Clinical Management Guidelines for Coronary Artery Disease for National Programme for Prevention and Control of Diabetes, Cardiovascular Disease and Stroke



Partners

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Abbreviations

ACS	Acute coronary syndrome
ACEI	Angiostensin converting enzyme inhibitor
AIVR	Accelerated idioventricular rhythm
AMI	Acute myocardial infarction
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
CHF	Congestive heart failure
CSA	Chronic stable angina
CHC	Community health centre
CVD	Cardiovascular disease
IABP	Intra aortic balloon pump
ICD	Implantable cardioverter defibrillator
LAD	Left anterior descending
LCX	Left circumflex
LMWH	Low molecular weight heparin
LVEF	Left ventricular ejection fraction
LVOTO	Left ventricular outflow tract obstruction
MI	Myocardial infarction
NPJT	Nonparoxysmal junctional tachycardia
NSTEACS	Non ST elevation Acute coronary syndrome
NSTEMI	Non ST elevation myocardial infarction
PCI	Percutaneous coronary intervention
PHC	Primary health centre
PSVT	Paroxysmal supraventricular tachycardia
RCA	Right coronary artery
STEMI	ST elevation Myocardial infarction
VAD	Ventricular assist device

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Executive Summary

India is currently experiencing an epidemic of Coronary artery disease (CAD). Statistics show that 20-25% of all medical admissions[19] and 25% of all mortality is due to CAD. After extreme poverty and infectious diseases, control of heart attack can be the most rewarding for Indians in the 21st century for saving productive life years. The unhealthy life style practices viz., unbalanced dietary pattern, lack of physical activity, tobacco consumption, ill effects of urbanization, psychosocial stress, all contribute to a greater risk of developing CAD in Indians. The increasing rates of CAD mortality and the projected rise in CAD mortality for 2020 in the developing world necessitate immediate prevention and control measures.

Experience in the developed world has shown that significant reductions in CAD prevalence and mortality can be achieved via primary and secondary preventive efforts as well as timely intervention and medical therapy. Despite this alarming burden of CVD, there are no definite guidelines at the national level to combat this serious problem. As thus, the need for clinical management guidelines was considered.

The clinical management guidelines for CAD for National Programme for Diabetes, CVDs and Stroke (NPDCS) has been designed as per the requirement of Indian Public Health Standard (IPHS) and National Rural Health Mission (NRHM) to make the assessment and management of coronary artery disease feasible, community oriented and evidence based as well as to prevent the risk of CAD in more easy and scientific way. This management guideline focuses the need of preventive measures, timely screening of high risk population, and immediate assessment, intervention as well as continued medical therapy once CAD is established.

The recommendations are based upon health service infrastructure data, local evidence based studies as well as major international guidelines. Available situation analysis was carried out to know the complete infrastructure of Indian health care delivery system. The recommendations were subsequently compiled and reviewed by the participants and experts investigators, senior cardiologist, and epidemiologist in multiple sessions. These guidelines are described as two broad categories: chronic stable angina and acute coronary syndrome. Recommendations for different levels of health care delivery systems in India, in a step-wise pattern are the principal objective of these guidelines. It is hoped that the recommendations will help the medical officers and physicians to effectively manage coronary artery diseases.

1. INTODUCTION

Coronary Artery Disease (CAD) is the leading cause of death globally where India has the highest burden. It causes 3 million deaths/ year, accounting for 25% of all mortality in India. Hospitals statistics reveal that 20-25 % of all medical admissions are due to Coronary artery disease[19]. According to the National Commission on Macroeconomics and Health (NCMH), there would be around 62 million patients with CAD by 2015 in India, and of these, 23 million would be patients younger than 40 years of age. By 2020, 60% of the world's heart disease is expected to occur in India.

The risk of CAD in Indians is 3-4 times higher than white Americans, 6 times higher than Chinese and 20 times higher than Japanese[23]. CAD is affecting Indians 5-10 years earlier than other communities; in some studies from South India, the percentage of patients below 45 years suffering from AMI is reported to be as high as 25-40%.

Asian-Indians have a 40% higher mortality rate from CAD than their white counterparts[22]. Despite a recent decline in the developed countries, both CAD mortality and the prevalence of CAD risk factors continue to rise rapidly in the developing countries. Clearly, there is a need for concerned efforts directed at prevention and effective treatment of this epidemic.

2. PURPOSE OF THESE GUIDELINES

India has highest burden of acute coronary syndromes in the world, yet little is known about the treatments and outcomes of this disease. The most striking feature of management of patients with cardiovascular disease in India, is its heterogenicity: from patients treated at tertiary and teaching hospitals, who receive the best possible evidence-based care, to patients who have poor or, even no, access to specialist care and whose condition, therefore, is poorly treated

Till date there are no standard guidelines in the national level to combat this serious problem. Though there are hundreds of guidelines in the world, none has focused the Indian health situation and are thus poorly applicable. Ministry of Health and Family welfare, Government of India has launched National Programme for Diabetes, CVDs and Stroke (NPDCS) in January 2008 on pilot basis in the country to formulate a standard management guideline for the same. These guidelines are intended to assist both cardiovascular specialist and non specialists in the proper evaluation, management and prompt referral of patients with an acute onset of symptoms suggestive of these conditions, based on the level of health care delivery system in India.

Application of these principles with carefully reasoned clinical judgment will definitely reduce the high mortality from this syndrome in the national level as well as reduce the cardiac damage caused by ACS

3. HEALTH CARE SYSTEM – THE STRUCTURE AND CURRENT SCENARIO

Centres	Population norms	Health care staff	Services(cardiac)
SUB-CENTRE	5000 (3000)*	Male health worker,female health worker,voluntary HW	-
PHC (4-6 beds)	30,000 (20,000)*	Medical officer, Pharmacist, Staff nurse, Female Health Worker,Health Educator,Health Assistant (M&F)	Limited Blood tests, Oxygen trolley, ECG
CHC (30 bedded)	1,20,000 (80,000)*	Physician,surgeon,obstetrician,pediatrician,anaesthetist,staffnurses,dresser,pharmacist/compounder, opthalmic assistant,laboratory technician,radiographer,ward boys	ECG,Defibrillator,Ultrsound,Blood tests,essential drugs
Sub-divisional (30-100 beds)	5-6 lakh people	Specialists (med,surg,obs,paed,anaesthsia,opthalmology,com. Health,skin & VD,dental care)	ECG,Defibrillator,Ultrsound,Blood tests, drugs
District Hospitals (101-500beds)	> 6 lakhs	Specialists (including cardiologists)	ECG,TMT,Holter,Echo,Thrombolytic therapy,ICU facilities (No cath lab)
Medical Colleges	For more coverage	Medical & Surgical Specialists	ICU facilities, Cath lab may or may not

& Tertiary Centres	in each state		be available

* population covered in hilly/tribal areas

4. RECOMMENDATIONS FOR LEVELS OF CARE

The facilities available at different levels of health services are heterogeneous at different states of the country. Moreover there is scope for further upgrade of the present setup. The working group has suggested tiered system of health care delivery for the management of patients with CAD. Therefore, we will describe the recommendations for four levels of health facilities**.**

Definitions of Levels

Level 1 has a physician trained to interpret ECG. ECG facility is available however a defibrillator is not available.

Level 2 has a medical specialist trained in thrombolysis if required. ECG and defibrillation facilities are available.

Level 3 has trained medical specialist/ Cardiologist. A CCU/ ward with ECG monitors is available. A TMT and echocardiography machine is also available. A catheterization lab is not available.

Level 4 centres are referral centres with ICU facilities and provide state of the art in management of CAD. A cardiac catheterization lab may or may not be available.

In the present scenario most of the PHCs correspond to level 1, CHCs & sub-divisional hospitals to Level 2 , district hospitals to level 3 and medical colleges (with facilities for percutaneous coronary interventions) & tertiary centres to level 4.

Sub-centres should concentrate on primary prevention. Level 1 can follow up diagnosed patients of coronary artery disease. Level 2 has a cardiac defibrillator .Management of acute coronary syndrome and thrombolysis should be done from this level onwards. Special Investigation for risk stratification and management of CAD like TMT and Echocardiogram can be done at level 3. Specialized care & evaluation will be provided at level 4 (Medical colleges/Tertiary centres).

Angiography and angioplasty can be done at those centres where cardiac catheterization lab facility is available.

5. DEFINITIONS AND SPECTRUM OF CORONARY ARTERY DISEASE

Definitions

Coronary artery disease is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium. It typically occurs when there is an imbalance between myocardial oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery or arteries (fig.1) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery

