve, anticoagulation may be needed. Avoid directly acting anticoagulants like apixaban, rivaroxaban etc during pregnancy and lactation. Heparin, LMWH and Vitamin K antagonist like warfarin can be used if needed. Vitamin K antagonist is to be avoided in first trimester of pregnancy, (in case of high risk for thrombosis, as in mechanical prosthetic valve and if the dose of warfarin is less than 5mg/day to maintain INR, warfarin can be given in first trimester after discussion with patient). If on Vit K antagonist, it may be changed to heparin or LWMH at 36 weeks or 2 weeks prior to planned delivery. After delivery, during lactation, beta blockers, ACE-I like enalapril, MRA like spironolactone, diuretics and warfarin can be given if needed.

12. Advanced modes of treatment

A. **Implantable Cardioverter Defibrillator (ICD)** -to be considered in HF patients who have survived a cardiac arrest. Can be considered in patients with symptomatic HF with LVEF ≤35% despite ≥3 months of OMT.

B. **Cardiac resynchronization therapy (CRT)** -For HF patients who continue to be symptomatic despite OMT and in sinus rhythm with QRS morphology of LBBB type and QRS duration more of than 150 milliseconds

C. **Mechanical circulatory support (Left Ventricular Assist Devices LVAD)-**can be considered in HF patients ***as a bridge to transplant***. It can be considered in patients with severe symptoms for last 2 months despite optimal medical and device therapy if LVEF <25%, and there are ≥3 HF hospitalizations in last 1 year

D. **Cardiac Transplantation-**May be considered for end-stage HF patient with severe symptoms, a poor prognosis, and no remaining alternative treatment options. Patient should be motivated, well informed, and emotionally stable and capable of complying with the intensive treatment required postoperatively

Chapter III. Heart failure with preserved EF (HFpEF)

1. Introduction

When a patient has symptoms and signs of Heart Failure but echo showing normal systolic function in the form of an Ejection Fraction above 50 % HFpEF is considered. Clinical presentation can be similar to patients with heart failure with reduced EF. The following table shows the approximate proportion of symptoms in HFpEF

Dyspnoea on Exertion	98%
Fatigue	59%
Edema	45%
Orthopnea	22%
JVP elevation	17%
Wheezing	11%
PND	7.6%
Dyspnoea at rest	4.6%

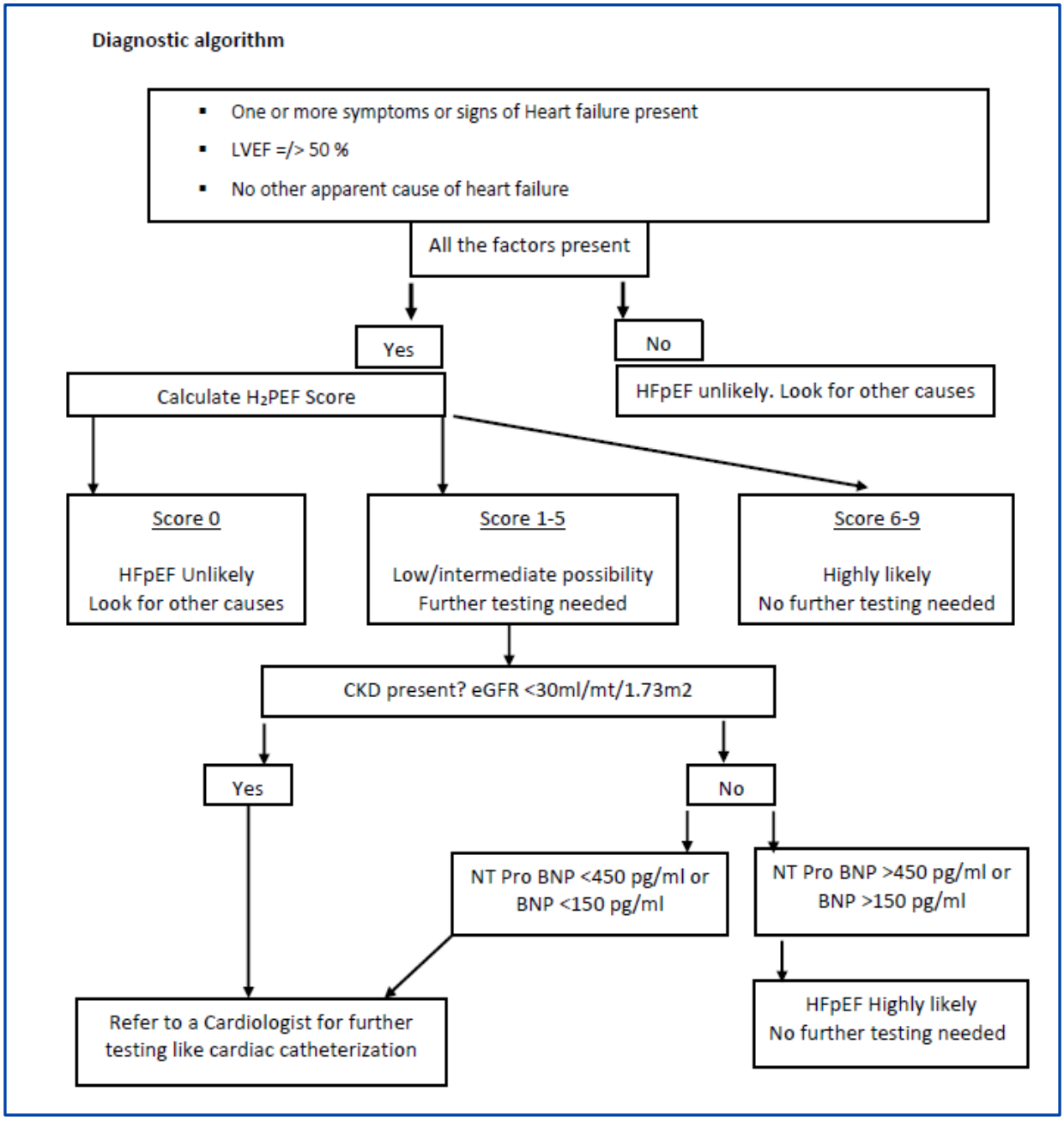
2. Diagnosis- Scoring system & Algorithm

