A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION

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In Partial Fulfillment of the Regulations

for the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

MADRAS MEDICAL COLLEGE, CHENNAI

BONAFIDE CERTIFICATE

This is to certify that **"A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION"** is a bonafide work performed by Dr.NAVEENKUMAR N., post graduate student with registration number **200120100519**, Department of Internal Medicine, MADRAS MEDICALCOLLEGE, Chennai-10, under my guidance and supervision in fulfillment of regulations of the Tamil Nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine).

PROF. Dr.P. MALARVIZHI M.D.,
Guide,
Professor of Internal Medicine,
Madras Medical College,
Chennai – 600 003.

PROF. Dr. C. HARIHARAN M.D.,
Director & Professor,
Institute of Internal Medicine,
Madras Medical College,
Chennai - 600 003.

PROF. Dr. E. THERANIRAJAN M.D., D.C.H.,

The Dean,

Madras Medical College & Rajiv Gandhi Government General Hospital,

Chennai- 600 003.

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PROF. Dr.P. MALARVIZHI M.D.,
Guide,
Professor of Internal Medicine,
Madras Medical College,
Chennai – 600 003.

PROFESSOR
INSTITUTE OF INTERNAL MEDICINE
MMC & RGGGH, CHENNAI-600 003

PROF. Dr. C. [illegible], M.D.,
Director & Professor of Internal Medicine,
Institute of Internal Medicine,
Madras Medical College,
Chennai - 600 003.

Dr. C. THARUHARAN, M.D.,
Reg. No.45626
Director & Professor of Medicine
Rajiv Gandhi Govt. General Hospital,
Madras Medical College, Chennai - 600 003.

PROF. Dr. E. THERANIRAJAN M.D., D.C.H.,
The Dean,
Madras Medical College & Rajiv Gandhi Government General Hospital,
Chennai- 600 003.

DEAN
MADRAS MEDICAL COLLEGE
CHENNAI – 600 003.

DECLARATION

I Dr.NAVEENKUMAR N., post graduate student with registration number **200120100519** solemnly declare that this dissertation "**A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION**" was prepared by me at Rajiv Gandhi Government General Hospital, Chennai, under the guidance and supervision of Prof. Dr.P. MALARVIZHI, M.D., Professor of Internal Medicine, Rajiv Gandhi Government General Hospital, Chennai.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the University regulations for the award of the degree of M.D. Branch I (General Medicine).

Place: Chennai Dr.NAVEENKUMAR N

Date:

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S.NO.	CONTEENTS	PAGE NO.
1	INTRODUCTION	9
2	REVIEW OF LITERATURE	10
3	AIM OF STUDY	73
4	BACKGROUND	75
5	MATERIALS AND METHODS	75
6	DATAANALYSIS	77
7	DISCUSSION	87
8	CONCLUSION	88
9	BIBLIOGRAPHY	89
10	PROFORMA	93

ABSTRACT

BACKGROUND

Fragmented QRS in ECG is indicative of myocardial necrosis or scarring

OBJECTIVES:

- To assess the usefulness of ECG as predictor of LV systolic dysfunction
- To compare the patients who don't have that ECG finding in NSTEACS and estimate it's usefulness.

MATERIALS AND METHODS

Patients with NON ST ELEVATION Myocardial infarction admitted in the intensive coronary care unit are included in the study. The data of each patient is collected in a specifically prepared proforma and includes relevant medical history, ECG findings on admission and serial ECGs, and Ejection fraction assessed by Echocardiogram within 48 hours of onset of symptoms. Patients with fQRS in the ECG were taken as cases and patients without fQRS and Echo is done to assess the Ejection fraction . Using statistical methods ejection fraction between the two groups was analyzed.

Results:

Analysis revealed mean ejection fraction of the cases group is 44.4% and the ejection fraction among the control group is 53.2% . This study revealed that there is a statistically significant correlation between fragmented QRS and ejection fraction

Conclusion:

It is possible to predict left ventricular dysfunction using fQRS. The outcome and mortality of patients with acute myocardial infarction can be predicted using fQRS.

INTRODUCTION

Acute coronary syndrome is now increasing and emerging as a major health problem. Owing to changing lifestyle, increasing prevalence of diabetes, hypertension adverse social habits like alcohol and smoking incidence of MI has increased. Novel therapies have reduced the mortality of MI drastically. Despite new therapies, poor outcome may still occur because Acute Coronary Syndrome is a heterogeneous disease in which outcome is influenced by many causative factors. The extent of myocardial injury and the resultant outcome from ischemia is largely dependent upon the physiological level by the severity and duration of the ischemia.(1)

Notching of QRS is poorly understood and underestimated. Various studies have shown the relation of fragmented QRS and ejection fraction.

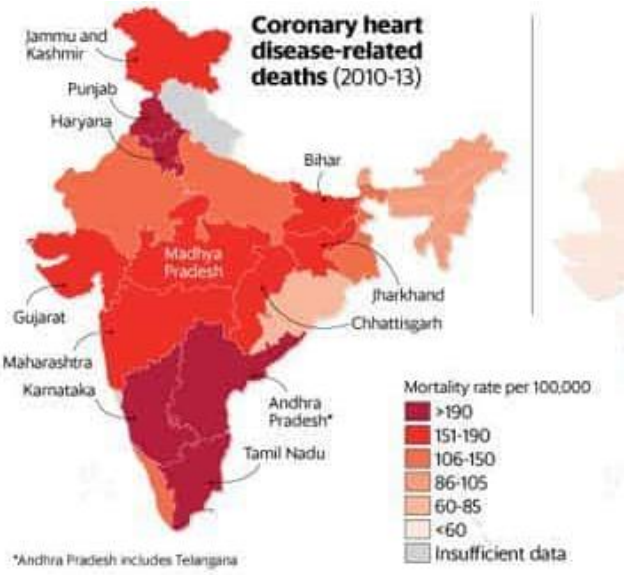
In this study we compare the NON STEACS with fragmented QRS and those without and its correlation with ejection fraction

REVIEW OF LITERATURE:

EPIDEMIOLOGY OF CAD IN INDIA

The main cause of death in India and around the world is coronary artery disease. Although it used to be believed that it was more common in wealthy nations, developing nations like India are currently where more people die from it. When compared to wealthy nations, the rates are rising unreasonably. It has a stronger effect on the socioeconomic development of a country (2).

Patient presenting with anginal type of chest pain, ECG is done initially and look for ST elevation, ST depression, T wave inversion. ST elevation if present is diagnosed as STEMI. When T wave inversion & ST depression present TROPONIN T is done. If troponin is positive it is diagnosed as NSTEMI. When TROPONIN T is negative its UNSTABLE ANGINA. (3)



Types of acute myocardial infarction

Type 1: atherosclerotic plaque rupture causing MI

Type 2: demand supply mismatch

Type 3: sudden cardiac death before the biomarkers are available

Type4: post PCI related MI

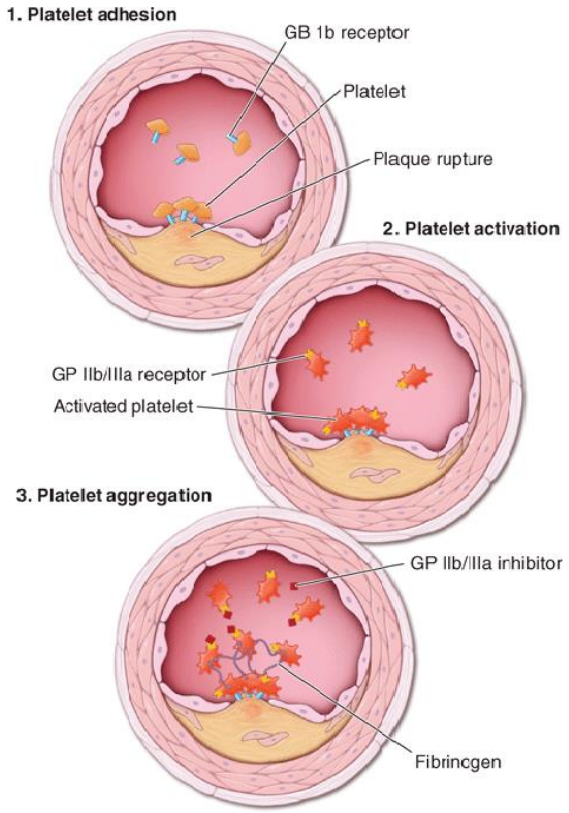
Type 5: post CABG related MI

PATHOPHYSIOLOGY OF ACUTE CORONARY SYNDROMES

The evolution of ACS has been shown to involve two distinct processes

- A fixed, irreversible process involving gradual narrowing of lumen(atherosclerosis)
- A dynamic, reversible process, in which there is slow progression to sudden and rapid coronary occlusion(thrombosis).

FIGURE: PROCESS OF THROMBUS FORMATION INVOLVING PLATELET ADHESION, ACTIVATION AND AGGREGATION



In many situations atherosclerosis predominates in chronic stable angina and thrombosis predominates in ACS(3). Hence the term atherothrombosis is used frequently

ATHEROSCLEROSIS

Atherosclerosis is initially triggered by endothelial dysfunction. Different cytokines, growth factors, hydrolytic enzymes, focal vascular wall necrosis, tissue healing, and fibrosis are all simultaneously induced by endothelial dysfunction or injury.(4) The development of fibro fatty plaque is the following phase. The plaque is made up of a stiff, white fibrous cap covering an elevated lesion with a soft, yellow lipid core (cholesterol and its esters) (4). The underlying media is weakened as a result, which, when it ruptures, results in acute thrombosis of the arterial lumen and blood flow restriction.

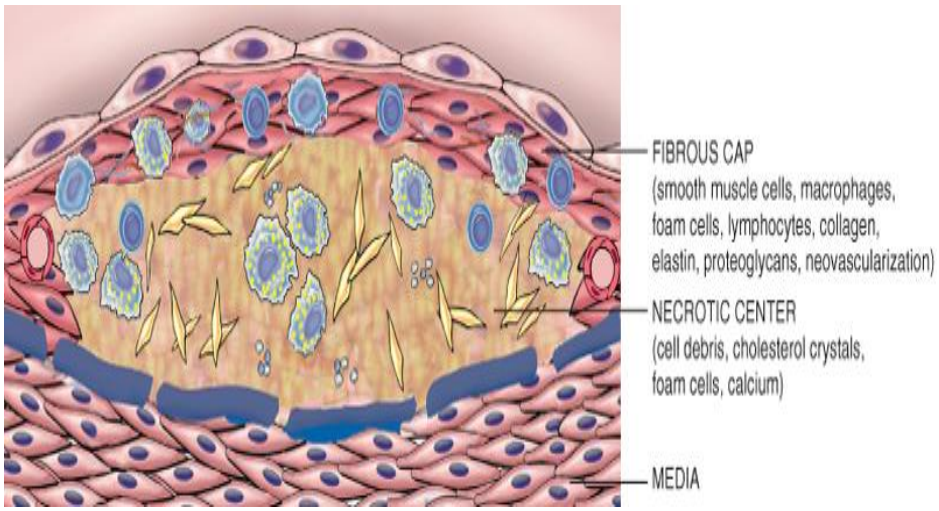
PLAQUE STABILTY:

It is dependent on three factors (5)

A) Mechanical stress acting on fibrous cap

B) Factors causing weakening of the cap

C) Inflammatory triggers.