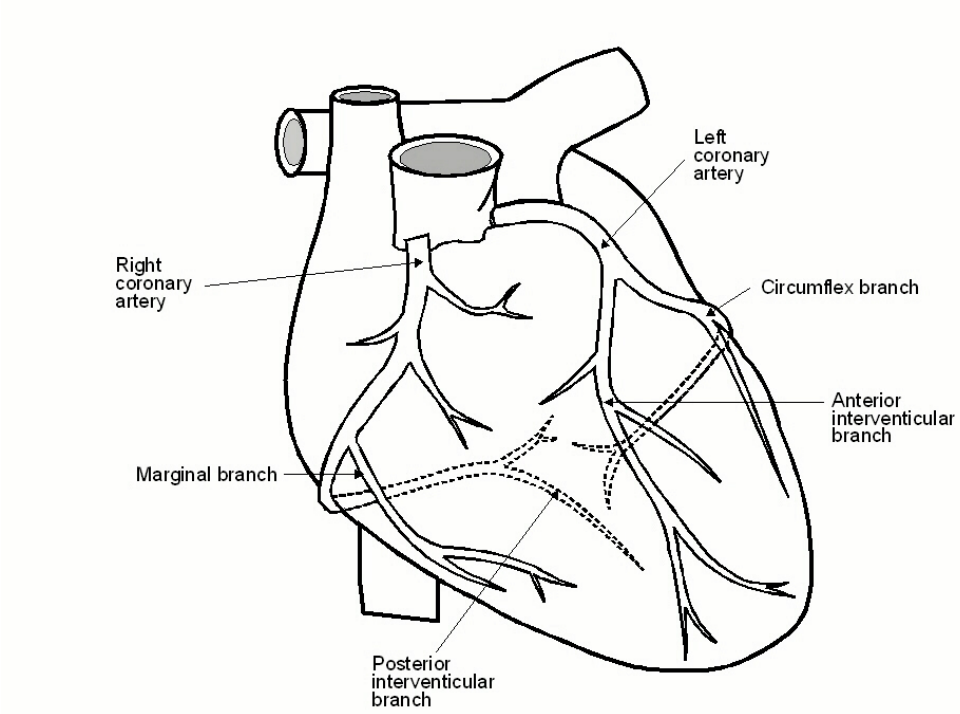


Fig.1: Relation of epicardial coronary arteries and the heart

- **Coronary Artery disease** (CAD) : Fifty percent or more stenosis of epicardial coronary arteries.
- **Acute Coronary Syndrome** (ACS): A spectrum of clinical conditions from unstable angina to ST-elevation MI consequent to myocardial ischemia. Clinically, acute chest pain, typical in character, lasting more than 15 minutes. 'Typical' defined as retrosternal discomfort (heaviness), brought about or increased with exertion and reduced with rest or nitrates. ECG and quantitative/ qualitative measurement of cardiac biomarkers viz Troponins T/I or CPK_{MB} helps to decide about the type of ACS.
- **Unstable Angina** (UA): A clinical syndrome subset of ACS defined by ECG ST-segment depression or prominent T wave inversion and no elevation of cardiac biomarkers of necrosis (Troponins T/I or CPK_{MB}).
- **NSTEMI**: A clinical syndrome subset of ACS defined by ECG ST-segment depression or prominent T wave inversion and/or positive biomarkers of necrosis in the absence of ST-segment elevation.
- **STEMI:** A clinical syndrome subset of ACS characterized by ST-segment elevation or new onset LBBB due to myocardial necrosis.
- **Chronic Stable Angina:** Chronic manifestation of CAD described as retrosternal discomfort (heaviness), brought about or increased with exertion and reduced with rest or nitrates lasting less than 10 minutes.

Spectrum Of coronary artery disease

Coronary artery disease is a dynamic process that involves cyclical transition between partial vessel occlusion to complete vessel occlusion or reperfusion. The clinical spectrums of CAD are shown in the diagram below (fig.2).

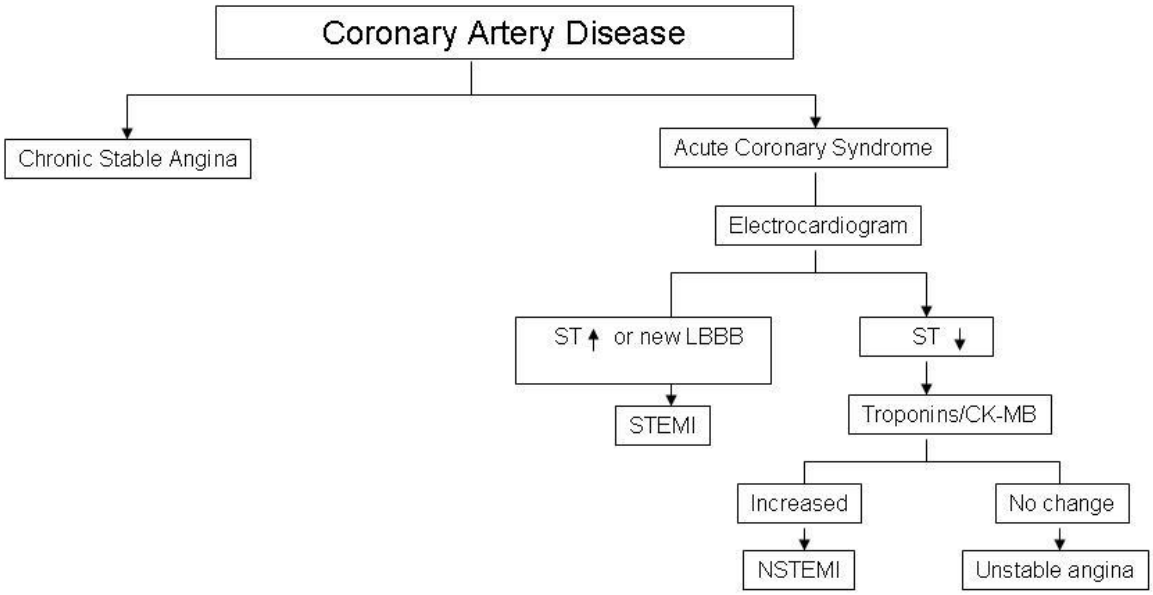


Fig.2: Flowchart showing the spectrum of CAD

Definition of myocardial infarction[3,17,29]

The defining criteria of myocardial infarction are a subject with ongoing changes as a result of scientific advances. In studies of disease prevalence by the World Health Organization (WHO), MI was defined by a combination of two of three characteristics: typical symptoms (i.e., chest discomfort), enzyme rise and a typical ECG pattern. This definition seems to be suitable in context to applicability.

6. RISK FACTORS FOR CAD

Epicardial coronary arteries are the major site of atherosclerosis. The major risk factors for atherosclerosis disturb the normal function of vascular endothelium. Subjects with $\geq$ 2 of the risk factors are at high risk for developing CAD. The commonly recognized risk factors of CAD are as follows:

- **Modifiable**
  - Smoking or tobacco use in any form
  - Dyslipidemia
  - Hypertension
  - Diabetes Mellitus or impaired glucose tolerance (IGT)[30]
  - Obesity Lack of regular physical activity

- Non-modifiable
  - Family history of CAD

- Age (male ≥ 35 years and female ≥ 45 years)[30]
- Genetic factor

CAD risk in Indians

The Risk of coronary artery disease in Indians is 3 to 4 times higher than white Americans, 6 times higher than Chinese and 20 times higher than Japanese[23]. CAD in Indian occurs 5-10 years earlier than other communities. Indians also have higher prevalence of Type 2 DM and IGT, abdominal obesity and dyslipidemia (high triglycerides and low HDL). Increased Apo-B and Apo-A1 levels have been recently identified as significant risk factors[12] but available data are limited.

7. PATHOGENESIS AND PATHOPHYSIOLOGY

Partial or complete epicardial coronary artery occlusion from plaques vulnerable to rupture or erosion is the commonest cause of myocardial infarction, accounting for around 70% of fatal events. This thrombotic process diminishes microcirculatory perfusion by reduced coronary artery flow through epicardial stenoses, as well as by distal embolisation of thrombus (fig.3).

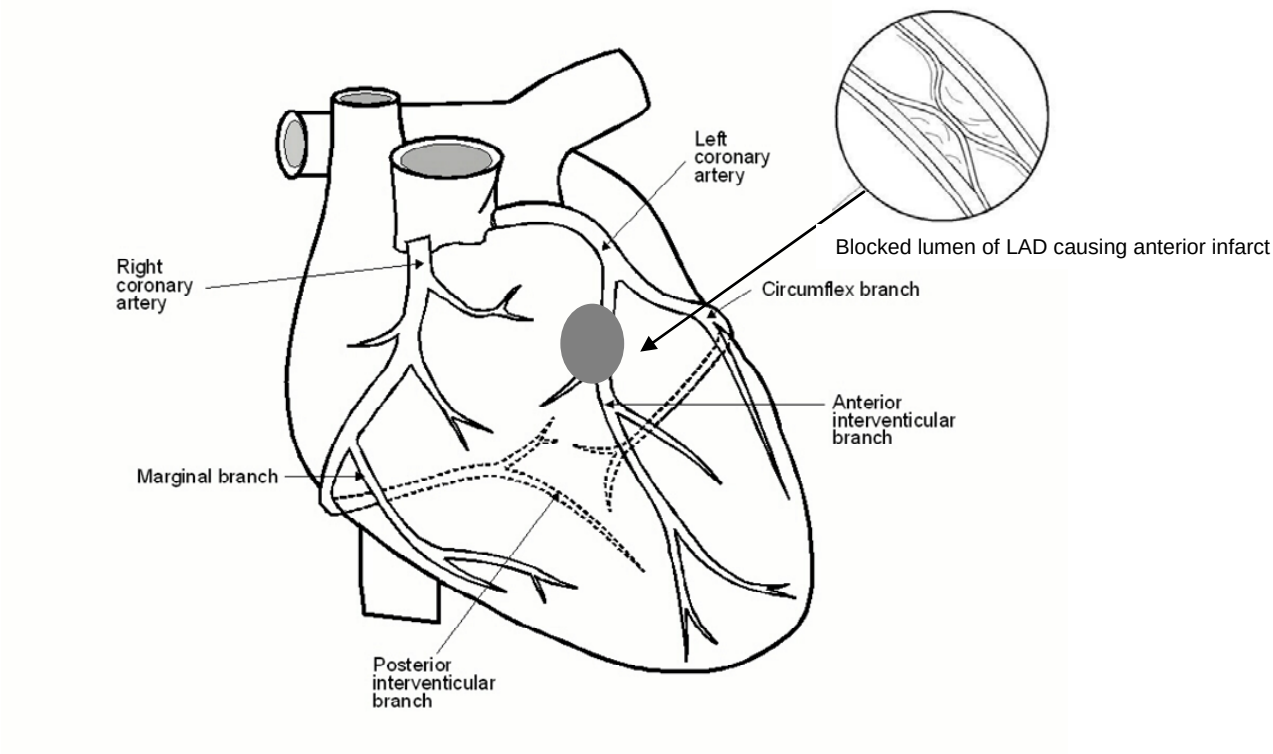


Fig.3: Myocardial infarct consequent to diminished microcirculatory perfusion

This pathophysiology provides the rationale for fibrinolytic and antithrombotic therapies, whereas residual epicardial stenoses are targets for percutaneous and surgical revascularisation approaches.

Vulnerable plaques likely to rupture or erode have evidence of inflammation with monocytes, macrophages, and sometimes T-cell infiltrates, together with thin fibrous caps and large lipid cores. This process involves the entire coronary vasculature, and the true culprit lesion can be difficult to define. Platelet hyper-reactivity and pro-coagulant states also contribute to this thrombotic disease and give rise to the idea of so-called vulnerable blood.

Additionally, coronary spasm, emboli, or dissection of the coronary artery are causes of infarction in the absence of occlusive atherosclerosis, and are reported in 5–10% of patients with STEMI and 10–15% of patients with NSTEMI. In up to half of cases, precipitating factors such as vigorous physical exercise, emotional stress, medical or surgical illness, are implicated in STEMI. Alcoholic binge & use of recreational drugs has been implicated as precipitating factors particularly in young MI (< 40 years) .