Diagnosis of HFpEF can be difficult as there are many mimickers for this condition and a single factor like Ejection Fraction is not useful. Many factors are associated with HFpEF and one can do a scoring initially and then follow an algorithm based on those factors. A popular scoring system is the H_2FPEF Scoring system. H_2FPEF is an acronym for the following H- Heavy, H- Hypertension, F- Atrial fibrillation, P- Pulmonary hypertension, E- Elderly and F- Filling pressure. Each parameter is given points and scoring is done as given in the table below

H_2FPEF Scoring

Acronym letter	Parameter indicated	Points given
H	Heavy	1
H	Hypertension	2
F	Atrial Fibrillation	3
P	Pulmonary Artery Systolic Pressure > 35 mm of Hg	1
E	Elderly(Age more than 60 years)	1
F	Filling Pressure elevation in ventricle as denoted by an Echo parameter Mitral E/e'	1

Diagnostic algorithm



3. Treatment of HFpEF

Drugs like ARNI/ACEI/ARB and Beta blockers used in HFrEF are found to be not that useful in HFpEF. The co morbidities should be well controlled in HFpEF. Exercise, diet, weight loss, and cardiac rehabilitation play a major role in treatment. Recent trials have shown the usefulness of SGLT2 Inhibitors as well as Semaglutide. Semaglutide is an antidiabetic medication used for the treatment of type 2 diabetes and an anti-obesity medication used for long-term weight management It is a peptide similar to the hormone glucagon like peptide 1 (GLP-1).The following algorithm can be used to guide treatment.

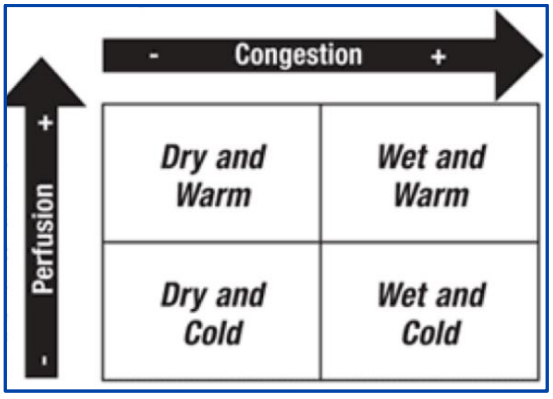
- Manage volume overload by using diuretics as in HFrEF
- If there is no contraindication start SGLT2 Inhibitors
- Check BMI
- If BMI > 30 kg/m^2 start Semaglutide. Assess the weight loss periodically. Once 10 % weight loss has occurred and despite that symptoms are persisting add MRA.
- If BMI< 30kg/m^2 start MRA if symptoms persist after SGLT2 Inhibitor use
- Finerenone is a third-generation non-steroidal mineralocorticoid receptor antagonist (MRA). This is particularly useful in HFpEF.

Chapter IV. Acute Decompensated Heart Failure (ADHF)

1. Definition and classification

ADHF is the rapid onset or acute worsening of symptoms and signs of heart failure. It requires urgent evaluation and treatment as it is a life-threatening emergency. ADHF may present as a first occurrence (20% cases) or, more frequently, because of acute decompensation in chronic HF patients (80% cases). In ADHF the symptoms can be of ***congestion*** (systemic and or pulmonary- the **WET** cases) or of ***low cardiac output*** (the **DRY** cases**).**There can be four different classes of patients.