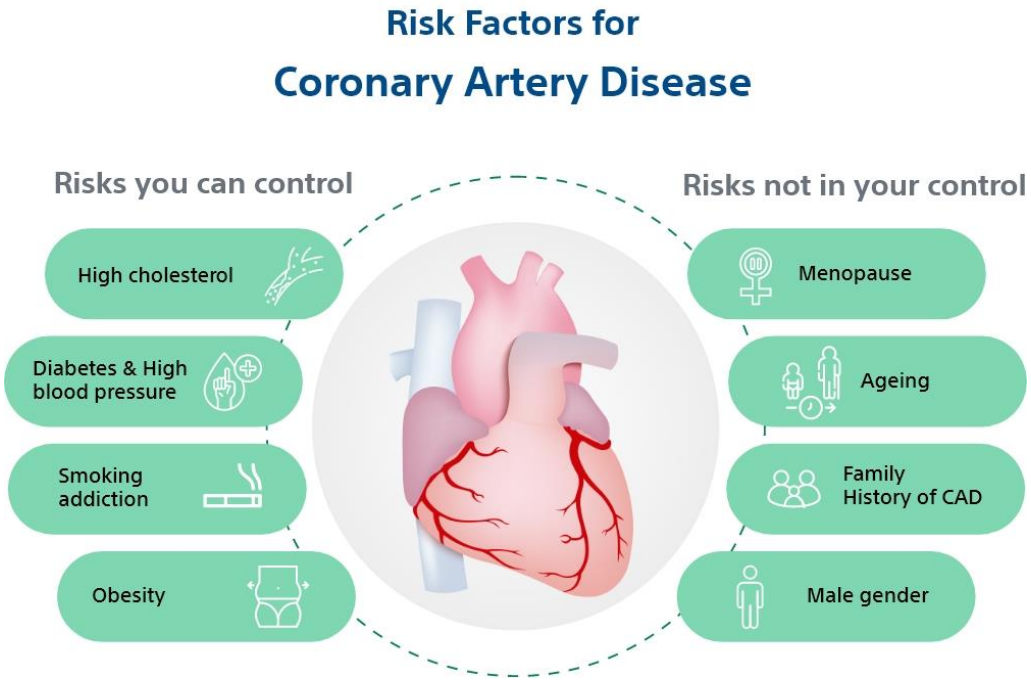
CULPRIT VESSEL PATHOLOGY:

- Plaque rupture – 60 to 70%
- Plaque erosion - 30 to 40%
- Calcified nodule - 5%

Histopathological feature of vulnerable plaque

- Thin cap fibro atheroma
- Thin cap less than 65 micron
- Necrotic lipid core
- Macrophage infiltration
- Decreased smooth muscle content
- Spotty calcification
- Neovascularization of plaque
- Extensive remodeling of segment

RISK FACTORS FOR ATHEROSCLEROSIS



NON MODIFIABLE RISK FACTORS

AGE:

It has a bigger impact on the development of atherosclerosis. Atherosclerotic plaque builds up gradually; clinical manifestation does not occur until it reaches a critical level. Despite the fact that the underlying lesions were developing before to this, the incidence of myocardial infarction in men increases by five times between the ages of 40 and 60.

GENDER:

Women are more likely than men to have atherosclerosis-related disorders after menopause. When compared to men their own age, premenopausal women are more resistant to atherosclerosis and its consequences. This is accounted for by oestrogen's favourable effects on the atherosclerotic process (6). This explains why premenopausal women rarely experience atherosclerotic complications like MI and stroke unless they also have other risk factors like dyslipidemia, systemic hypertension, or diabetes.

GENETIC FACTORS:

It is well knowledge that there is a hereditary predisposition to atherosclerosis and coronary artery disease, which is impacted by a number of factors. In a few cases, it's because the same family has other risk factors like systemic hypertension or diabetes . Others, such familial hypercholesterolemia, include clearly identified hereditary derangements in lipid metabolism.

MODIFIABLE RISK FACTORS

HYPERLIPIDEMIA:

This is a well-known important atherosclerosis risk factor(7). Dyslipidemia alone, even in the absence of additional risk factors, can result in the development of atherosclerosis. Low density lipoprotein (LDL) cholesterol is the main lipid found in blood that is linked to coronary artery disease. Its main job is to get cholesterol to the tissues in the periphery. High-density lipoprotein [HDL], on the other hand, carries cholesterol from atheroma to the liver where it is eliminated into the bile. Therefore, the risk of cardiovascular disease is reduced when serum HDL levels rise.

Diet and medication that increase blood HDL or lower LDL are both of great interest. The amount of animal fat, butter, and eggs consumed increases serum cholesterol levels. On the other hand, plasma cholesterol levels are reduced by diets low in cholesterol and high in polyunsaturated fatty acids (PUFA). Omega-3 fatty acids are advantageous, but unsaturated fats made by hydrogenating polyunsaturated oils have a detrimental effect on cholesterol levels. In contrast to obesity and smoking, which lower HDL levels, aerobic activity and moderate alcohol consumption enhance them.

HYPERTENSION:

Hypertension is another significant risk factor for atherosclerosis; both systolic and diastolic blood pressure levels are crucial. Comparing populations with normal blood pressure to hypertensive patients, the incidence of coronary artery disease rises by about 60%. About half of untreated hypertensive individuals will die from CAD or congestive heart failure, and another third will pass away from the onset of stroke.

CIGARETTE SMOKING

Cigarette smoking is associated with increased risk in both sexes. Daily smoking of more than 1 pack of cigarettes increases the death rate from CAD by 200%. When it is stopped the risk is reduced dramatically.

DIABETES MELLITUS

Diabetes worsens. Atherosclerosis and dyslipidemia are more common. Diabetes doubles a person's risk of suffering an acute myocardial infarction compared to non-diabetics.

ATHEROSCLEROSIS OF CORONARY ARTERIES

Large vessels like the epicardial coronary artery are the principal sites of atherosclerotic activity. Increased plasma low density lipoprotein (LDL), decreased serum high density lipoprotein (HDL), smoking, systemic hypertension, and diabetes mellitus are all associated with atherosclerosis. These factors interfere with the functions of the vascular lumen endothelium, including control of local vessel tone, maintenance of an antithrombotic state over the surface, and regulation of inflammatory cell adhesion and diapedesis.

Pathologic occlusion, thrombus development, aberrant interactions between blood cells (WBC, RBC, PLATLETS), and most significantly between macrophages and platelets and activated luminal endothelial cells, occur when these qualities are compromised. These are the functional adjustments that cause subintimal lipid, intercellular matrix, smooth muscle cell, and fibroblast collections that result in atheroma development.

Where there is more turbulent flow in coronary arteries, such as at branching points in the epicardial arteries, atherosclerotic plaques are more likely to form. When myocardial demand is higher and luminal diameter is lowered to 50% of epicardial artery diameter, the potential to increase coronary flow is constrained. Blood flow may be affected when the diameter is reduced by 80% or more, even

during periods of rest, and a further minor drop in the stenosed vascular orifice area can significantly impair coronary flow and cause myocardial infarction or ischemia at rest or simply with little exertion.

Most often, epicardial coronary artery narrowing due to atherosclerosis is brought on by the development of a plaque, which can cause the cap to rupture or erode, separating the plaque from the bloodstream. Most often, epicardial coronary artery narrowing due to atherosclerosis is brought on by the development of a plaque, which can cause the cap to rupture or erode, separating the plaque from the bloodstream.

The activation and aggregation of platelets as well as the activation of the coagulation process, which leads to the deposition of fibrin strands, are two significant and linked processes that happen when the contents of the plaque are exposed. The thrombus, which mostly consists of platelet aggregates and fibrin strands combined with red blood cells, can restrict coronary blood flow and cause myocardial ischemia's clinical symptoms.

The location of the thrombus impacts the number of myocardial cells that become ischemic and is crucial in deciding how severe the clinical characteristics are (13). Consequently, significant obstructions that reduce the diameter of key coronary vessels, such as those in the left main coronary artery (LMCA) and the

proximal left anterior descending (LAD) coronary artery, are linked to worse outcomes. Collateral blood vessels eventually emerge as a result of chronic, severe constriction of coronary vessels that causes myocardial ischemia, especially when the narrowing happens gradually. When collaterals are fully established, they can sustain myocardial viability and provide adequate blood flow on their own at rest but not during times of elevated demand, such as during exercise.

HISTORY AND CLINICAL PRESENTATION

The most typical symptoms of acute coronary syndrome include pain in the left retrosternal area or discomfort in the epigastric area, which frequently spreads to the neck, left shoulder, right shoulder, and back but more usually to the left shoulder and arm. This sensation is typically very intense, crushing, and may be felt as genuine agony.

Breathlessness and discomfort in the epigastric region are two examples of "anginal analogues" that a patient with CAD may experience in addition to chest pain. Diabetics and women are more likely than men to experience these symptoms (15). The physical examination is frequently not well prominent at all and is very similar to that seen in patients with stable angina. Clinical signs comparable to those seen in patients with ST elevation, such as diaphoresis, pale and cold

peripheries, tachycardia, gallop rhythm, pulmonary crepitation, and shock, can be present in a patient with acute myocardial ischemia or widespread UA/NSTEMI.

When evaluating individuals with suspected STEMI, UA, or NSTEMI, it is important to consider whether of the following three groups the chest discomfort belongs to: high, medium, or low probability.

CLINICAL FEATURES WITH HIGHEST LIKELIHOOD OF CARDIAC CHEST PAIN:

- Chest pain with radiation to right shoulder (8)
- Radiation to both shoulder
- Associated with exertion
- Diaphoresis
- Associated nausea and vomiting

Clinical features with less likelihood of cardiac chest pain

- Chest of pleuritic type
- Positional and sharp pain

Anginal equivalents:

They are clinical feature with which angina is consider clinically. For the following patients angina equivalents are considered(9)

- ✓ Elderly
- ✓ Diabetes
- ✓ Women
- ✓ Post CABG/ transplant

Symptoms of angina equivalents:

- ✓ Dyspnea
- ✓ Diaphoresis
- ✓ Fatigue
- ✓ Atypical chest pain

ELECTROCARDIOGRAPHY:

An easy to use 12 lead ECG can be used to determine whether a MI is present and to predict the outcome. The protocol is to perform a first ECG on the casualty within 10 minutes after arrival1 (10). T-wave alterations in the ECG are specific for myocardial ischemia only when they are fresh, deeper, and of a magnitude 0.3 millivolt. It is advised to monitor the ECG continuously because periodic ECG may not correctly reflect the dynamicity.

STEMI is defined as ST elevation by 1mm in limb and chest leads (contiguous) expect V2 where cut off is 2.5mm for Male <40 yrs, 2mm for male >40 yrs, 1.5mm for woman.

With ST elevation the culprit artery can be localized to LAD (Left anterior descending), LCX (left circumflex), RCA (right coronary artery).(11)