Coronary artery occlusion is a dynamic process from deposition of atherosclerotic plaque and partial occlusion to complete artery occlusion (fig 4). 8.

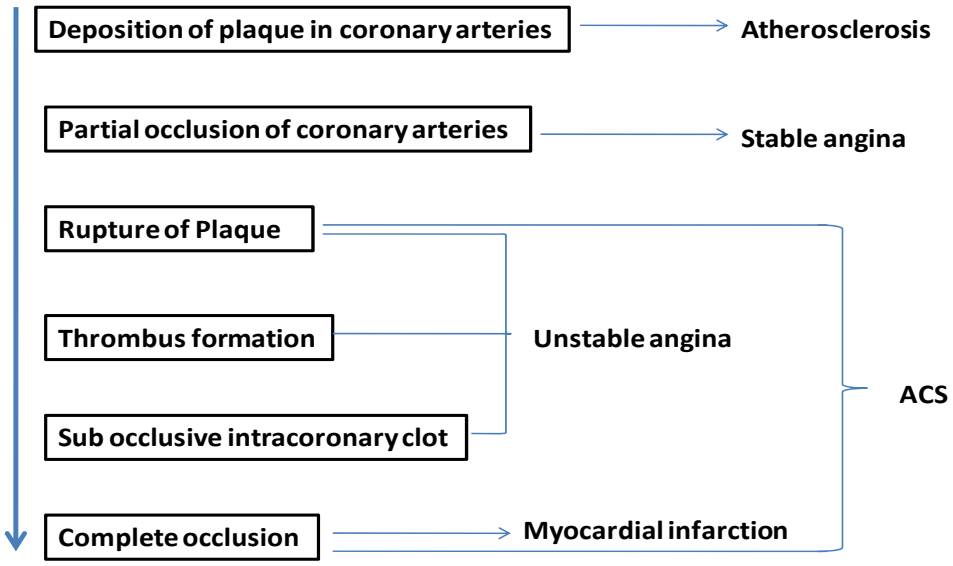


Figure 4. Pathogenic spectrum of coronary artery disease

8. LIKELIHOOD FOR CORONARY ARTERY DISEASE

Likelihood of ACS:

The signs, symptoms, ECG features and cardiac biomarkers which represent ACS secondary to obstructive CAD are mentioned in the table below: (Table 1)

Table1. Likelihood that signs and symptoms represent an ACS secondary to CAD

Features	**High risk ACS (any of below)**	**Low risk (no high/ intermediate, any of below)**
History	Typical angina, history of CAD, age>70 years, male, diabetes	Atypical symptoms
Exam	Extracardiac vascular disease (PAD or Cerebrovascular) Hypotension, transient mitral regurgitation murmur, S3 , S4	Pain reproduced on palpation

ECG	Old Q waves , New transient ST depression (≥1.0 mm), T wave inversion in multiple precordial leads	T wave flattening or inversion < 1 mm with dominant R wave or Normal ECG
Biomarkers	Positive Troponins or CK-MB	Normal

9. CLINICAL MANIFESTATIONS

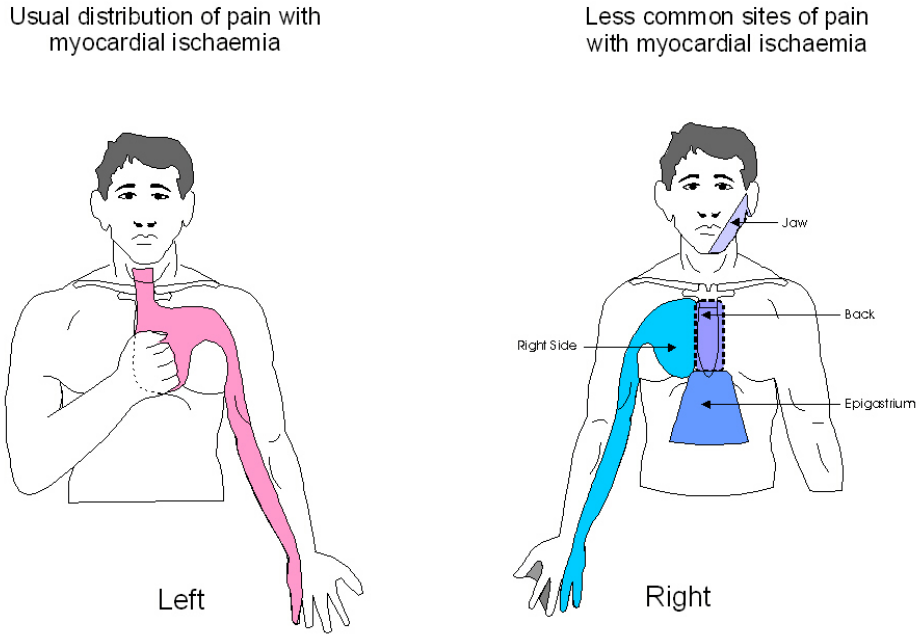


Fig.5: Figures showing the usual and unusual site of pain of angina

Chest pain (angina) is the commonest symptom

- **Typical angina**: Substernal pressure radiating to neck, jaw, arm (Fig. 5) with duration <20-30 minutes which may be associated with dyspnea, diaphoresis, palpitations, nausea-vomiting, or lightheadedness; increases with exertion, decreases with rest or NTG. (**Note**: Rest angina is angina occurring at rest and prolonged, usually greater than 20 minutes; new-onset angina is new onset angina of at least class III severity (Table 2); increasing angina means more frequent, with longer duration or increase by ≥1 class to at least class III severity.

Table2. Grading of angina pectoris according to CCS (Canadian Cardiovascular Society)

classification:

This classification helps in risk stratification of chronic stable angina and deciding the line of management

Class	Symptoms
Class I	• No angina with ordinary physical activity (e.g., walking, climbing stairs) • Angina with strenuous or prolonged exertion
Class II	• Early-onset limitation of ordinary activity (e.g., walking rapidly or walking >2 blocks; climbing stairs rapidly or climbing >1 flight) • angina may be worse after meals, in cold temperatures, or with emotional stress
Class III	• Marked limitation of ordinary activity e.g. walking 1-2 blocks on the level and climbing 1 flight of stairs under normal condition and at a normal pace
Class IV	• Inability to carry out any physical activity without chest discomfort • Angina occurs during rest

- **MI**: Has increased angina intensity and duration >30 min. Twenty five percent of MIs are clinically silent. Proportion of painless STEMIs is greater in patients with diabetes mellitus and increases with age.

Killip Classification:

The Killip classification, published in 1967, categorizes patients with an acute MI based upon the presence or absence of simple physical examination findings that suggest LV dysfunction. The higher the Killip class on presentation, the greater the subsequent mortality.

Class	**Exam findings**
I	No signs of heart failure
II	S3, elevated JVP, rales less than half of posterior lung fields
III	Overt pulmonary edema
IV	Cardiogenic shock

Angina equivalents: Older patients, diabetics, patients with chronic renal failure and female patients are more likely to present with **dyspnea** as their primary symptom. Some patients may have no chest discomfort but present solely with jaw, neck, ear, arm, shoulder, back, or epigastric discomfort or with unexplained dyspnea without discomfort. If these symptoms have a clear relationship to exertion or stress or are relieved promptly with NTG, they should be considered equivalent to angina.

Associated symptoms: Weakness, nausea/vomiting, sweating, apprehension, anxiety, sense of impending doom.

Other presentations, with or without pain

- Sudden-onset breathlessness, loss of consciousness confusional state or sensation of profound weakness
- Rhythm abnormalities or unexplained decrease in arterial pressure
- Evidence of peripheral embolism

Features not characteristics of myocardial ischemia:

- Sharp pain brought by respiratory movement or cough,
- Pain that may be localized by the tip of one finger, particularly over the left ventricular apex or a costochondral junction.
- Very brief episode of pain that lasts a few seconds
- Pain reproduced by movement or palpation over the chest
- Constant pain that lasts for many hours without other ischemic symptoms

Physical examination

- **Focused clinical examinations** for evidence of heart failure, peripheral hypo-perfusion (pallor, diaphoresis, cool extremities), heart murmur, elevated JVP, pulmonary edema should be noted quickly without delaying treatment.
- The presence of severe underlying coronary disease is suggested in patients with clinical **evidence of LV dysfunction, congestive heart failure**
- Pulse rate and blood pressure: Arterial pressure is variable. In most transmural infarctions, systolic pressure decreases by approximately 10–15 mmHg from the preinfarction state.
  - Many patients have normal pulse rate and blood pressure within the first hour of STEMI.
  - Patients with large infarctions have hypotension (systolic blood pressure <100 mmHg and/or sinus tachycardia >100/min)
  - Anterior infarction: About one-fourth of patients have manifestations of sympathetic nervous system hyperactivity (tachycardia and/or hypertension).
  - Inferior infarction: Up to half of patients show evidence of parasympathetic hyperactivity (bradycardia and/or hypotension).
- In right ventricular (RV) infarction, Jugular venous distention is common.
- Look for signs of ventricular dysfunction
  - Third and fourth heart sounds

- Transient mid-systolic or late systolic apical systolic murmur due to dysfunction of the mitral valve apparatus may be present. New, loud (≥Gr 3/6) precordial systolic murmur may be present in ruptured ventricular septum and mitral regurgitation

- Pericardial friction rub in pericarditis (usually develops 24-96 hours after MI)

10. ELECTROCARDIOGRAM IN CAD

- **A 12 lead resting ECG** (± RV3, RV4 for right ventricular MI) should be obtained immediately in patients with ongoing chest pain as rapidly as possible with in 10 minutes of presentation

- A **normal ECG does not exclude** the presence of severe CAD, and should be repeated if strong suspicion in every 4-6 hrs or earlier

- **ECG abnormality** includes:

– Resting **ST segment changes** (depression ≥ 0.5 mm horizontal or downsloping in NSTEACS, convex elevation > 1mm in ≥2 consecutive leads in STEMI, pseudo normalization of ST segment or dynamic changes)

– New pathological **Q-waves** (>0.4 seconds) is considered diagnostic of MI, but may occur with prolonged ischemia

– **T wave**-inversion(≥ 2 mm symmetrical) or a peaked upright T waves may be the first ECG manifestations of Myocardial Ischemia

– Recent onset **LBBB**

– **RVMI** is diagnosed with ST segment elevation in lead V4R, ST elevation in V1 in the presence of ST elevation in inferior leads

– **Non-specific ST and T changes:** ST depression <0.5 mm, T wave inversion <2mm, isoelectric T wave or asymmetric T inversion is less suggestive of myocardial ischemia.