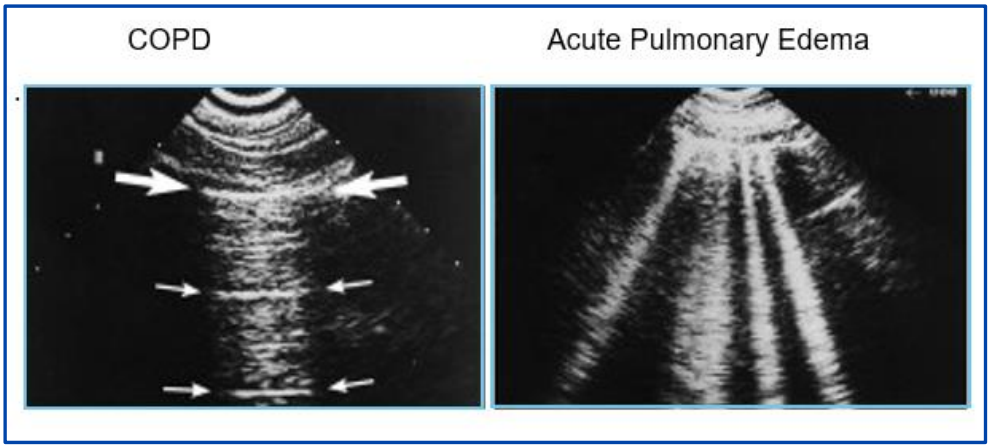
- **wet and warm** : congestion with normal cardiac output
- **wet and cold** : congestion with low cardiac output
- **dry and cold** : no congestion but low cardiac output
- **dry and warm** : no congestion and normal cardiac output



2. Investigations(Similar to Chronic Heart Failure)

- **X-ray Chest**: Confirms pulmonary congestion(may even show the typical bat's wing appearance of pulmonary edema) and excludes respiratory causes
- **ECG** : Assess rhythm and any ongoing ischemia
- **Natriuretic Peptides**: Plasma BNP levels less than 100 pg/ml or NT- Pro BNP less than 300 pg/ml excludes AHF (Cut off values are higher in the acute setting)

- **Echocardiogram**: Limited bedside Echo can assess LV function by noting the ejection fraction, systemic congestion by noting inferior venecaval distension more than 21 mm and pulmonary congestion by noting comet sign on lungs. Echo also helps to differentiate acute HF from conditions like pulmonary embolism, cardiac tamponade, exacerbation of COPD
- **Lung Ultrasound**: Look for comet tail artefacts in lung fields which are specific for acute pulmonary edema. This is done by keeping an ultrasound probe in the intercostal spaces