Fig 1

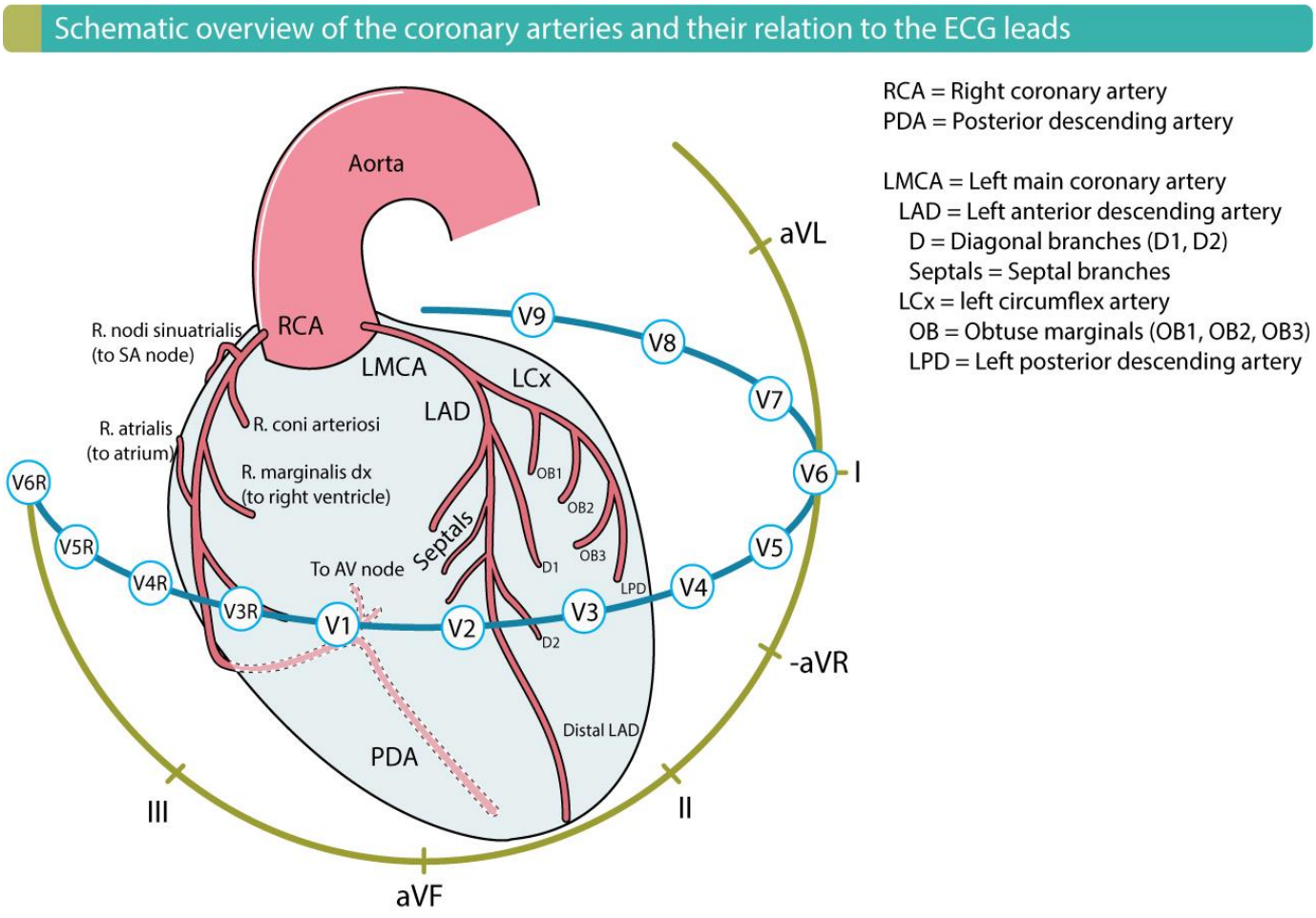
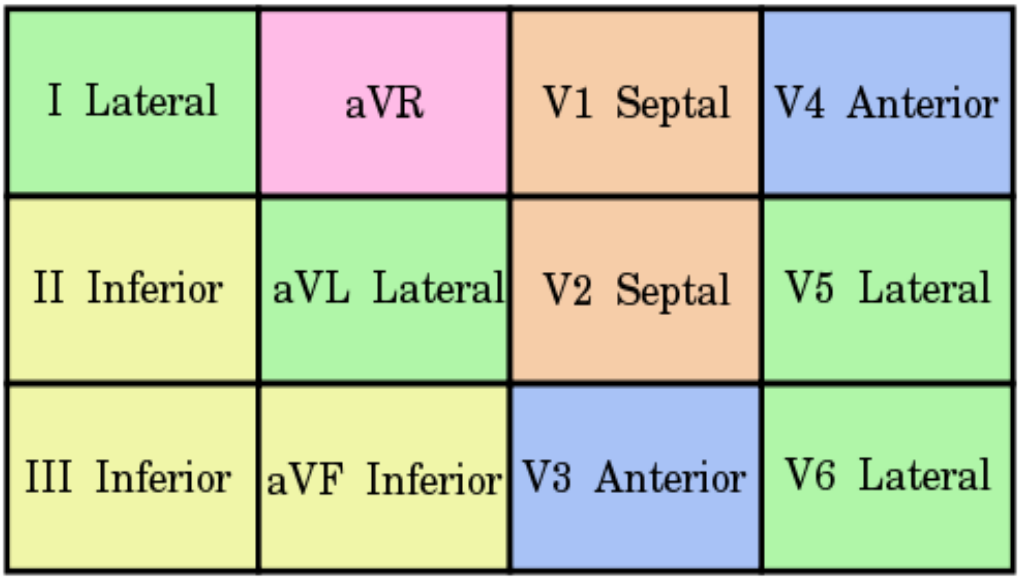


Fig2



Causes of ST elevation:

- MI
- Pericarditis
- Left ventricular hypertrophy
- Brugada syndrome
- Hypercalcemia
- Aneurysms
- Hypothermia
- Cerebral hemorrhage

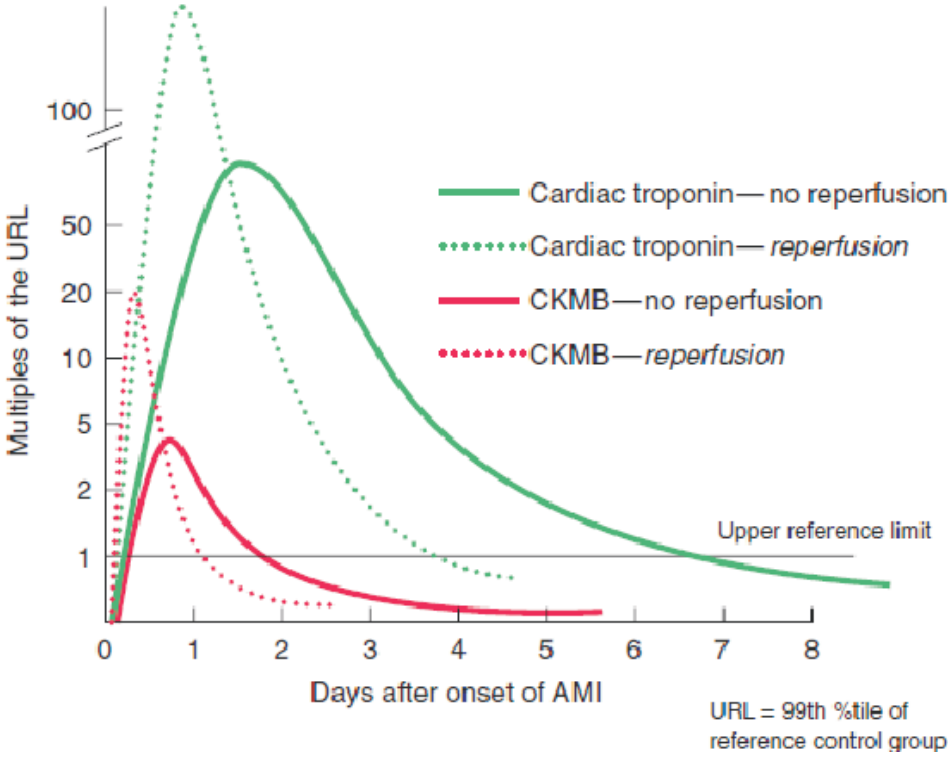
Presence of Q waves point to Old myocardial infarction. Q wave depends on the size of infarction not on depth.

CARDIAC BIOMARKERS:

Cardiac biomarker gets elevated after a cellular injury following an arterial occlusion. Even before the ECG and echocardiographic changes occur the biomarkers gets elevated. The first biomarker to get raised is Myoglobin followed by troponin, CKMB, AST, LDH. Among this the most widely used one is the troponin T. Troponin T exhibits dynamics and it can be used for finding reinfection(12). In case of CKD, Troponin I is used as indicator. Quantitative Troponin is a better tool than qualitative assay.

Troponin will start raising 3-4 hrs after the onset of symptoms. There is no single value that can discriminate between the cause of raise. The acute raise and fall pattern establishes the presence of acute myocardial injury in the form of infarction, myocarditis, stress cardiomyopathy (takotsubo). The ambiguous pattern is defines as troponin values that exceed the 99^{th} percentile upper reference limit and doesn't meet the criteria of acute MI. in ambiguous pattern repeat cardiac troponin at 3 hrs is done, if it raises up acutely it is due to AMI. If it continue to be elevated but without clinical suspicion supports the diagnosis of non ischemic myocardial injury.

Fig 3



Causes of TROP T elevation

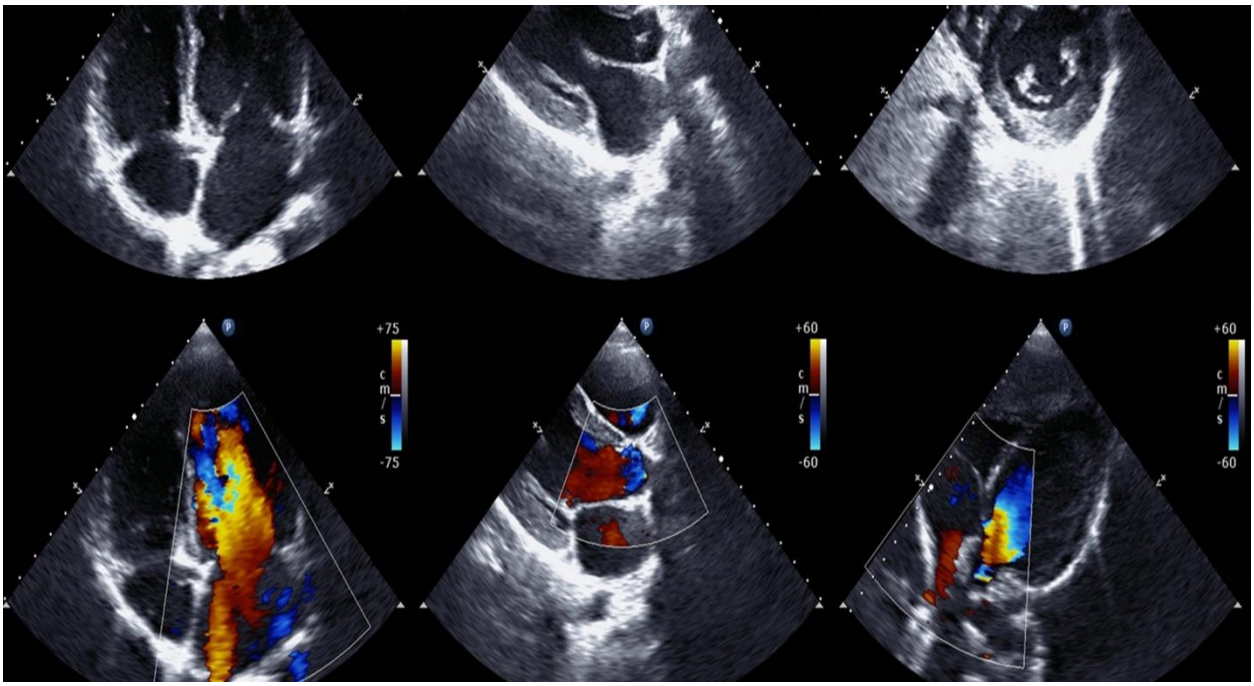
CARDIAC	NON CARDIAC
• Myocardial infarction	• Subarachnoid hemorrhage
• Myocarditis	• Sepsis
• Heart failure	• CKD
• Cardiac abalation	• Pulmonary embolism
• DC shock	• Infective endocarditis
• Cardiac contusion	• Critically ill patients

ECHOCARDIOGRAPHY

It is a non invasive cardiac imaging by which we can assess the structural mobility of myocardium, valve mobility and other complication of myocardial infarction like ventricular septal rupture, Mitral regurgitation, free wall rupture, cardiac tamponade.