- The **range of normal ST-segment** deviation differs between men and women. ST-elevation (concave upwards) in the V2 or V3 leads of 2·0 mV or less in men and 1·5 mV or less in women, or 1·0 mV or less in other leads, is normal
- ECG **changes that mimic MI** may result from pre-excitation, pericarditis, myocarditis, cardiomyopathy, COPD, pulmonary embolism, cholecystitis, and hyperkalemia; thus the treating physician should be aware.
- Figure 6 shows the evolution of ST changes In MI.

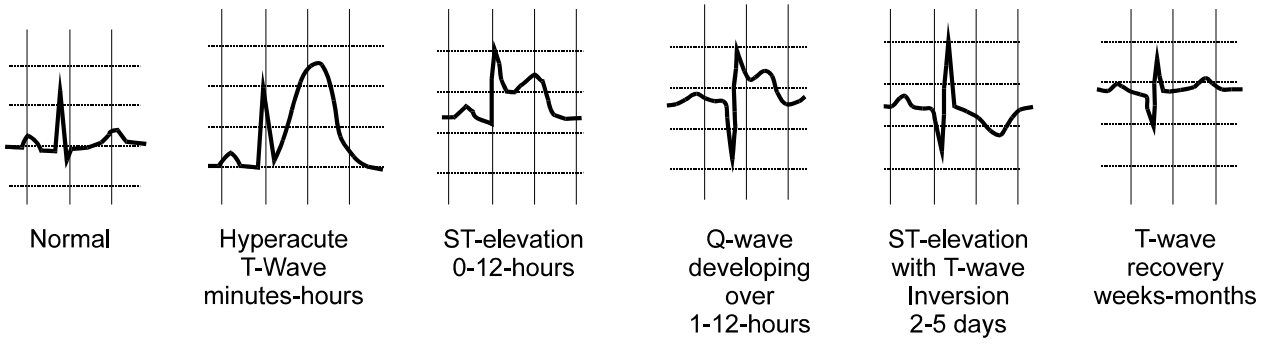


Figure 6. Evolution of ECG changes in Myocardial infarction

The Coronary Circulation:

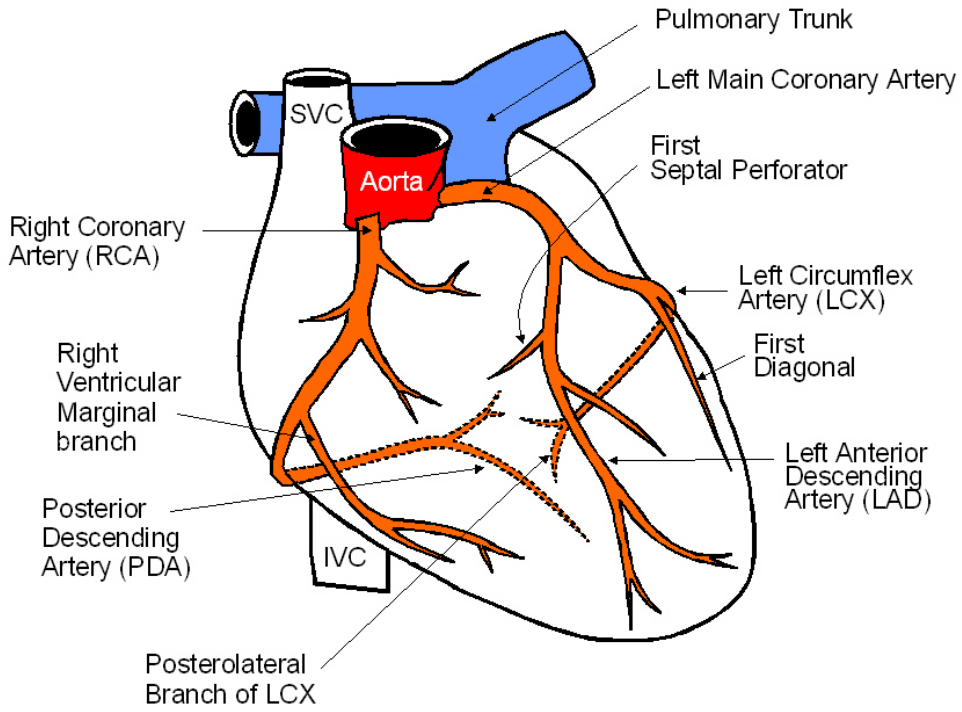


Fig 7: The coronary circulation representing specific arterial territory of the heart

ECG Localization of MI (Table 3): ECG may be helpful to clinically localize the arterial territory (Fig.7) in Acute Myocardial infarction (AMI)

Table 3: ECG localization of AMI[28]

Anatomic area	**ECG leads with ST elevation**	**Coronary artery**
Anterior	V1-V4	LAD
Inferior	II, III, aVF	RCA (85%), LCx (15%)
RV	V1-V2 and V4R	RCA

Posterior	ST depression V1-V2	RCA or LCx

11. LABORATORY STUDIES

- **Blood samples** should be sent for cardiac enzymes (biomarkers Troponin I or T and CK-MB)-for diagnosis of ACS; Hemogram, blood urea, creatinine, electrolytes, FBS -for monitoring and Fasting lipid profile- for secondary prevention. Cardiac specific troponin is the preferred biomarker (Table 4 and Fig 8) for diagnosis of STEMI. Troponin I is not altered in renal failure.
- A portable **chest radiograph** is useful to exclude other causes of acute chest pain but it should not delay the initiation of therapy
- Imaging:
    - 2D echocardiography: Abnormalities of wall motion are almost universally present in STEMI. Estimation of left ventricular (LV) function is useful prognostically. It may identify the presence of right ventricular (RV) infarction, ventricular aneurysm, pericardial effusion, and LV thrombus.
    - Doppler echocardiography: Useful in detection and quantitation of a ventricular septal defect and mitral regurgitation.

Table 4: Time course of serum markers in acute MI

Test	**Onset**	**Peak**	**Duration**
Creatine kinase - total and MB	3-12 hours	18-24 hours	36-48 hours
Troponins	3-12 hours	18-24 hours	Upto 10 days

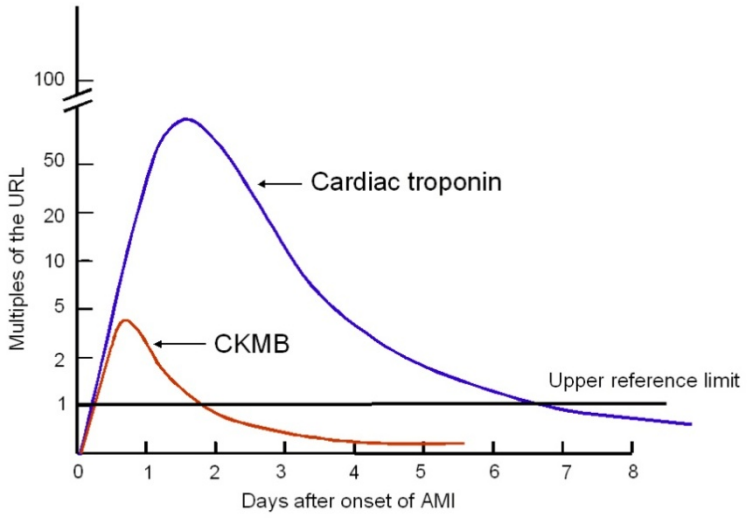
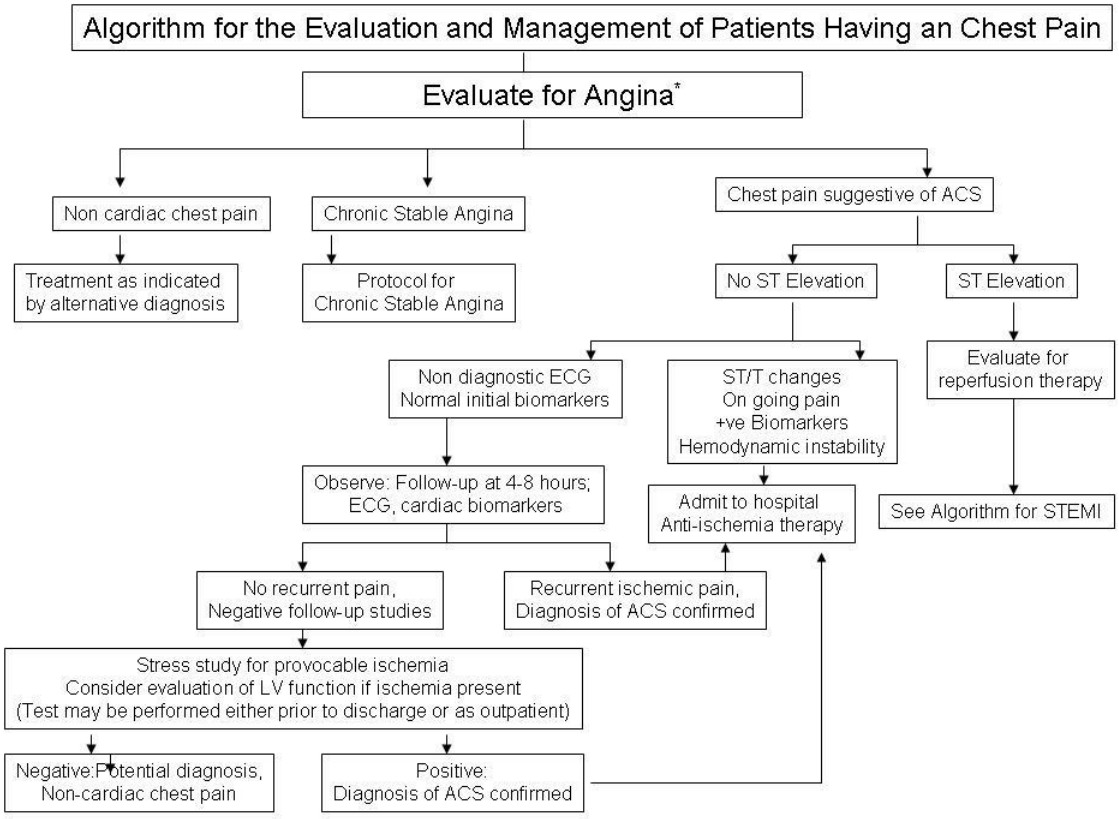


Figure 8: Timing of cardiac biomarkers in acute myocardial infarction

12. ALGORITHM FOR EVALUATION AND MANAGEMENT OF PATIENTS WITH CHEST PAIN

Because symptoms are similar, the differentiation of CSA, UA/NSTEMI and STEMI from that of a non coronary chest pain requires medical evaluation and judgment. The following algorithm is helpful (figure 9)



* History, ECG, stress tests

Figure 9. Algorithm for Evaluation and Management of Patients with Chest Discomfort .