3. Management

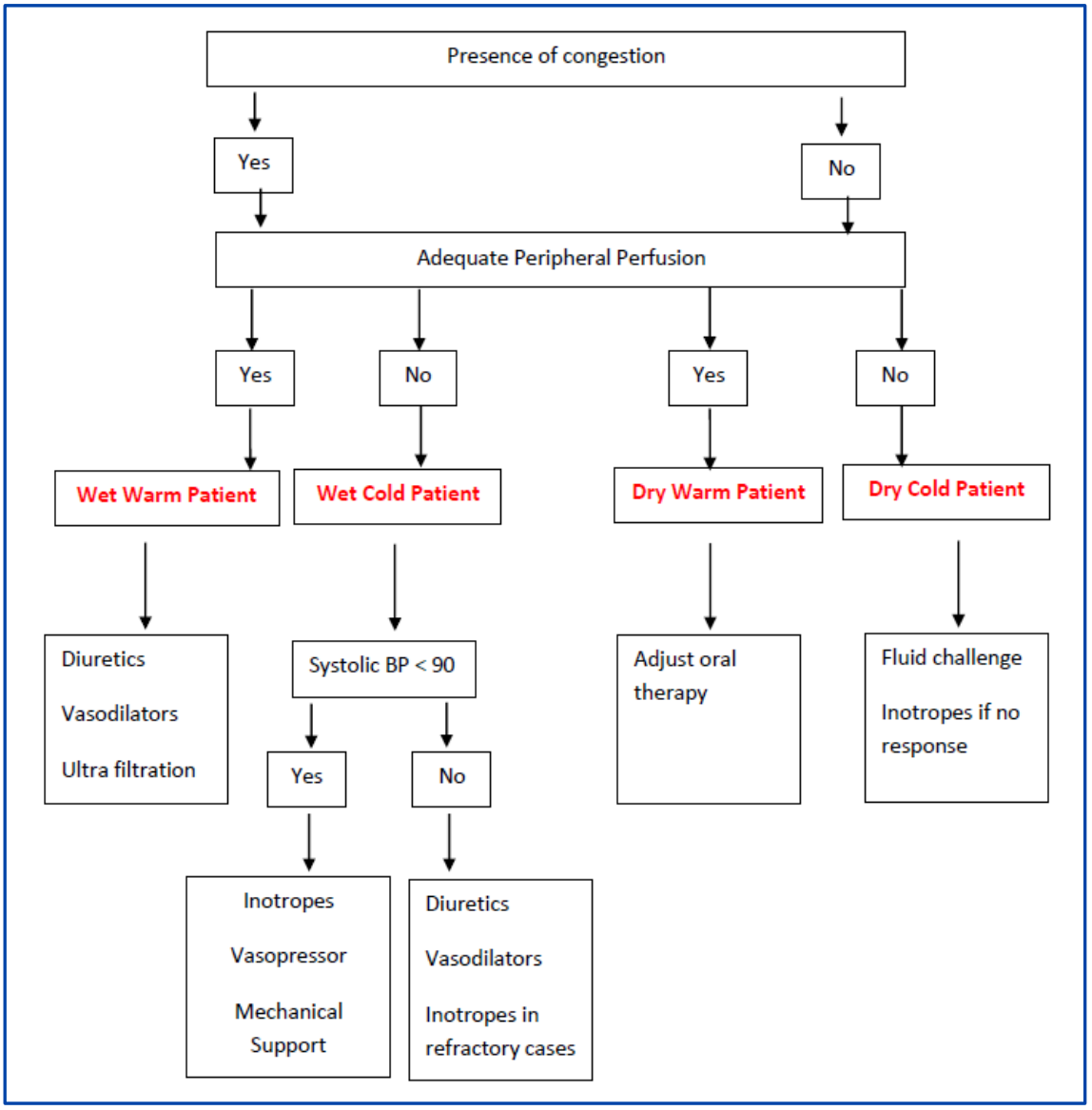
- Identify the cause or precipitating factor of ADHF and correct it. It may be high blood pressure, arrhythmia, acute ischemia, infection or drugs
- Oxygen can be given to all patients to relieve dyspnoea. Non-invasive and even invasive ventilation can be given if oxygen saturation remains persistently low.
- Intravenous Diuretics- Furosemide can be given IV. Dose can be decided based on blood pressure. If systolic BP is above 90 mm of Hg, 40-60 mg Furosemide can be given. If BP is lower than 90 mm of Hg smaller doses 20- 40 mg can be given.
- Intravenous Acetazolamide : Acetazolamide is a carbonic anhydrase inhibitor that reduces proximal tubular sodium reabsorption. This can improve the efficiency of loop diuretics, potentially leading to more and faster decongestion in patients

with acute decompensated heart failure with volume overload. This has been demonstrated in ADVOR trial. It has to be given along with high dose IV loop diuretics. The dosage is yet to be specified. 500 mg IV once daily for 3 days was the dose used in ADVOR trial. Smaller doses like 250 mg once daily has also been tried in patients with body weight less than 60 kg. This drug is particularly beneficial in patients with alkalosis as well as hypochloremia.

- Intravenous vasodilators: Are to be given if blood pressure is high. The agents used are
  - Nitro-glycerine- start with 20 micro gram /min; can go up to 400 microgram/min
  - Nitroprusside- start with 10 micro gram /min; can go up to 350 microgram/min
- Intravenous inotropes: Are to be given if BP is low and there are signs of hypo perfusion. The agents used are
  - Dopamine: 5-10 micro gm/kg/min can increase to 10-15 micro gm/kg/min
  - Dobutamine: 5-10 micro gm/kg/min can increase to 10-20 micro gm/kg/min
  - Milrinone: bolus 50 micro gm/kg followed by 0.125 – 0.75 micro gm/kg/min
  - Levosimendan: loading dose of 6-12microgm/kg followed by 0.05 – 0.2 microgram/kg/min as infusion
- Adjustment of pre-existing drugs
  - ACEI/ARB- can be continued in most cases. Temporary withdrawal or reduction in dose may be needed in case of severe symptomatic hypotension or creatinine rise
  - Beta Blockers : Can be withheld if patient has pulmonary edema
  - Mineralocorticoid antagonists : With hold if creatinine is more than 2.5 mg/dl or potassium more than 5.5 meq/L
  - Digoxin can be started if not already being given

- If not improving
  - Ultra filtration: can be tried if diuretics are not producing adequate response
  - LV assist devices
  - Look for any unidentified underlying cause
  - Refer to a dedicated heart failure team

Management algorithm



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