Regional wall motion abnormalities are movement abnormalities occurs globally or segmentally in particular arterial territory(13). It cannot distinguish between the onset of even whether it is acute or chronic cause

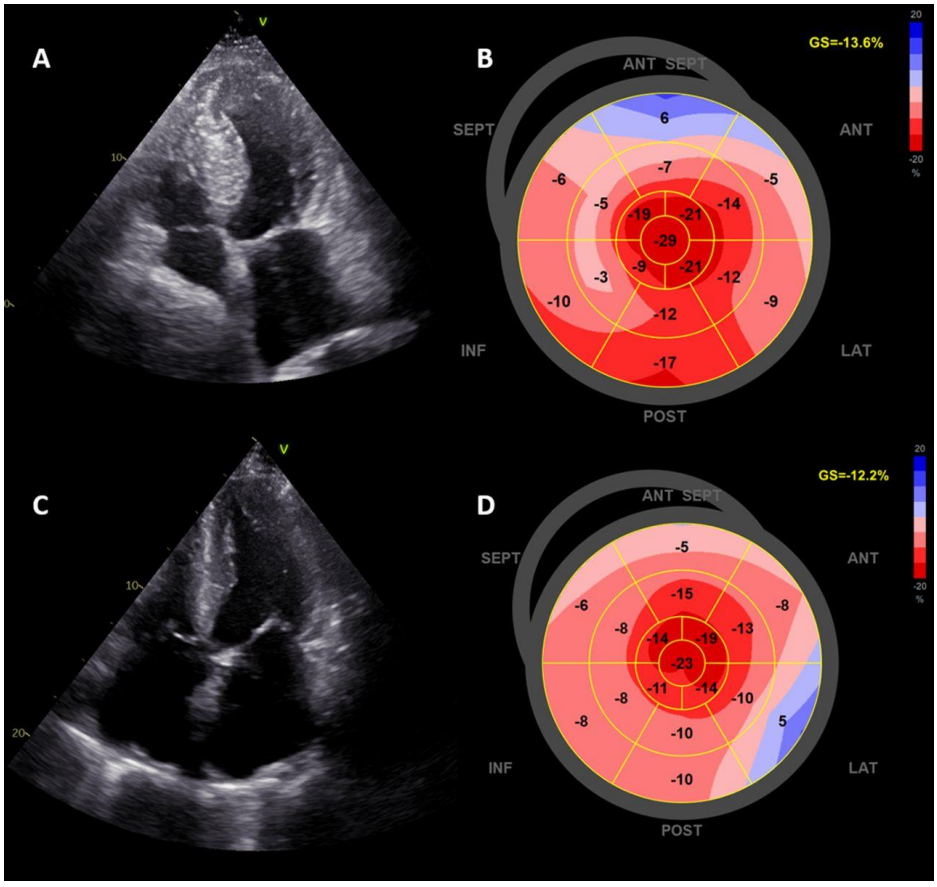
Fig 4



GLOBLA LONGITUDINAL STRAIN:

GLS is a straightforward parameter that calculates the percentage of longitudinal shortening. Compared to LV ejection fraction, left ventricular global longitudinal strain (LV GLS) is a more reliable indicator of LV systolic function. Early detection of LV systolic dysfunction is possible with LV GLS. GLS generally fluctuates with age, sex, and LV loading circumstances, hence it is difficult to define aberrant GLS. While GLS >18% (sic) is acceptable in adults, GLS 16% to 18% is borderline and GLS 16% (sic) is abnormal in adults.(14)

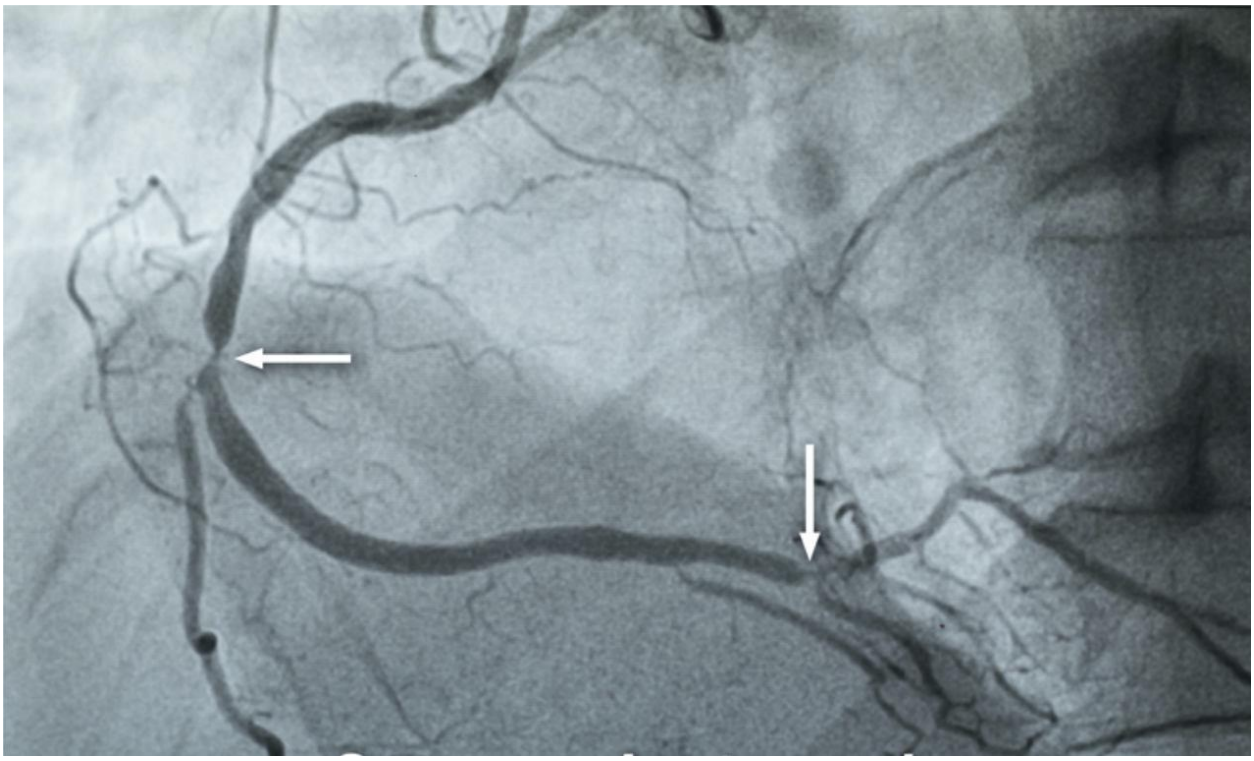
Fig5



ANGIOGRAPHY:

In this imaging modality we study the coronary anatomy which can be doesn't both invasively and non-invasively. CT Coronary angiography is equally effective as invasive angiography. Imaging can be done in less than 10-15 seconds. It can detect the critical occlusions of the artery.

Fig 6



MANAGEMENT:

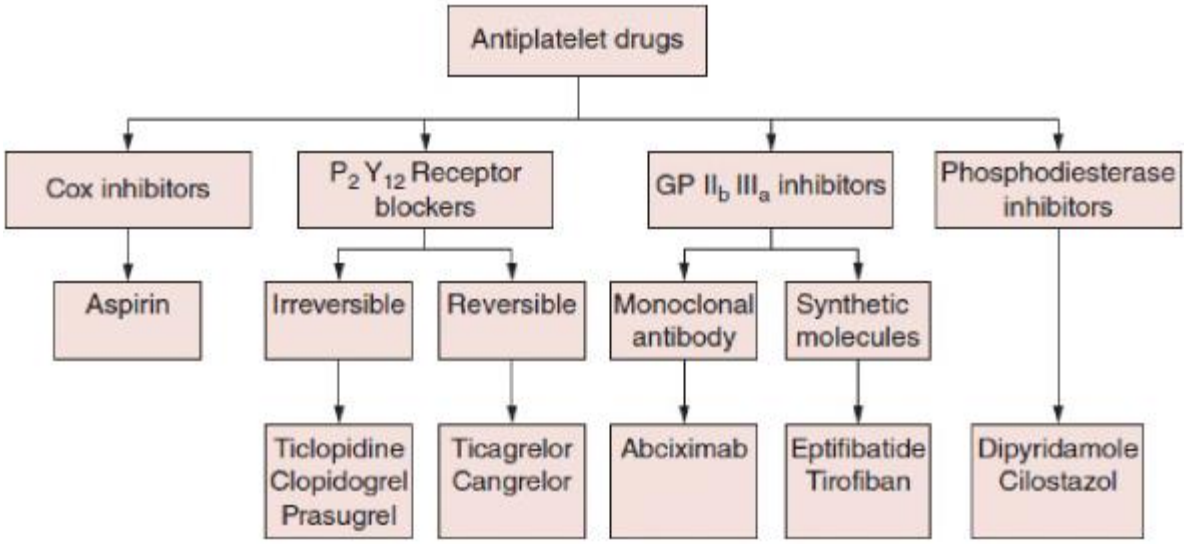
It is important to reopen the occluded the coronaries rather that the mode of reperfusion. Whenever a patient reaches the FMC (First medical contact) which the facility will provide medical or paramedical staff who are able to interpret the ECG,with provision for defibrillatior (15). The from FMC to ECG to diagnosis of STEMI is 10 min.

Once the ECG is done, diagnosis of STEMI or NSTEACS is made. In case of STEMI, the main goal is directed against reperfusion, which can be achieved by PCI or thrombolysis. The initial management in myocardial infarction involves

- Opoids to relieve chest pain
- Oxygen support if Spo2 is <90%
- Loading dose of drugs
    - ❖ Non enteric coated Aspirin 325mg
    - ❖ Clopidogrel 300mg
    - ❖ Atorvastatin 80mg
    - ❖ Sublingual nitrates
- Heparin (UFH/ LMWH)

ORAL ANTIPLATELET THERAPY:

Fig 7



ASPIRIN:

It serves as the primary therapeutic strategy for people with unstable angina/NSTEMI. By blocking the cyclooxygenase enzyme, it prevents thromboxane A2 from being produced again. 150–365 are typical loading dosage ranges. For long-term therapy, low doses (75–150 mg/d) are administered. Five to ten percent of people get "aspirin resistance" . It could be brought on by an insufficient dose, a lack of compliance, poor absorption,

or a quick metabolism. The common side effects associated with it is gastritis.

CLOPIDOGREL AND TICLOPIDINE

Both are thienopyridine group members and ADP antagonists. By prolonging the bleeding process, they lessen blood viscosity. An inert prodrug called clopidogrel is transformed into an active metabolite. When used with aspirin, it has been demonstrated to significantly lower death rates from MI, stroke, and cardiovascular events when compared to aspirin alone. Both patients at low and high risk experience this impact.

An absolute one percent increase in severe bleeding episodes is a drawback of this combination. Clopidogrel is administered orally in loading doses of 600 mg and 75 mg each day. Because it causes less gastric symptoms and it is the preferred agent for long term therapy.

CURE trial involving ASA + clopidogrel vs. ASA showed that dual theraphy for 1 year has significant mortality benefit.

NEWER ANTIPLATELET DRUGS

Recently, newer, more potent antiplatelet agents, chiefly **prasugrel and ticagrelor**, abciximab, eptifibatide, tirofiban have become available.

PRASUGRAL:

it is a thienopyridine drug irreversibly inhibits p2y12 receptor. It is a prodrug and requires 1 step bioactivation via cytochrome p459 enzyme. The loading dose in 60mg and the maintanence dose is 10mg/day and I avoided in prior stroke patients, old age(>75yrs).

TICAGRELOR:

It is non-thienopyridine drug reversibly inhibits P2Y12 receptor. With a 36% bioavailability, ticagrelor is quickly absorbed from the gut and reaches its peak concentration after 1.5 hours. According to the PLATO trial, ticagrelor outperformed clopidogrel in lowering the rate of vascular mortality, MI, and stroke in patients with acute coronary syndromes. In persons with non-ST elevation acute coronary syndrome, ticagrelor was found to have a lower total mortality rate than clopidogrel in a post-hoc subgroup analysis of the PLATO study. However, as it wasn't the PLATO trial's main goal, this finding should only be regarded as

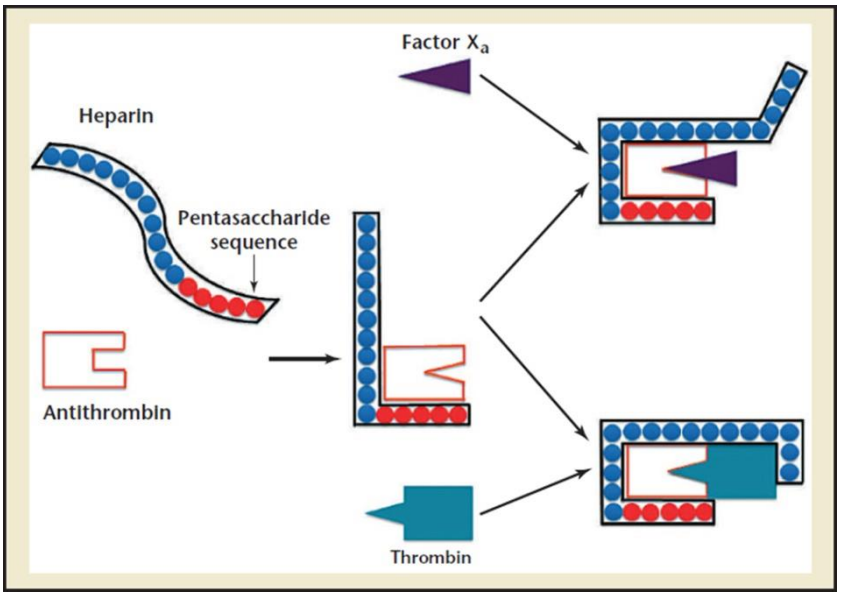
exploratory. The effectiveness of ticagrelor in reducing total mortality has also been questioned in further research.