13. CHRONIC STABLE ANGINA: (approach)

A. History: Clinical Classification of Chest Pain

- Typical angina (**definite** if all 3 present)

1. Retrosternal chest discomfort with a characteristic quality and duration that is
2. Provoked by exertion or emotional stress and
3. Relieved by rest or nitroglycerin

- Atypical angina (**probable**)

Meets 2 of the above characteristics

- Non-cardiac chest pain

Meets ≤1 of the typical angina characteristics

B. Initial **Laboratory Tests, ECG, and Chest X-Ray** for diagnosis:

1. Hemoglobin.
2. Fasting glucose.
3. Fasting lipid profile, including total cholesterol, HDL, triglycerides, and calculated LDL cholesterol.
4. Rest electrocardiogram (ECG) in patients without an obvious non-cardiac cause of chest pain.
5. Rest ECG during an episode of chest pain.
6. Chest X-ray in patients with signs or symptoms of congestive heart failure, valvular heart disease, pericardial disease, or aortic dissection/aneurysm.

C. Stress testing (Tread Mill Test or stress thallium) and **coronary angiography for risk stratification** as indicated.

D. **Management:** This includes pharmacotherapy, risk factor reduction and revascularization (if required). The pneumonic **ABCD** for **A**spirin and antianginals (nirates, ACE-inhibitors), **B**-blockers, **C**alcium channel blockers & **D**iet holds good approach for the treatment of CSA patients. **(See below)**

E. Follow up

Treatment Guidelines for patients with Chronic Stable Angina (see management algorithm in figure 10)

• Identify precipitating factors such as anemia, hyperthyroidism, valvular heart disease (e.g., aortic stenosis), tachyarrhythmia, and hypertension.

• Start sublingual nitroglycerin (for sos purpose), oral nitrates, ß-blockers, aspirin, statins and consider ACE inhibitors.

• Start risk factor modification such as statins medication to the ATP III goal of cholesterol <200 mg and LDL cholesterol <100, life style modification inclusing healthy diet, regular exercise & weight reduction.

• Optimize beta blocker dose with check on pulse rate and blood pressure..

• Count the use of sublingual nitroglycerin to monitor the success of treatment.

• Use of nitroglycerin patch at bedtime for nocturnal angina.

• Consider coronary angiography if angina pectoris symptoms are refractory or if the exercise electrocardiogram is abnormal, especially with poor work capacity

Algorithm for management of CSA

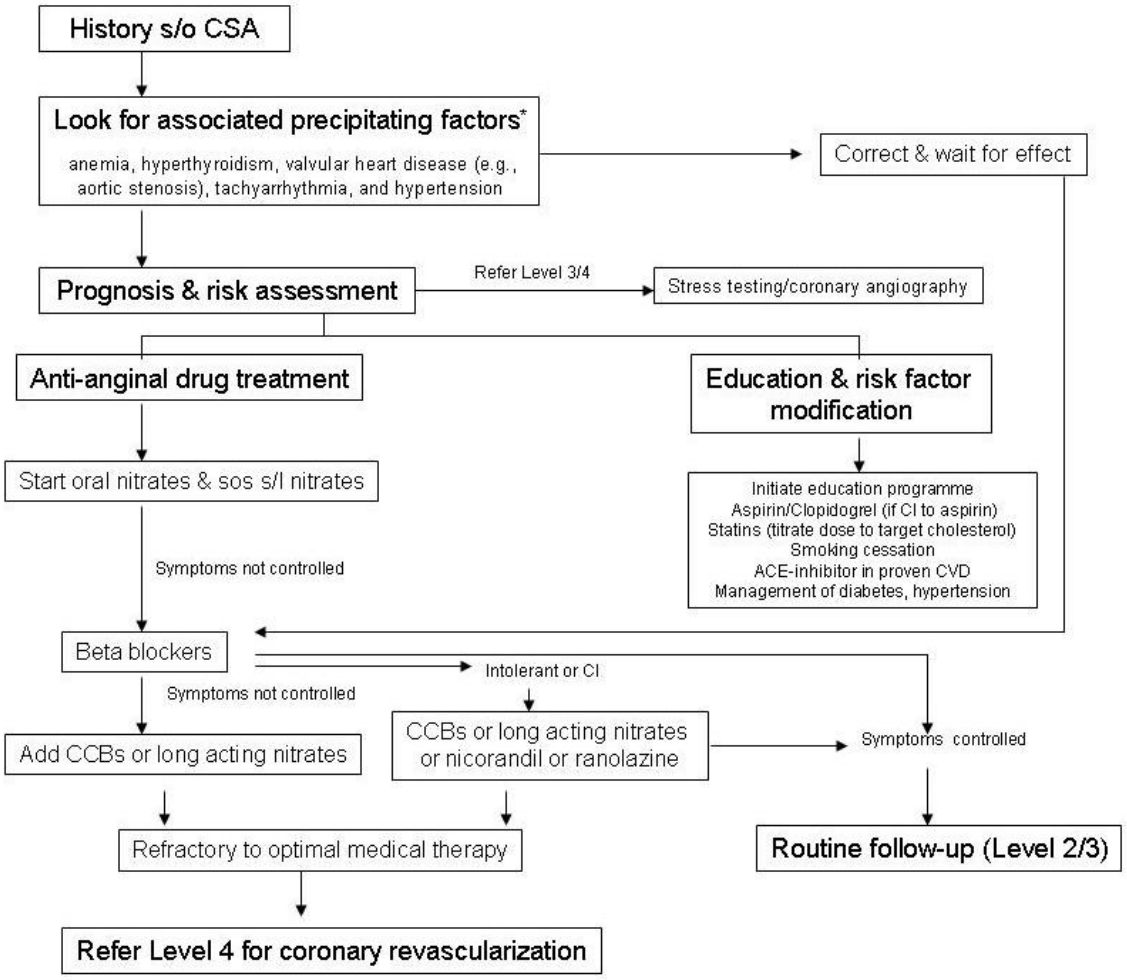


Figure10. Management Algorithm of chronic stable angina

14. MANAGEMENT OF ACUTE CORONARY SYNDROMES: standard of care (Figure 11)

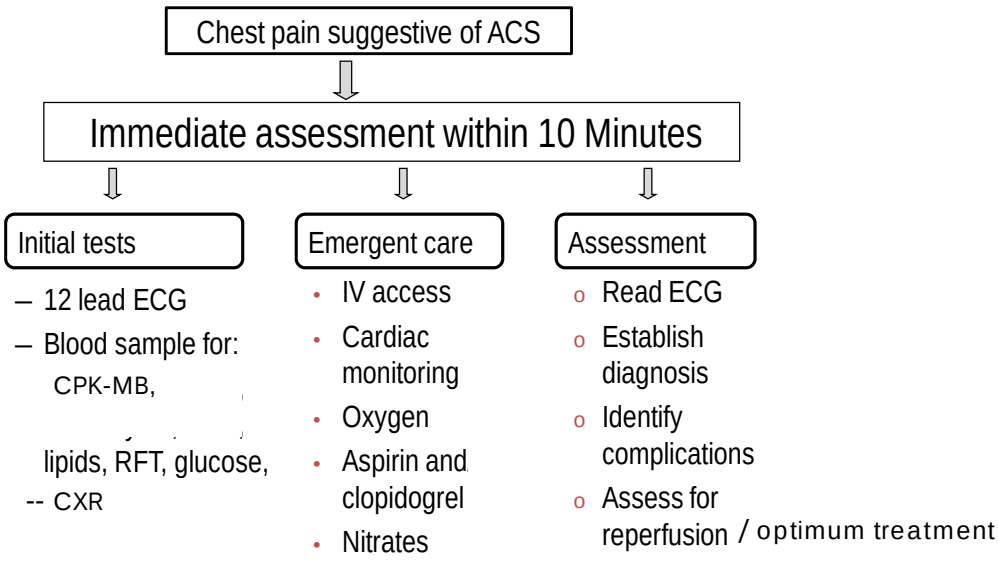


Figure11. Management ACS in emergency department

STEMI cardiac care:

Assessment/ planning the therapy

- Time since onset of symptoms
- Is this high risk STEMI?
  - KILLIP class (≥3)
  - If higher risk: manage with more invasive treatment
- Determine if fibrinolysis candidate
  - Who meets criteria with no contraindications
- Determine if PCI candidate
  - Based on availability and time to balloon treatment

Determine preferred reperfusion strategy

Fibrinolysis is the preferred strategy at Levels 2 & 3 irrespective of time since onset of symptoms

For Level 4:

Fibrinolysis is preferred if:

- Time <3 hours from onset

- PCI not available/delayed
  - door to balloon > 90min
  - door to balloon minus door to needle > 1hr

PCI preferred if:

- PCI available
- Door to balloon < 90min
- Fibrinolysis contraindications
- Late Presentation > 3 hr
- High risk STEMI
- Killip 3 or higher

Fibrinolysis indications

- ST segment elevation >1mm in two contiguous leads
- New LBBB
- Symptoms consistent with ischemia
- Symptom onset less than 12 hrs prior to presentation

Absolute contraindications for fibrinolysis

- Any prior intracranial hemorrhage
- Known structural cerebral vascular lesion (e.g., arterio-venous malformation)
- Known malignant intracranial neoplasm (primary or metastatic)
- Ischemic stroke within 3 months except acute ischemic stroke within 3 hours
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed-head or facial trauma within 3 months

Relative contraindications for fibrinolysis

- History of chronic, severe, poorly controlled hypertension
- Severe uncontrolled hypertension on presentation (SBP >180 or DBP >110 mmHg)
- History of prior ischemic stroke greater than 3 months, dementia, or known intracranial pathology not covered in contraindications
- Traumatic or prolonged (> 10 minutes) CPR or major surgery (< 3 weeks)
- Recent (< 2 to 4 weeks) internal bleeding
- Non-compressible vascular punctures
- For streptokinase / anistreplase: prior exposure (> 5 days ago) or prior allergic reaction to these agents
- Pregnancy
- Active peptic ulcer
- Current use of anticoagulants: the higher the INR, the higher the risk of bleeding
-

Common Thrombolytic Regimens:

The dosages for the current fibrinolytic agents, co-therapy and contraindications are provided in Table 5.

Table 5: The current fibrinolytics in acute myocardial infarction

Drug	**Initial treatment**	**Co-therapy**	**Contraindications**
Streptokinase (STK)	1.5 million units in 100 ml 5%DA or NS over 30-60 minutes	None or iv heparin x 24–48 hours	Prior STK or Anistreplase
Urokinase	2.5 lakhs units iv over 10 minutes followed by 5 lakhs units iv over next 60 minutes. Alternatively given as intracoronary infusion of 6000 unit/min for 2 hour	iv heparin x 24–48 hours	Non antigenic and does not cause hypersensitivity, can be used if STK allergy or prior STK
Tenecteplase *	*Single iv bolus* *30 mg if <60 kg* *35 mg if 60 kg to <70 kg* *40 mg if 70 kg to <80 kg* *45 mg if 80 kg to <90 kg* *50 mg if ≥90 kg*	*iv heparin* x *24–48 hours*	

*** Either of the above can be used depending on availability** (STK is cheaper and is the usual fibrinolytic agent used in our set-up)

Indicators of successful thrombolysis:

Resolution of ST segment elevation by ≥ 50%

Resolution of ischemic discomfort or chest pain or hemodynamic instability

Early peak of biomarkers (12-18 hours) suggests reperfusion

Medical Therapy (To consider as per the available facilities at the setup) **:**

Hospitalize in the critical care unit with continuous ECG monitoring.
Intravenous line for emergency arrhythmia treatment
The pneumonic for medicines used for ACS can be remembered as [MONA + BAH]

- **M**orphine (Analgesia, reduces pain & anxiety, decreases sympathetic tone, systemic vascular resistance and oxygen demand)
    - 2–4 mg IV every 5–10 minutes until pain is relieved or side effects develop side effects. (nausea and vomiting, respiratory depression, hypotension)

- **O**xygen (May limit ischemic myocardial damage by increasing oxygen delivery and reducing ST elevation)
    - 2–4 L/min by nasal cannula to maintain oxygen saturation > 90%
    - Up to 70% of ACS patient demonstrate hypoxemia.
- **N**itroglycerin (Dilates coronary vessels—increase blood flow & reduces systemic vascular resistance and preload)
    - Sublingual: sorbitrate 5-10 mg every 5 minutes, up to 3 doses (If systolic blood pressure > 100 mmHg)
    - Intravenous: Begin at 10 μg/min and titrate upward to a maximum of 100μg/min with monitoring of blood pressure closely.
    - Avoid when there is clinical suspicion of RV infarction.

- **A**spirin (Irreversibly inhibits platelet aggregation, stabilizes plaque and arrests thrombus, reduces mortality in patients with STEMI)
    - Administer aspirin immediately, unless the patient is aspirin intolerant.
    - Dosage: 150-300 mg chewed at presentation, then 150 mg PO OD
    - Be careful with active PUD, hypersensitivity and disorders. If contraindicated, give clopidogrel instead

- **β**-Blocker (Reduces myocardial oxygen consumption, limits infarct size, and reduces mortality. Specially useful in patients with hypertension, tachycardia, or persistent ischemic pain)
    - Oral beta-blocker therapy should be initiated in the first 24 hours (metoprolol, 25-50 mg every 12 hours, titrate dose upto 100 mg every 12 hours based on BP and HR)
    - Contraindications: signs of heart failure , increased risk for cardiogenic shock (age > 70 years, systolic blood pressure < 120 mm Hg, heart rate > 110 or < 60 bpm), systolic blood pressure <100 mmHg, heart rate <60 beats/min, PR interval > 0.24 secs or second- or third-degree heart block, active asthma or COPD.
    - Reassess for therapy as contraindications resolve
- **ACE inhibitors (**Reduces systemic vascular, resistance and cardiac afterload, also reduce aldosterone release with consequent reduction of circulating fluid load and lower cardiac preload, attenuation of the remodelling process after large infarctions, reduces reinfarction & sudden cardiac death)
    - ACE inhibitors should generally be started within the first 24 hours, ideally after fibrinolytic therapy has been completed and blood pressure has stabilized.

    - ACE inhibitor therapy after STEMI should start with low dose oral administration and increase steadily to achieve a full dose within 24 to 48 hours. (Captopril 6.25 mg TID, titrate up to 50mg BD, Ramipril 2.5-5mg BD)
    - May be discontinued at six weeks in low risk patients (without heart failure, diabetes or uncontrolled hypertension, and with small infarctions and relatively preserved left ventricular function). In the higher risk patient,ACE inhibitors can be justified for up to one year.
    - Indefinite treatment: patients with symptomatic heart failure, patients with diabetes, particularly with nephropathy, and hypertensive patients who have

achieved normotensive control on these agents. Use of ACE inhibitors should never preclude treatment with beta blockers in postinfarction patients in whom long term benefit has been well established.

- **H**eparin
    - LMWH (subcutaneous Enoxaparin 1mg/kg BD, Dalteparin 120 unit/kg BD till hospitalization), easy to administer & no need of monitoring. Should be initiated with fibrinolytic agents other than streptokinase. Elective use with streptokinase (after 6 hours of thrombolysis).

Use heparin in combination with aspirin and/or other platelet inhibitor.

Additional medication therapy

- **Clopidogrel** (Irreversible inhibition of platelet aggregation)
    - A 300-mg loading dose (not to be given to elderly > 75 years especially when they have been thrombolysed) followed by a 75-mg/d maintenance dosage is useful for fibrinolysis-enhanced patency
    - Clopidogrel 75 mg per day orally should be added to aspirin in patients with STEMI regardless of whether they undergo reperfusion with fibrinolytic therapy or do not receive reperfusion therapy. Treatment with clopidogrel can be given for atleast 1 year. (Continue clopidogrel maintenance for at least 12 months in patients who have undergone PCI with drug-eluting stents and at least 1 month in patients with bare metal stents).
    - Continue clopidogrel indefinitely in patients intolerant to aspirin.

Additional standard treatment

- Activity**:** Bed rest for first 12 hours**.** In the absence of complications, allow ambulating in room by second to third day. By day 3, increase ambulation progressively.
- Mild sedation and anxiolysis: Alprazolam (0.25-0.5 mg) sos or at bedtime for sleep if required.
- Diet : Nothing by mouth or clear liquids for first 4–12 hours followed by soft diet
- Stool softeners.
- Patients with anterior location of the infarction, severe LV dysfunction, CHF, a history of embolism, 2-dimensional echocardiographic evidence of mural thrombus, or atrial fibrillation should receive full-dose IV heparin (partial thromboplastin time 1.5–2 times control values) **or** Low-molecular-weight heparin (e.g., enoxaparin, 1 mg/kg SC every 12 hours) followed by 3–6 months of warfarin therapy with INR = 2-3 x normal.
- Calcium channel antagonists are not recommended.
- If chest pain or ST elevation persists >90 minutes after fibrinolysis, consider referral for rescue PCI.

- Later coronary angiography after fibrinolysis generally reserved for patients with recurrent angina or positive stress test

- Usual duration of hospitalization is 4–5 days.

- Recommended activity on return home from hospital
    - First 1–2 weeks: Increase activity indoors and outdoors.
    - After 2 weeks: Coordinate level of activity with patient on the basis of exercise tolerance. May resume normal sexual activity
    - Patients after an acute myocardial infarction (MI) without complications such as left ventricular dysfunction or exercise-induced myocardial ischemia may safely resume their previous work: for light office work 2 weeks of sickness absence are recommended, for average manual work 3 weeks, and for strenuous physical work 6 weeks.

Recommended antithrombotic therapy in unstable angina/NSTEMI

Oal antiplatelet therapy

Tab Aspirin, 300 mg (enteric coated) to be chewed stat followed by 150 mg OD

Tab Clopidogrel (alone if Aspirin sensitive or in combination with Aspirin) 300 mg stat followed by 75 mg OD

Heparins

Inj LMWH (Enoxaparin1 mg/kg SC Q12 h for 48 to 72 h or Dalteparin 120 IU/kg SC (max 10,000 IU) Q12 h, until PCI or till hospital admission usually 5-7 days)

Recommendations for early invasive strategy in NSTE ACS/ Unstable angina:

Recurrent angina/ischemia at rest or with low level activities despite intensive anti-ischemic therapy

Homodynamic instability, CHF symptoms, S3 gallop, pulmonary edema, worsening rales, new or worsening mitral regurgitation, sustained VT or elevated Troponin T or I

High risk findings on non-invasive stress testing or depressed LV systolic function (EF<40%)

PCI within previous 6 months or prior CABG

15. MANAGEMENT OF POST MI COMPLICATIONS

Complications of Myocardial Infarction

In-hospital mortality from AMI is primarily caused by circulatory failure from severe LV dysfunction or from one of the complications of MI. These complications may be classified as:

A. **Mechanica**l: Ventricular septal defect, papillary muscle rupture, large ventricular aneurysm, LV pump failure, RV failure, cardiogenic shock

B. **Electrical** or arrythmic :Bradyarrythmias (sinus bradycardia, sinus node dysfunction, AV conduction block), Tachyarrythmias (Supraventricular {Atrial fibrillation, atrial flutter, PSVT}, Ventricular {Idioventricular rhythm, premature ventricular beats, NSVT, sustained VT})