ANTICOAGULANTS:

UNFRACTIONATED HEPARIN:

It is a time tested drug mainly used in unstable angina. One of the main challenge associated with heparin therapy is target level of anticoagulation. Activated partial thromboplastin (aPTT) is frequently monitored. Heparin induced thrombocytopenia (HIT) is one of the life threatening complication associated. It is due to formation of platelet and heparin complex and results in disseminated intravascular coagulation (DIC).Lepirudin, argotroban and fondaparinux is used in treatment. Protamine sulphate can be used for the reversal of the agent

Fig 8



LOW MOLECULAR WEIGHT HEPARIN

Close monitoring as like with unfractionated heparin is not required in low molecular weight heparin. There is low incidence of HIT and bleeding complications(16).

DOSAGE;

Enoxaparin-1 mg/kg SC q12h

Dalteparin-120iu/kg SC q12h

GFR-10 to 50 ml/min: usual dosage

GFR-<10 ml/min:50 % of usual dosage.

FONDAPARINUX

It is a synthetic pentasaccharide, specific inhibitor of factor Xa. It allows a fixed dosage, simple, once a day regimen of subcutaneous route.

NITRATES:

The mainstay of angina treatment is this. Nitrates are converted to nitric oxide, which causes the capacitance arteries to dilate. This causes a reduction in afterload and a decrease in venous return (preload). Therefore, there is less stress and O2 demand on the myocardium. Nitrates can be given sublingually (0.3-0.6 mg) or by buccal spray to patients who are suffering chest pain. Intravenous NTG

[5-10 microgram/min] should be given if the patient continues to experience pain despite receiving three doses spaced five minutes apart. Every three to five minutes, the rate of the infusion can be increased by 10 micrograms/min until the symptoms go away or the systolic blood pressure falls below 100 mmHg. Typical side effects include headache and hypotension. Hypotension and past usage of sildenafil group medications are the only absolute contraindications to utilizing nitrates.

BETA BLOCKERS

Within 24 hours of admission, oral beta blockers should be started, as per class l guideline. They work by preventing beta adrenergic receptor activity. It lessens sinus nodal rate, AV nodal conduction, and myocardial contractility. It lengthens diastole, lengthening coronary blood flow in the process. Maintaining a target heart rate of 50 to 60 bpm. Sinus bradycardia (50 bpm), any second- or third-degree block, hypotension, and a history of asthma or COPD are all cautions against using it.

CALCIUM CHANNEL BLOCKERS:

They work by reducing myocardial contractility, heart rate, and coronary vasodilation. Drugs like diltiazem and others in the calcium channel blocker class are used in patients who do not experience symptom relief despite receiving the

maximum recommended dose of nitrates and beta blockers, as well as in patients for whom beta blockers are contraindicated due to conditions like asthma. Additionally, it is applied to prinzmetal angina. In severe MI, nifedipine is hazardous.

MANAGEMENT OF STEMI

The critical period in myocardial infarction is within 6hrs. Thrombolytic agent 1986 GISSI trail proved the utility of streptokinase. Subsequently other generation tPA introduced

I st generation	Streptokinase urokinase
II nd generation	Alteplase Duteplase Prourkinase
III rd generation	Reteplase Lanoteplase Tenecteplase staphylokinase

Third generation thrombolytics are fibrin specific.

MI within 0-6 hrs	Fibrinolysis is indicated
6 – 12 hrs	Fibrinolysis decreases mortality by 25% (LATE TRIAL)
12 – 24hrs	Risk of thrombolysis overweighs the benefit of it. Hence PCI is preferred

ABSOLUTE CONTRAINDICATION OF THROMBOLYSIS

- Patients with cerebral or subarachnoid hemorrhage.
- Structural cerebral vascular malformation
- Brain tumors
- Ischemic stroke within 3 months
- Suspected aortic dissection
- Active bleeding or bleeding diathesis excepts menses
- Closed head injury with i3 moths
- Severe uncontrolled hypertension unresponsive to drugs
- Streptokinase usage within 6 moths

Anticoagulation post myocardial infarction is necessary since the lysed clot exposes the raw are which is a potential site for thrombus formation. The duration

of anticoagulation is until revascularization or duration of hospital stay for a maximum of 8 days

SUCCESSFUL THROMBOLYSIS:

After starting the thrombolytic agents a repeat ECG is done after 60 – 90min ST elevation fall by more than 50% suggest a successful thrombolysis (17).

STEMI – PCI

DURATION OF MI	**LEVELOF RECOMEDATION for PCI**
0- **3 HRS**	IA
3- 12 hrs	I a
12- 48hrs	Ilia
Beyond 48hrs	ilia

In PCI stenting is preferred over balloon angioplasty. Drug elution's stent have added advantage over bare metal stent. Radial access is preferred over femoral access(18).

NON CULPRIT ARTERY PCI

- Can be done in stable patient in the same setting or before discharge
- In MI with cardiogenic shock, non culprit artery PCI is not recommended

TYPES OF PCI:

- Primary PCI
- Pharmaco invasive PCI
- Rescue PCI
- Facilitated or systematic PCI

PRIMARY PCI:

PCI is done immediately when the patient presents to hospital. It is also known as primary angioplasty. 95 % of patients treated with primary PCI obtain complete perfusion ,whereas only 50 to 60 % of patients have complete reperfusion when treated by thrombolysis..

PHARMACO INVASIVE PCI:

After a successful thrombolysis, PCI is done after 2 – 24 hrs

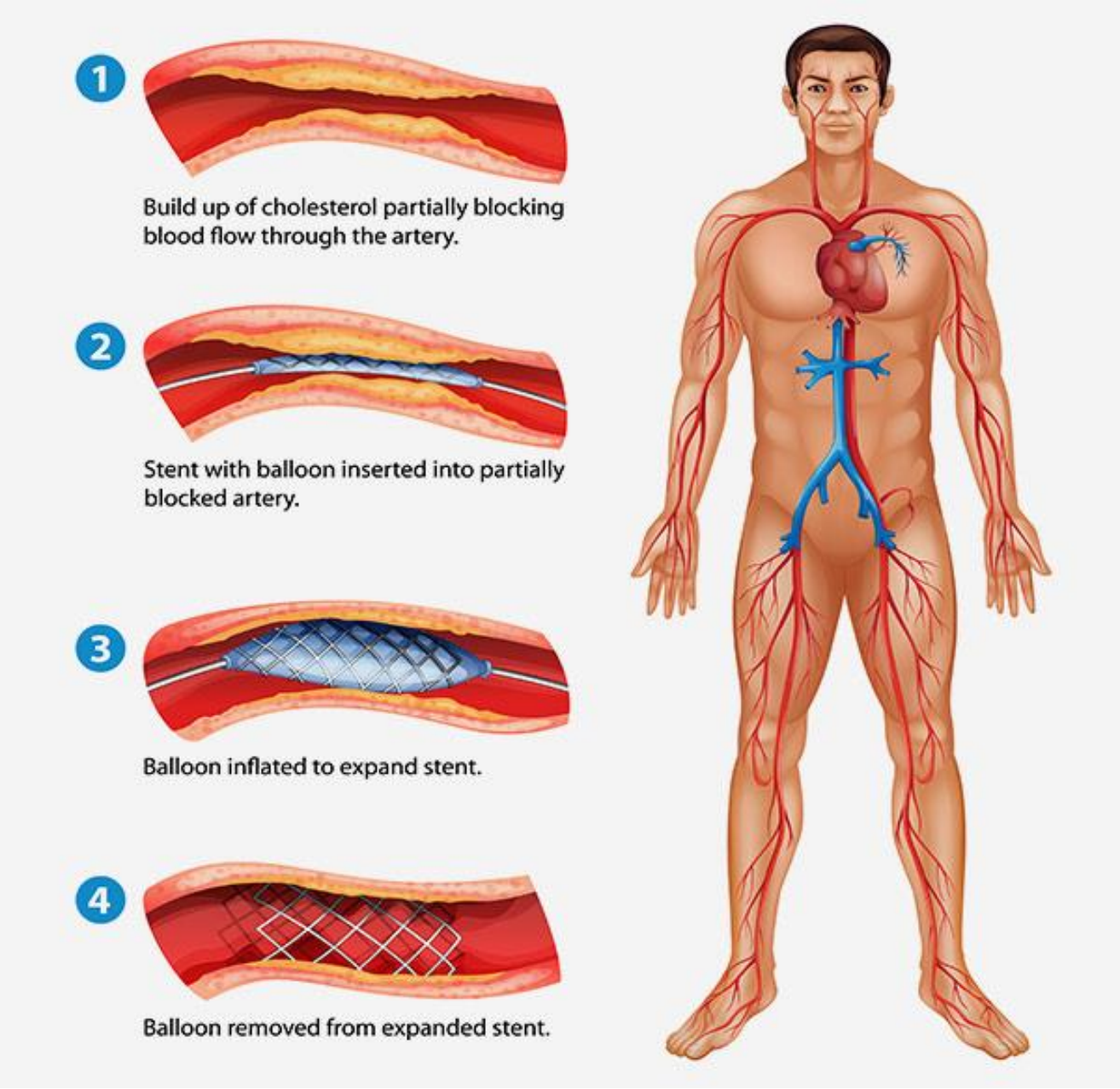
RESCUE PCI

After a failed thrombolysis, PCI is done immediately

FACILITATED PCI

Thrombolysis followed by PCI within 3hr irrespective of fibrinolysis success. It is outdated now because of high bleeding risk.

Fig 9



ASSESMENT OF REPERFUSION AFTER THROMBOLYSIS/ PCI

- Sudden decrease in chest pain
- Achievement of electrical and hemodynamic stability after a period of instability
- ST resolution

- Early t wave in version within 4hrs
- Accelerated idioventricualr rhythm
- Sinus bradycardia with associated hypotension
- Late diastolic VPC
- Disappearance of afib/ BBB

NONSTEACS:

NSTEMI – trop positive

Unstable angina – trop negative

In NSTEMI/ UA initial management involves as described as described above. Apart from the conventional management of oral drugs recent guideline recommends invasive strategy in NONSTEACS

Patient with NSTEMI ARE categorized according to the TIMI/ GRACE score. Based on this score, patient are classified

Very high risk:

- **Hemodynamic instability or cardiogenic shock**
- **Recurrent or ongoing chest discomfort**
- **Life threatening arrhythmia**

- **Mechanical complications of Myocardial infarction**
- **Acute heart failure**
- **Recurrent dynamic st to changes**

High risk:

- **Rise and fall in cardiac troponin compatible with mi**
- **DYNAMIC STT CCHANGES without symptoms**
- **Elevated TIMI**

Intermediate risk:

- **Diabetes mellitus**
- **Renal insufficiency**
- **LVEF <40%**
- **Early post MI angina**
- **Prior PCI**
- **Post CABG**

Low risk:

- **None of the characters above mentioned**

ISCHEMIA-GUIDED STRATEGY VERSUS EARLY INVASIVE STRATEGIES:

The tow treatment strategies have emerged for managing patients with NSEACS. In both strategies patients should receive optimal medical therapy as previously described. On presentation patients who are very high risk such as those

with hemodynamic instability should be referred for an immediate invasive coronary angiogram. At the other extreme patients without recurrent symptom are who are deemed to be a low risk for subsequent ischemic d\events may be managed with an ischemic guided strategy including a noninvasive stress test to assess for inducible ischemia. In particular patients with acute chest pain and a low likelihood of ACS who are troponin negative especially women should preferentially managed with ischemia guided strategy

If any of the follow if occur the patients is considered to have failed initial ischemia guided strategy and should be referred for invasive angiography

- Refractory angina at rest or with minimal exertion despite optimal medical therapy
- Objective evidence of ischemia (dynamic STT changes on ECG demonstrated y noninvasive stress imaging study)
- Very high prognostic risk score

COMPLICATION OF MYOCARDIAL INFARCTION:

- Arrhythmic complications
- Mechanical complications

ARRHYTHMIC COMPLICATIONS:

A small percentage of patients with acute ischemia experience early arrhythmogenesis, which frequently manifests as polymorphic ventricular tachycardia (VT) or ventricular fibrillation (VF) and is frequently correlated with genetic predisposition (20). A cardiac arrhythmia of some kind occurs during or right away after an acute myocardial infarction (AMI) in about 90% of patients. Such rhythm irregularities appear within the first 24 hours in 25% of patients. The risk of life-threatening arrhythmias, like ventricular fibrillation, is highest in the first hour and then gradually decreases in this group of individuals. A non-ST-elevation myocardial infarction has a lower incidence of arrhythmia while an ST-elevation myocardial infarction has a higher rate (NSTEMI).

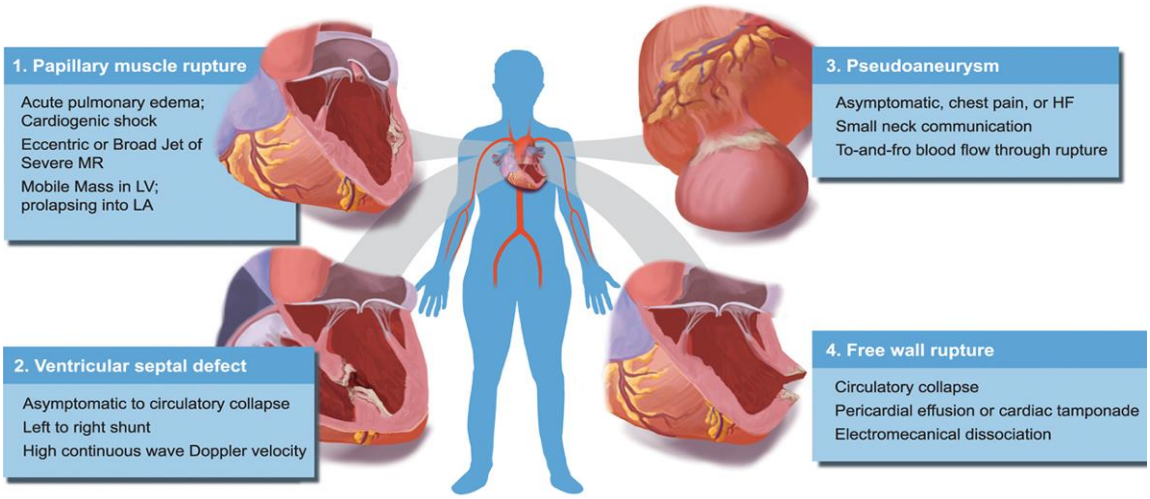
MECHANICAL COMPLICATIONS:

Mechanical complications of acute myocardial infarction (AMI) are ventricular septal defect (VSD), papillary muscle rupture or dysfunction, cardiac free wall rupture, ventricular aneurysm, dynamic left ventricular (LV) outflow tract

(OT) obstruction, and right ventricular (RV) failure(21). All of these conditions could potentially lead to LV failure with cardiogenic shock.

A thorough understanding of the mechanical complications of AMI and their risk factors can help clinicians make an early diagnosis. For favorable patient outcomes, prompt diagnosis with appropriate medical therapy and timely surgical intervention are required. Important factors for in-hospital mortality from mechanical complications of MI include advanced age, cardiogenic shock, and cardiorespiratory failure.

Fig 10



VENTRICULAR SEPTAL RUPTURE:

It occurs 3-5 days after transmural infraction. Symptoms range from isolated murmur to circulatory collapse. Free wall rupture is the most often mechanical

consequence of AMI, its prevalence is unclear because it typically manifests as sudden cardiac death that occurs outside of a hospital and because routine autopsies are not routinely performed. The initial trials of fibrinolysis versus placebo showed an early increased risk of free wall rupture after 24 hours with fibrinolysis therapy, which supports a higher risk of rupture with delayed reperfusion therapy even though the overall incidence of rupture has decreased with prompt acute reperfusion therapy for STEMI. This is a result of myocardial rupture, myocardial dissection, and intramyocardial bleeding.

Any patient who experiences hemodynamic instability or collapse following an AMI should be checked for free wall rupture, especially if reperfusion therapy is delayed or inadequate. In the clinical examination typically reveals jugular venous distension, a pulsus-paradoxus or outright electromechanical disassociation, and muffled heart sounds. The pericardium becomes irritated when in touch with blood, which can occasionally be accompanied by nausea and chest pain.. Free wall rupture usually results in immediate death, but on rare occasions, an early bedside echocardiography confirms the diagnosis and justifies immediate surgical correction. Despite the fact that surgery can save lives, over 35% of people who have undergone surgical repair die in hospitals.

VENTRICULAR SEPTAL DEFECT:

VSD presents in 3-5 days of myocardial infarction. The risk factors associated with it are old age, female sex, late reperfusion. Symptoms are dyspnea, orthopnea, cold peripheries. VSD can occur anteriorly in Anterior myocardial infarction and Posterior VSD is seen in inferior MI. ECHO shows the direction of Blood shunting.

Management involves mainly surgical or device closure, Minor VSD without any hemodynamic compromise doesn't warrant correction. The high mortality rate associated with uncorrected defects approaches 80% at 30 days. Effective afterload reduction to decrease the left-to-right shunt is essential. IABPs with pharmacotherapy are used in >80% of emergency and 65% of urgent repairs. When a patient has multiple organ failure, possible ECMO support may be taken into account to help with end-organ failure and serve as a transitional step toward surgical candidacy.

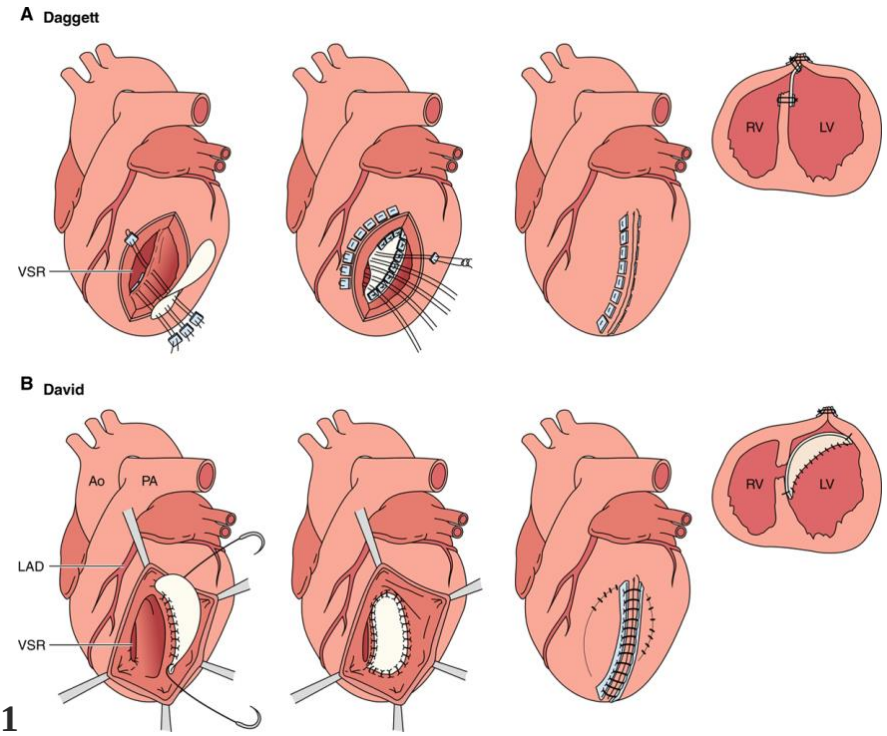


Fig. 11

MITRAL REGURGITATION:

The incidence of acute severe mitral regurgitation (MR) from papillary muscle rupture (PMR) has reduced in the reperfusion era, similar to other mechanical consequences of AMI (range, 0.05%-0.26%). Older age, female sex, a history of heart failure, chronic kidney disease, and a delayed presentation with a first AMI are all risk factors for PMR. In most cases, inferior or lateral STEMIs coexist with posteromedial PMR, albeit anterolateral PMR is incredibly uncommon. The severity of the clinical symptoms may vary depending on whether the PMR is full or partial. Patients presents with pulmonary edema. Examination may reveal Pan systolic murmur. ECHO can show the direction of jet. Acute PMR

is a surgical emergency requiring immediate evaluation by a surgical team. In patients with PMR and obstructive coronary disease, concurrent coronary artery bypass graft (CABG) surgery should be taken into consideration. However, the surgeon must balance the advantages of CABG revascularization against the dangers of extending the procedure.

ANEURETIC VENTRICLES

Acute aneurysms may be severe enough to result in cardiogenic shock and congestive heart failure. During systole, it enlarges. Aneurysms that have been present for longer than six weeks are considered chronic. Normally, they don't enlarge during systole.LV aneurysm is suspected when ST elevation persists for more than 4 weeks. Localized bulge may be shown on a chest x-ray. The preferred inquiry is ECHO. False aneurysms lack the wide neck that true aneurysms have. Myocardial infarction results in wall motion abnormalities and can impair the Left ventricular function and pumping capacity resulting in heart failure.

HEART FAILURE:

HF is a complex clinical syndrome with symptoms and signs that result from any structural or functional impairment of ventricular filling or ejection of blood(22). The diagnostic approach to patients with suspected heart failure involves the assessment of clinical history, physical examination, ECG, Labs and then patients are done Echocardiography. Based on ejection fraction Heart failure is classified in to

- **Heart failure with reduced ejection fraction LVEF ≤40%**
- **Heart failure with midrange ejection fraction LVEF 41%–49%**
- **Heart failure with preserved ejection fraction LVEF ≥50%**

Heart failure symptoms and signs:

Patients with heart failure may complain with vast array of symptoms

The symptoms associated with heart failure are

- Fatigue
- Shortness of breath
- Tachypnea
- Diminished exercise capacity
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Weight gain
- Edema
- Loss of appetite

- Tachycardia
- Tachypnea
- Elevated JVP
- Reales, ronchi, wheeze
- Parasternal lift
- S3,s4
- Tricuspid or mitral regurgitant murmur
- Presacral edema

BIOMARKERS:

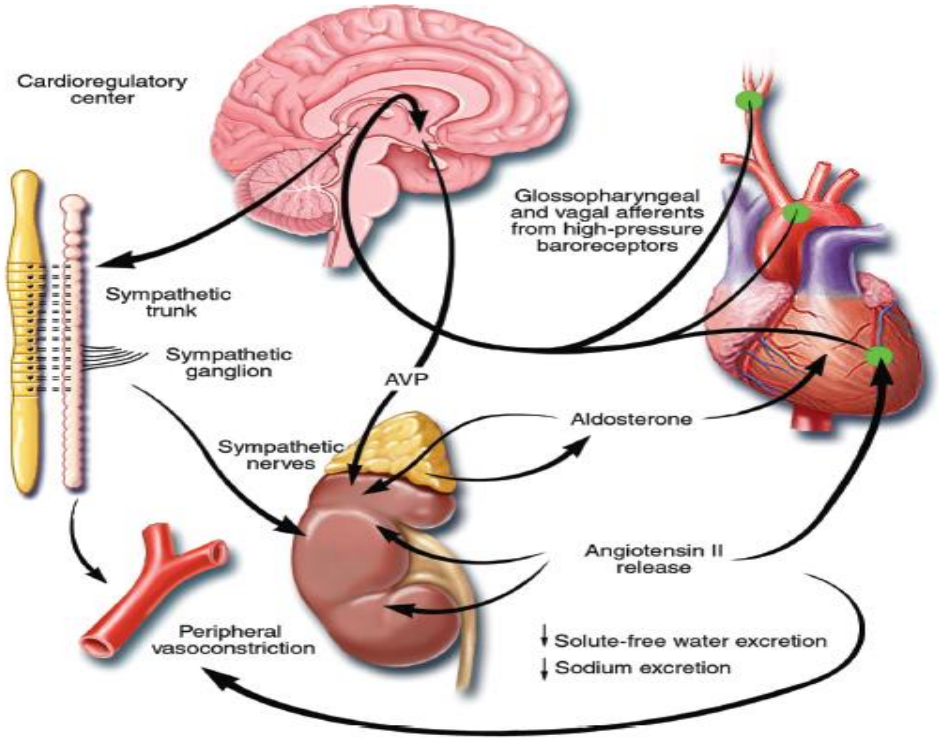
A family of significant counter-regulatory hormones in HF with vasodilatory and other effects is the natriuretic peptides. Clinical practice guidelines now strongly advise the use of both brain (B-type) natriuretic peptide (BNP) and N-terminal pro-BNP (NT-proBNP) in the setting of AHF to aid in the differential diagnosis of patients coming to the emergency department (ED) with dyspnea. The negative predictive value (NPV), or the capacity to rule out HF as the source of dyspnea, is typically greater than the positive predictive value (PPV), for diagnostic natriuretic peptide (NP) testing in the context of AHF (i.e, the ability to definitively identify a diagnosis of HF as the cause of dyspnea).