C. **Ischemic**: Infarct extension, Post infarct angina, Inhospital reinfarction

D. **Embolic** (higher with AWMI): Stroke, limb ischemia, renal infarction, intestinal infarction

E. **Pericarditis**: Early pericarditis, Late pericarditis (Dressler's syndrome)

Management

- **LV failure** - Diuresis (Furosemide as per requirement)
  - IV NTG
  - Ionotropes if CHF despite diuresis, use Dopamine, Dobutamine.
  - For cardiogenic shock: Ionotropes, IABP, revascularization (Refer to higher center where invasive facilities & surgical therapy available)
- **Heart block**: Atropine, temporary pacemaker (preferable).
- **Hypotension**: IVF to optimize preload, dobutamine, pacing as necessary, reperfusion, mechanical support.
- **Mechanical complications** (Brackets includes the preferred managements)
  - **Free wall rupture** (Volume resuscitation, ionotropes, pericardiocentesis, surgery)
  - **VSD, Papillary muscle rupture** (Diuresis, vasodilators, IABP, surgery)
  - **LV thrombus** (Anticoagulation for 3-6 months)
  - **Ventricular aneurysm, pseudo-aneurism (**Surgery if recurrent CHF)
  - **Pericarditis** (High dose aspirin, minimize anticoagulation)
  - **Dressler's syndrome** (High dose aspirin)

Arrhythmias during acute phase of STEMI:

The electrical instability during acute phase of ACS are mentioned below in table 6 with preferred management

Table 6: The common arrhythmias during ACS and the management

Arrhythmia	Treatment
VPBs	Monitoring K+ , Mg++, beta blocker
VT	Amiodarone, DC shock
AIVR	Observe unless hemodynamic compromise
Sinus tachycardia	Treat cause; beta blocker
AF/ A. flutter	Treat cause; slow ventricular rate; DC shock (if hemodynamic compromise)
PSVT	Vagal maneuvers; beta blocker, DCshock (if hemodynamic compromise)

Prognosis

- Natural history of MI evolves through following temporal stages
  - Acute (first few hours to 7 days)
  - Healing (7–28 days)
  - Healed (≥29 days)
- The prognosis in STEMI is largely related to the occurrence of complications such as arrhythmias and pump failure.
- Community studies have consistently shown that the overall case fatality rate of patients with presumed myocardial infarction or acute coronary syndrome in the first month is _50%, and of these deaths about half occur within the first 2 h. With the widespread use of coronary interventions, fibrinolytic agents, antithrombotic therapy, and secondary prevention, the overall 1-month mortality has since been reduced to 4–6%, at least in those who participated in the latest randomized large-scale trials and qualified for fibrinolysis and/or coronary interventions.
- Most out-of-hospital deaths are due to sudden development of ventricular fibrillation. Most deaths due to ventricular fibrillation occur within the first 24 hours of the onset of symptoms, and, of these, over half occur in the first hour.
- Survival is markedly reduced in elderly patients (age >75 years).
- Factors associated with increased cardiovascular risk after recovery from STEMI are
  - Persistent ischemia (spontaneous or provoked)
  - Depressed LV ejection fraction (<40%)
  - Rales above the lung bases on physical examination or congestion on chest radiography

16. PRE-DISCHARGE CHECK LIST AND LONG-TERM ACS MANAGEMENT

- Risk stratification:
  - Stress test if anatomy undefined or residual CAD
  - Echocardiogram to assess left ventricular ejection fraction
- Medications (baring contraindications):
  - Antiplatelets (aspirin, clopidogrel for atleast 1 year) , B-blocker, ACEI, Statin, Nitrates, Aldosterone antagonist (LVEF < 40%)
- Risk factors and lifestyle modification
- Patient education before discharge ("teachable moment")

- Cardiac catheterization with coronary angiography is advised for patients at high risk for recurrent MI such as:
  - Angina induced at relatively low workload
  - Large reversible defect on perfusion imaging or a depressed ejection fraction
  - Demonstrable ischemia
  - Symptomatic ventricular arrhythmia provoked by exercise

17. MANAGEMENT BASED ON HEALTH CARE SYSTEM

Chronic stable angina

Level 1

Explain lifestyle modifications to the patient. Stress on counseling and health education Refer to Level 2 for detailed evaluation.

Level 2

A detailed history should be taken including risk factor assessment. Physical examination should be done. Order an ECG. A haemoglobin, blood sugar, serum creatinine and total cholesterol should be obtained. Chest X-ray should be ordered in patients with signs or symptoms of congestive heart failure or suspected valvular heart disease, or aortic dissection/aneurysm.

Aortic Stenosis and Hypertrophic cardiomyopathy (HCM) can also cause angina. An ejection systolic murmur radiating to carotids suggests Aortic Stenosis. Left ventricular hypertrophy in the absence of significant hypertension suggests HCM.

If the history and ECG changes are typical of angina, treatment for CAD should be started. If pain is atypical then refer to level 3 for TMT.

Identify **precipitating factors** such as anemia, hyperthyroidism and severe hypertension. The treatment should include

- Start sublingual nitroglycerin (for sos purpose), oral nitrates, ß-blockers, aspirin, statins and consider ACE inhibitors.
- Start risk factor modification such as statins medication to the ATP III goal of cholesterol <200 mg and LDL cholesterol <100, life style modification inclusing healthy diet, smoking cessation, regular exercise & weight reduction.
- Optimize beta blocker dose with check on pulse rate and blood pressure..
- Count the use of sublingual nitroglycerin to monitor the success of treatment.
- Use of nitroglycerin patch at bedtime for nocturnal angina.
- Refer to level 3 for **TMT** if angina not controlled despite medication for risk stratification and prognostication. Refer for **coronary angiography (Level 4)** if angina pectoris symptoms are refractory, Canadian class III, IV or if the exercise electrocardiogram is abnormal, especially with poor work capacity.

Level 3

Evaluation and management as for level 2. At this level patients can be undertaken for TMT (if available) to prognosticate the symptoms. Patients with intermediate to low Duke scores can be managed on optimal medical treatment and can even be referred to level 2 for follow-up. A detailed evaluation of left ventricular function can be performed with use of echocardiography. Echocardiography can also identify secondary causes for angina like valvular heart diases or hypertrophic cardiomyopathy.

- Refer for **coronary angiography (Level 4)** if angina pectoris symptoms are refractory, Canadian class III, IV or if the exercise electrocardiogram is abnormal, especially with poor work capacity.

Level 4

Level 4 will work as referral centre.

- The risk stratification of the referred stable angina patients will be done at this level if facilities not available at level 3.
- Angiography and revascularization at centre having these facilities.

The patients after definitive management should follow up at the nearest Level 2 or 3 centre.

Acute Coronary Syndromes

Level 1

The recommendations are as below:

- Take History, Prompt ECG (if available), if diagnosis suggestive of ACS:
- Tab **Aspirin** 300 mg (non-enteric coated) stat to be chewed followed by 150 mg OD
- Tab **Clopidogrel** 300 mg stat PO followed by 75 mg OD
- **Nitrates** (Tab sorbitrate 5mg or angised 0.5 mg) sublingual stat and s.o.s not more than 3 times with an interval of 5 minutes each
- R**efer the patient quickly** after above medications to Level 2 or higher depending on the possibility.

Level 2

Reassess history

Evaluate to rule out other causes of acute chest pain, give s/l nitrate

Order an **ECG** on arrival and analyse

If ECG suggestive of ACS:

- Check patient has received Aspirin and clopidogrel, if not loaded as above
- I/v morphine if pain is continuing

- Intravenous nitroglycerine if ongoing pain with LV failure, hypertension.
- Th**rombolyse** with intravenous streptokinase under ECG monitoring if STEMI within window period after checking for contraindications
- Start

  - betablocker (start in small doses, titrate according to BP & HR)

  - ACE inhibitors (initiate in small doses & then titrate)
- **Statins**
- **Anticoagulation** therapy (LMWH)
- **Oxygen** by ventimask or nasal prongs
- ECG monitoring.

- **NSTEMI/USA** :
    - Refer level 3 for further risk stratification if associated with low risk features
    - Refer level 4 for early invasive therapy if associated with high risk features
- **STEMI** :
    - Refer level 3 for further risk stratification if lysis successful
    - Refer level 4 for primary PCI in case of contraindication for lysis or rescue PCI for failed lysis or further invasive management in case of high risk features (LV failure or shock)

Level 3

- **Reassess** history
- **Directed physical examination** (to detect hemodynamic instability, pulmonary congestion, murmurs, limited neurological and vascular examination i.e. pulse and bruit)
- Analyze **ECG**

- Check patient has received aspirin and clopidogrel
- Send **cardiac markers (CPK-MB)**
- Administer oxygen and obtain IV access
- Continuous ECG monitoring and standby defibrillator should be readily available
- Regular monitoring of pulse and blood pressure
- In **STEMI**:
    - Assess window period, if less than 12 hours and there is ongoing pain, consider **thrombolysis** with intravenous streptokinase under ECG monitoring after checking contraindications for thrombolysis
    - **Betablocker, ACE inhibitor, high dose statin,** i/v NTG if ongoing pain with LV failure, hypertension. (Nitrates contraindicated in RVMI)
    - LMW Heparin (**enoxaparin**) 6 hours after thrombolysis followed by 12 hourly for 7 days .
    - Watch for resolution of pain and ST segment (**successful thrombolysis** if pain subsides and /or ST resolution more than 50% within 90 minutes)
    - **Refer to higher centre** for urgent coronary angiography and intervention if failed thrombolysis, post infarct angina, LV failure or shock (if transferrable)
- In **NSTEMI:**
    - LMWH(e.g. enoxaparin 1 mg/kg/dose) should be given stat and every 12 horly for 7 days
    - **Betablocker, ACE inhibitor, high dose statin, i/v NTG according to indications.**
    - **Refer to higher centre** for coronary angiography and intervention if ongoing angina, high risk features.

Level 4

Level 4 are the referral centres.

- Facilities for emergent and experienced PCI
- High risk ACS patients will be managed at this level.

After management of the acute coronary syndrome, the patient can follow up at the nearest Level 2 or 3 centre.