As with all biomarker tests, it is possible to get false positive results (for example, from a MI or pulmonary embolism) as well as false negative results (mainly from obesity, which lowers NP levels for a specific degree of HF). Despite the fact that patients with HFpEF typically have lower NP levels than those with impaired systolic function, NP testing is unable to accurately differentiate between HFpEF and systolic HF in a given patient. Since elevated levels of cardiac troponin are linked to worse in-hospital and post-discharge outcomes, it has been previously mentioned that patients with AHF typically have elevated cardiac troponin measurements. The percentage of individuals with AHF who have elevated circulating troponin levels has drastically grown as a result of the introduction of

progressively more sensitive troponin assays. Now advised is the measurement of cardiac troponin in AHF patients. Current clinical practice guidelines now propose measuring cardiac troponin in AHF patients since it can help determine prognosis and determine the possibility of concomitant ACS.

PATHOPHYSIOLOGY:

Fig 12



It is a progressive condition that begins as a result of an index event that either destroys the heart muscle, leading to the death of cardiac myocytes that are still functional, or, alternatively, impairs the myocardium's capacity to produce

force, preventing the heart from contracting appropriately. A growing collection of experimental and clinical data points to the possibility that the progression of HF is mediated by the overexpression of physiologically active molecules that can have detrimental effects on the heart and circulation.

ACTIVATION OF SYMPATHETIC NERVOUS SYSTEM:

HF is decreased cardiac output triggers a number of compensatory modifications aimed at preserving cardiovascular homeostasis. The sympathetic nervous system is activated, one of the most significant adaptations. Early on in the development of HF, the sympathetic (adrenergic) nervous system (SNS) is affected. Parasympathetic tone is simultaneously withdrawn when the SNS is activated in HF. In healthy individuals, the central nervous system (CNS) receives inhibitory signals from "high-pressure" carotid sinus and aortic arch baroreceptors and "low-pressure" cardiopulmonary mechanoreceptors that restrict the sympathetic outflow to the heart and peripheral circulation..

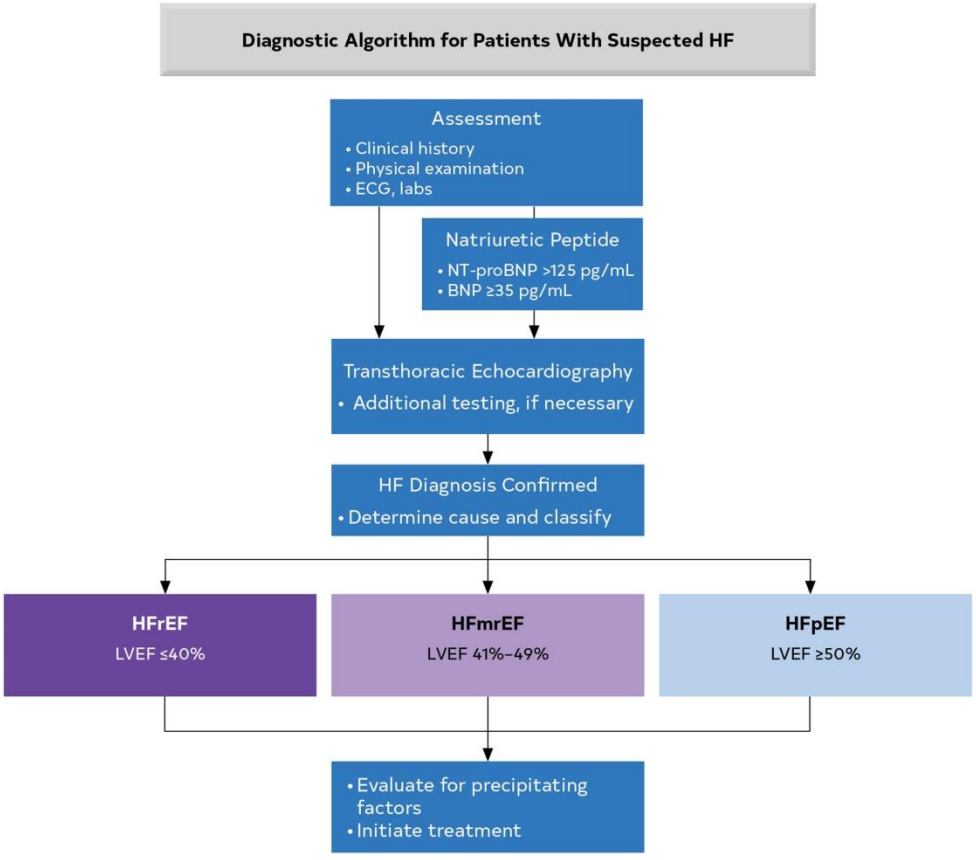
ACTIVATION OF THE RENIN-ANGIOTENSIN SYSTEM:

In HF, the RAS's constituent parts are activated relatively later. Renal hypoperfusion, a reduction in filtered sodium reaching the macula densa in the distal tubule, and enhanced sympathetic stimulation of the kidney, which results in

an increase in renin release from the juxtaglomerular apparatus, are some of the presumed pathways for RAS activation in HF. The AT1 receptor is the most prevalent angiotensin receptor in the vasculature. However, the persistent expression of angiotensin II is maladaptive, resulting in heart, kidney, and other organ fibrosis. According to research, angiotensin 1-7 inhibits LV remodelling and counteracts the effects of angiotensin II.

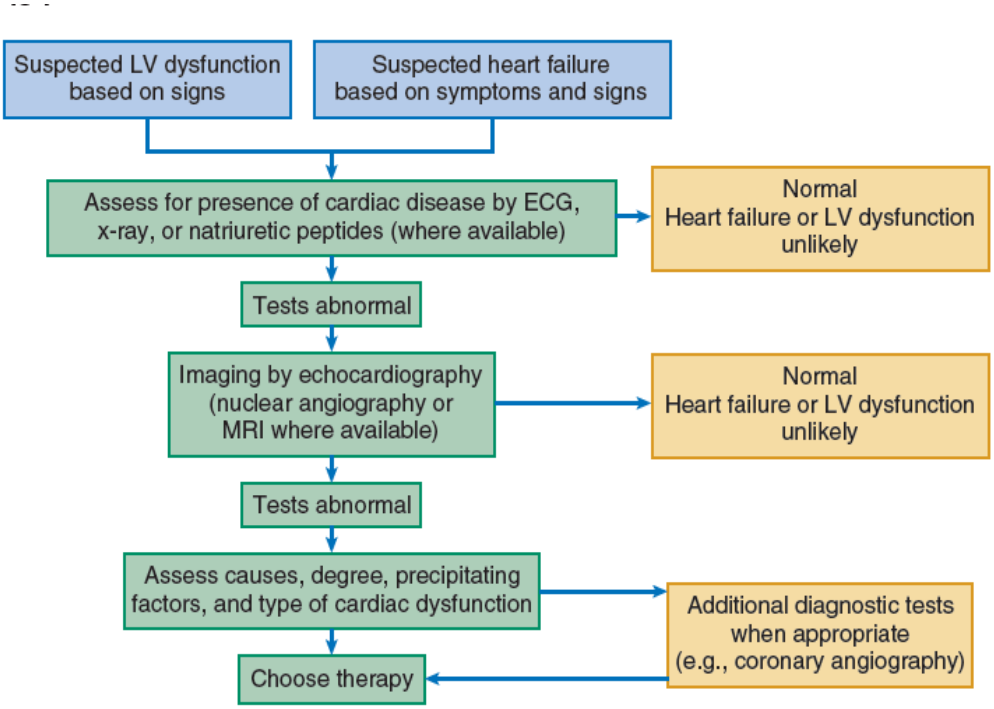
DIAGNOSIS:

Fig13



MANAGEMENT:

Fig14



DIURETICS:

LOOP DIURETICS:

The agents classified as loop diuretics, including *furosemide*, *bumetanide*, and *torsemide*, act by reversibly inhibiting the Na+-K+-2Cl− symporter (co transporter) on the apical membrane of epithelial cells in the thick ascending loop of Henle. Loop diuretics are believed to improve symptoms of congestion by several mechanisms. First, loop diuretics reversibly bind to and reversibly inhibit

the action of the Na+-K+-2Cl− cotransporter, thereby preventing salt transport in the thick ascending loop of Henle Loop diuretics also exhibit several characteristic effects on intracardiac pressure and systemic hemodynamics. Furosemide acts as a venodilator and reduces right atrial and pulmonary capillary wedge pressure (PCWP) within minutes when given intravenously (0.5 to 1.0 mg/kg)

THIAZIDE AND THIAZIDE-LIKE DIURETICS:

The benzothiadiazides, also known as thiazide diuretics, were the initial class of drugs that were synthesized to block the Na+Cl transporter in the cortical portion of the ascending loop of Henle and the distal convoluted tubule**.** *Metolazone,* a quinazoline sulfonamide, is a thiazide-like diuretic that is used in combination with furosemide in patients who become resistant to diuretics

MINERALOCORTICOID RECEPTOR ANTAGONISTS:

Mineralocorticoids such as *aldosterone* cause retention of salt and water and increase the excretion of K+ and H+ by binding to specific mineralocorticoid receptors. *Spironolactone* (first-generation MRA) and *eplerenone* (second-generation MRA) are synthetic mineralocorticoid receptor antagonists (MRAs) that act on the distal nephron to inhibit Na+/K+ exchange at the site of aldosterone action. Spironolactone has antiandrogenic and progesterone-like effects, which may cause gynecomastia or impotence in men and menstrual irregularities in

women. Eplerenone was developed by replacing the 17α-thioacetyl group of spironolactone with a carbomethoxy group. Although spironolactone and eplerenone are both weak diuretics, clinical trials have shown that both have profound effects on CV morbidity and mortality by virtue of their ability to antagonize the deleterious effects of aldosterone in the CV system. These agents are used in HF for their ability to antagonize the RAAS (see later) rather than for their diuretic properties.

SODIUM-GLUCOSE TRANSPORTER-2 INHIBITORS

The sodium-glucose cotransporter-2 (SGLT-2) is a high-capacity, low affinity transporter located in the S1 and S2 segments of the proximal tubule in the kidneys. SGLT-2 accounts for 90%ofglucose reabsorption by the kidney, whereas the lower-capacity higher-affinity sodium-glucose transporter-1 (SGLT -1), located in thes3 segment of the proximal tubules, accounts for the remaining 10%of glucose absorption. Sglt-2 is also responsible for proximal tubular reabsorption of sodium and the passive absorption of chloride that is driven by the resulting electrochemical gradient in the proximal tubule lumen.