18. SUMMARY OF RECOMMENDATIONS FOR ACS AT DIFFERENT LEVELS OF HEALTH CARE

Levels of care	Level 1 (PHC)	Level 2 (CHCs, sub-divisional hospitals)	Level 3 (District hospital)	Level 4 (Medical colleges with facilities for PCI & Tertiary centers)
History	Chest pain, associated symptoms angina equivalent, Orthopnea, presyncope/syncope	Reassess history	Reassess history	Reassess history
Examination	Pulse, BP, Cardiac auscultation, Chest auscultation	Directed physical examination	Directed physical examination	Directed physical examination
Investigations	ECG	ECG, Cardiac biomarkers (Trop Tor I / CK-MB), Hemogram, FBS, Lipids, Serum electrolytes & Renal function tests	As for Level 2 and TMT & Echo (if available) - Risk startification for CSA & low risk ACS	As for Level 3 and Cardiac catheterization lab & surgica facilities
Management	ECG Aspirin, clopidogrel, s/l nitrate Prompt referral – Level 2 & higher (as per possibility)	Aspirin, clopidogrel, (if not given) s/l nitrate Analgesia-morphine Anti-ischemic therapy (BB, nitrates) ACEI / ARBs if LV dysfuntion Anticoagulant therapy (heparins) as per protocol Statins Thrombolysis for STEMI Refer – Level 3 for further evaluation of low risk ACS Level 4 for Primary PCI in case of CI to thrombolysis or Rescue PCI for failed lysis, Early intervention for high risk ACS Counselling & health education	Treatment protocol as for Level 2 and Thrombolysis for STEMI Refer – Level 4 for Primary PCI in case of CI to thrombolysis or Rescue PCI for failed lysis, Early intervention for high risk ACS Counselling & health education	State of the art management, including, Primary & rescue PCI.

*** At sub-centre: give Tab. Aspirin 300mg stat with prompt referral to Level 1 care**

19. TREATMENT GUIDELINES AT HEALTH SUB-CENTRES

A health sub-centre has one male health worker and one female health worker and covers population norms of 5000.

The recommendations for Sub-centre is **"**Tab Ecospirin 150 mg- 2 tablets stat to be chewed along with prompt and quick referral to PHC or CHC (Level 1 care) whichever is nearer including counseling and health education"

20. TREATMENT GUIDELINES AT PRIMARY HEALTH CENTRE (PHC), Level 1

PHC is a 4-6 bedded hospital which covers population norms of 30,000 and has Medical Officer, Pharmacist, Staff Nurse, Female Health Worker, Health Educator, Health Assistant (M & F). There is provision of limited blood tests, oxygen trolley & facility for ECG.

The recommendation for PHC for ACS is as below:

- Take History, if suggestive of ACS:
- Tab **Aspirin** 300 mg (non-enteric coated) stat to be chewed followed by 150 mg OD to be continued lifelong
- Tab **Clopidogrel** 300 mg stat PO followed by 75 mg OD to be continued 12 months
- **Nitrates** (Tab sorbitrate 5mg or angised 0.5 mg) sublingual stat and s.o.s not more than 3 times with an interval of 5 minutes each
- Every attempt should be made to **obtain an ECG** as quickly as possible for early diagnosis of STEMI.
- Prompt referral to Level 2 or higher level care (as per possibility) for further management.

21. TREATMENT GUIDELINES AT COMMUNITY HEALTH CENTRES (CHC) & SUB-DIVISIONAL HOSPITALS, Level 2

CHC is a 30 bedded hospital covering 1,20,000 population norms, while sub-divisional hospitals have 30-100 beds catering to a population of 5-6 lakhs. Medical staff includes physician, surgeon, obstetrician, pediatrician, anesthetist, staff nurses, dresser, pharmacist/ compounder, ophthalmic assistant, laboratory technician, radiographer and ward boys. They have facilities of ECG, defibrillation, X-ray, ultrasound, blood tests and essential drugs facility.

The management recommendations at CHC & sub-divisional hospitals are as below:

Reassess history

Evaluate to rule out other causes of acute chest pain, give s/l nitrate

Order an **ECG** on arrival and analyse

If ECG suggestive of ACS:

- Check patient has received Aspirin and clopidogrel, if not load as above
- Send **cardiac markers (CPK-MB, Trop T/I)**
- I/v morphine if pain is continuing
- Intravenous nitroglycerine if ongoing pain with LV failure, hypertension.
- Prepare to **thrombolyze** (IV acess) for STEMI or new onset LBBB if within window period
- Start
  - betablocker (start in small doses, titrate according to BP & HR)
  - ACE inhibitors (initiate in small doses & then titrate)
- Statins
- Anticoagulation therapy (LMWH,)
- **Oxygen** by ventimask or nasal prongs
- ECG monitoring.

If no changes, repeat ECG after 30 minutes:

- If changes present manage accordingly (refer above)
- If no changes, reassess patient

If contraindication to lysis, **refer to higher centre (Level 4 care)** for primary PCI.

If failed thrombolysis or ongoing chest pain with non resolution of ECG, refer patient (Level 4 care). Even patient can be referred with ongoing thrombolytic drip if high risk

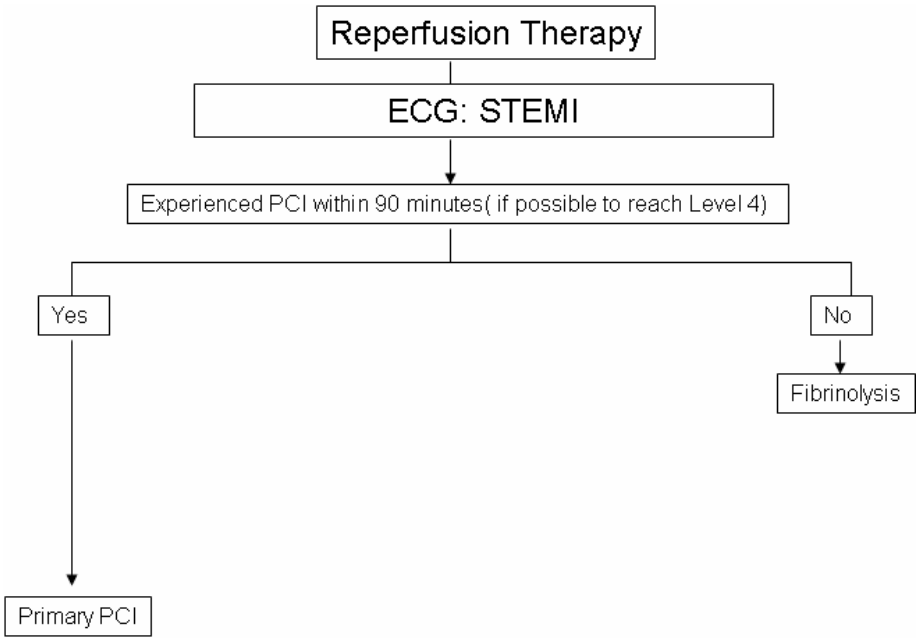
Refer to higher centre (Level 4 care) for early invasive therapy for high risk ACS patients.

Refer to district hospital (Level 3 care) for risk stratification of patients with chronic stable angina & low risk ACS.

22. TREATMENT GUIDELINES AT DISTRICT LEVEL, Level 3

District level hospitals have specialist care for optimum medical management and thrombolysis of ACS. The recommendations are:

- **Reassess** history
- **Directed physical examination** (to detect hemodynamic instability, pulmonary congestion, murmurs, limited neurological and vascular examination i.e. pulse and bruit)
- Analyze **ECG**
- Check patient has received aspirin and clopidogrel
- Send **cardiac markers (CPK-MB, Trop T/I)**
- Administer oxygen and obtain IV access
- Continuous ECG monitoring and standby defibrillator should be readily available
- Regular monitoring of pulse and blood pressure
- In **STEMI**:
    - Assess window period, if less than 12 hours and there is ongoing pain, consider **thrombolysis** under ECG monitoring after checking contraindications for thrombolysis.
    - **Betablocker, ACE inhibitor, high dose statin,** i/v NTG if ongoing pain with LV failure, hypertension. (Nitrates contraindicated in RVMI)
    - LMW Heparin (**enoxaparin**) 6 hours after thrombolysis followed by 12 hourly for 7 days .
    - Watch for resolution of pain and ST segment (**successful thrombolysis** if pain subsides and /or ST resolution more than 50% within 90 minutes)
    - **Refer to higher centre** for urgent coronary angiography and intervention if contraindications to lysis, failed thrombolysis, post infarct angina, LV failure or shock (if transferrable) .
- In **NSTEMI:**
    - LMWH(e.g. enoxaparin 1 mg/kg/dose) should be given stat and every 12 horly for 7 days
    - **Refer to higher cente** for coronary angiography and intervention if ongoing angina, LV failure or shock .



* Markers of successful lysis : decrease in chest pain, ST resolution of 50% or more and the development of a terminal negative T wave in the lead with the highest ST elevation

Figure12. Reperfusion strategies for STEMI: valid for all levels

23. MEDICATION DOSING & ADMINISTRATION

Aspirin
• 300 mg chewed and swallowed (150 mg × 2) upon presentation, then 150 mg daily indefinitely.

Clopidogrel
• 300-mg oral loading dose, then 75 mg PO daily for 9 to 12 mo.

Heparin
• LMWH (Enoxaparin1 mg/kg SC Q12 h or Dalteparin 120 IU/kg SC (max 10,000 IU) Q12h or, until PCI or till hospital admission)

ß-Blockers (should be initiated in first 24 hours if no contraindications in small doses)
• Oral Metoprolol 25-50 mg PO BD.
• Carvedilol 6.25-25mg BD. (if LV dysfunction)
• Patient with early contraindication should be reevaluated for b-blocker therapy for secondary prevention

Nitroglycerin

• 0.4 mg sublingual Q 5 min × 3 for persistent ischemic pain or IV infusion starting at 5-10 μg/min with up titration for persistent ischemic pain. Oral long acting nitrates once/twice daily.

Morphine sulfate

• 2–4 mg IV every 5–10 minutes until pain is relieved or side effects develop

•Side effects: Nausea, vomiting, respiratory depression and hypotension .

Oxygen

• 2–4 L/min by nasal cannula to maintain oxygen saturation > 90%

ACE inhibitors

• Captopril 6.25 mg TID, titrate up as tolerated

- Ramipril 2.5-5mg BD
- ARBs, (Losartan 25-50 mg OD, Valsartan 20-160 mg BD) in patients intolerant to ACE inhibitors with evidence of LV dysfunction.
- **A**ldosterone blockers (spironolactone 25mg OD, eplerenone 25-50 mg OD)
    - Post-STEMI patients who meets the following
        - No significant renal failure (Cr < 2.5 men or 2.0 for women)
        - No hyperkalemia > 5.0
        - LVEF < 40%
        - Symptomatic Congestive heart failure or Diabetes Mellitus

Insulin consider insulin infusion in first 48 hours to normalize blood glucose

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