The increased absorption of sodium and chloride in the proximal tubule results in lower chloride concentration delivered to the distal tubule, which in turn results in dilation of the afferent arteriole and increase glomerular filtration through tubule glomerular of sodium and glucose uptake in the proximal tubule of the

kidney. This leads to increased concentration of chloride in the distal tubule, and a resetting of the tubulo-glomerular feedback mechanism, the results in a contraction of the plasma volume without activation of the sympathetic nervous system. Agents in the sglt-2 class of inhibitors include *canagliflozin, dapagliflozin,* and *empagliflozin.*

PREVENTION OF DISEASE PROGRESSION

Drugs that interfere with the excessive activation of renin-angiotensin aldosterone system (RAAS) and the adrenergic nervous system can relieve the symptoms of HF with a depressed EF by stabilizing and/ or reversing cardiac remodeling

ANGIOTENSIN-CONVERTING ENZYME INHIBITORS.

There is ample proof that patients with a reduced EF (40%), both symptomatic and asymptomatic, should take ACEIs. The enzyme that transforms angiotensin I into angiotensin II is inhibited by ACEIs, which disrupt the RAS. The effects of angiotensin suppression may be further enhanced by bradykinin overexpression, which is a potential side effect of ACEIs because they also inhibit kininase II. ACEIs improve patient symptoms, stop LV remodelling, avert hospitalization, and extend life. It is best to maximize the dose of a diuretic before beginning an ACEI because fluid retention can blunt the benefits of ACEIs. To

avoid symptomatic hypotension, it might be required to lower the dose of diuretic when starting an ACEI.

If smaller dosages have been well tolerated, ACEIs should be started at low doses and then increased gradually. Every 3 to 5 days, doses should be doubled to accomplish titration. Increase the ACEI dosage until it is comparable to the dosages that have been proven to be beneficial in clinical trials. Higher doses had a greater impact on hospital avoidance than lesser doses. The majority of ACEI side effects are brought on by RAS suppression. Blood pressure drops and mild azotemia, which are frequently experienced during the start of therapy, are typically well tolerated and don't call for a reduction in ACEI dosage.

AGENT	INITIATING DOSE	MAXIMAL DOSE
Angiotensin-Converting Enzyme Inhibitors		
Captopril	6.25 mg 3 times	50 mg 3 times
Enalapril	2.5 mg twice	10 mg twice
Lisinopril	2.5-5.0 mg once	20 mg once
Ramipril	1.25-2.5 mg once	10 mg once
Fosinopril	5-10 mg once	40 mg once
Quinapril	5 mg twice	40 mg twice
Trandolapril	0.5 mg once	4 mg once

Fig 15

ANGIOTENSIN RECEPTOR BLOCKERS:

Clinicians can now choose from a number of ARBs that have been licensed for the treatment of hypertension. Losartan, valsartan, and candesartan have each undergone thorough evaluations in the context of HF. Although they both inhibit the RAAS, ACEIs and ARBs work in a distinct ways. While ARBs block the actions of angiotensin II on the angiotensin type 1 receptor, the receptor subtype

responsible for the majority of the harmful biologic consequences related to angiotensin II on cardiac remodelling, ACEIs block the enzyme that converts angiotensin I to angiotensin II..

Although one meta-analysis suggests that ARBs and ACEIs have comparable effects on all-cause mortality and HF hospitalizations30 and although ARBs may be considered as initial therapy rather than ACEIs following MI, the general consensus is that ACEIs remain first-line therapy for the treatment of HF, while ARBs were advised for patients who were intolerant to ACEIs. ARBs have comparable effects on potassium, renal function, and blood pressure.

As a result, each of these agents will have comparable difficulties with symptomatic hypotension, azotemia, and hyperkalemia. Angioedema has also been found in certain patients, albeit less frequently than with ACEIs.

ANGIOTENSIN RECEPTOR NEPRILYSIN INHIBITORS:

Recently, a novel therapeutic class of drugs has been created that inhibits the neutral endopeptidase system and antagonises RAAS. The first substance is a mixture of sacubitril, a neprilysin inhibitor, and valsartan, an AT1 receptor antagonist. Angiotensin receptor neprilysin inhibitor (ARNI) use increases diuresis, natriuresis, and cardiac relaxation by delaying the breakdown of natriuretic peptides, bradykinin, and adrenomedullin. Additionally, it prevents the

release of renin and aldosterone, and by specifically inhibiting the AT1 receptor, it lessens myocardial hypertrophy, salt and water retention, and vasoconstriction.

Fixed-dose sacubitril/valsartan was found to significantly reduce all-cause mortality, CV mortality, and HF hospitalizations in the Prospective Comparison of ARNI with ACEI to Determine Impact on Global Mortality and Morbidity in Heart Failure Trial (PARADIGM-HF) when compared to an ACEI (enalapril) alone in patients with mild to moderate HF (NYHA Classes II to IV; LVEF 35%). This was indicated by either hospitalisation within the previous 12 months and mildly elevated natriuretic peptide levels (BNP >150 pg/mL or NT-proBNP 600 pg/mL) or by mildly elevated natriuretic peptide levels (BNP 100 pg/mL or NT-proBNP 400 pg/mL) in patients who were also able to tolerate both a target dose of (200 mg twice daily). In patients who are ACEI/ARB-naive, ARNIs should be given in low dosages (sacubitril 24 mg/valsartan 26 mg twice daily) or moderate doses (sacubitril 49 mg/valsartan 51 mg twice daily) in those who are tolerant of ACEIs/ARBs. In PARADIGM-HF, the target dose of sacubitril/valsartan was 97 mg/103 mg twice day.

ARNIs are advised as a replacement for ACEI/ARB in patients with HFrEF NYHA Class II or III who are tolerating an ACEI or ARB in order to further reduce morbidity and mortality, despite the most recent update of the

ACC/AHA/HFSA guidelines not recommending initiating HFrEF patients on ARNIs.

Angioedema is relatively uncommon and is only associated with hypotension (around 18% of patients), hyperkalemia (12%), cough (5%), and usage of an ARNI. It is not recommended to use ACEIs and ARNIs simultaneously since doing so can result in angioedema when oral neprilysin inhibitors are combined with them.

BETA BLOCKERS:

The use of beta-adrenergic blockers in the treatment of HF patients with a low EF is a significant advancement. Beta blockers compete with one or more alpha- and beta-adrenergic receptors (1, 1, and 2) in order to counteract the negative consequences of prolonged central nervous system activation. Although blocking all three receptors may have some advantages, the 1-adrenergic receptor is primarily responsible for the harmful effects of sympathetic activity. When used in conjunction with ACEIs, beta blockers alleviate patient symptoms, delay hospitalisation, and prolong life by reversing the remodelling of the LV. Therefore, patients with symptomatic or asymptomatic HF and a depressed EF less than 40% should take beta blockers.

In individuals with chronic HF, three beta blockers have been demonstrated to be useful in lowering the risk of death: Carvedilol competitively blocks the 1, 1,

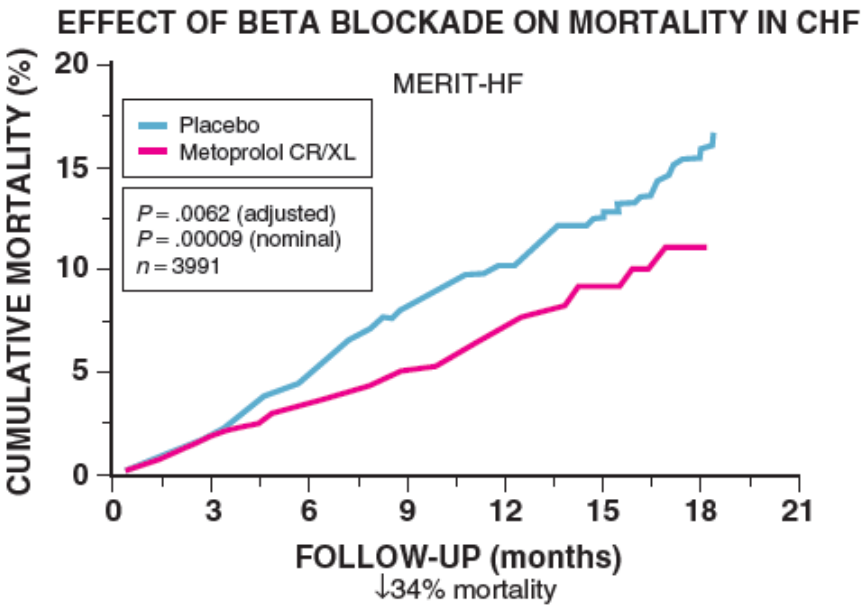
and 2 receptors, while bisoprolol and sustained-release metoprolol succinate both block the 1 receptor..

Similar to how ACEIs are used, beta blockers should be started at low dosages and then increased gradually if the lower doses have been well tolerated. When the levels employed are comparable to those that have been found to be beneficial in clinical studies, the beta blocker dose should be increased. To avoid exacerbating fluid retention caused by the abrupt withdrawal of adrenergic support for the heart and circulation, beta blockers should not be started or increased in dosage until after two weeks have passed. This is in contrast to ACEIs, which may be uptitrated more quickly. Therefore, it's crucial to optimise the diuretic dose before beginning beta blocker therapy. If worsening fluid retention does happen, it will probably happen 3 to 5 days after therapy starts and show up as an increase in body weight or worsening HF symptoms. Increasing the diuretic dose typically works to reduce the increased fluid retention. Because the majority of patients participated in the beta-blocker trials were not using high doses of ACEIs, patients were not required to be taking high-dose ACEIs before being eligible for therapy with a beta blocker.

Additionally, adding a beta blocker to patients who are on a low-dose ACEI results in a better improvement in symptoms and a further decrease in the risk of mortality than increasing the ACEI dose. According to recent research, beta blockers can be

safely administered before discharge even in HF hospital patients as long as they are stable and don't need IV HF therapy. Contrary to early reports, clinical trial data reveal that the vast majority of HF patients (>85%), including those with concomitant conditions including diabetes mellitus, chronic obstructive pulmonary disease, and peripheral vascular disease, take beta-adrenergic blocker medication well. However, a small percentage of patients (between 10% and 15%) continue to be refractory to beta blockers due to worsening fluid retention or

Fig16



MetoprololCR/XL Randomized Intervention Trial in Congestive Heart Failure (MERITHF), metoprolol CR/XL provided a significant relative risk reduction of 34% reduction in mortality in patients with mild to moderate HF and moderate to severe systolic dysfunction compared with the placebo group Importantly, metoprolol CR/X reduced mortality from both sudden death and progressive pump failure. Further, mortality was reduced across most demographic groups, including older versus younger subjects, nonischemic versus ischemic etiology, and lower versus

Because not every study including beta blockers has been a complete success, it is not absolutely necessary to consider their effects as a class effect. As a matter of fact, early experiments using the first-generation of nonspecific 1 and 2 receptors without ancillary vasodilating qualities (such as propranolol) led to a significant deterioration of HF and death..

The negative effects of beta blockers are typically connected to the predicted side effects that result from adrenergic nervous system interference. These responses typically appear a few days after therapy starts and can usually be controlled by changing concurrent drugs, as previously mentioned. We have spoken about the issue of fluid retention. Beta blocker therapy may cause sensations of overall weakness or exhaustion. In the majority of people, the increased weariness goes away on its own within a few weeks or months; in a few, it can be severe enough to need lowering the beta blocker dose, stopping the medication altogether, or changing the course of therapy.

Beta blocker therapy can worsen heart block and cause bradycardia. Beta blockers can also cause vasodilatory adverse effects, especially those that inhibit the 1 receptor. Therefore, if the heart rate drops to less than 50 beats per minute, second- or third-degree heart block develops, or symptomatic hypotension develops, the dose of beta blockers should be reduced. Even if a dose reduction might be required, continuing beta blocker therapy during an episode of acute

decompensation is safe. Patients with asthma who are experiencing an active bronchospasm are not advised to take beta blockers..

HOSPITAL CARE:

The enhancement of the patient's volume status, hemodynamic profile, and clinical symptoms; to start or enhance chronic HF therapy(23). These are the goals for managing an AHF patient throughout the inpatient phase, the acute diagnostic that were initiated during the initial presentation of the patient's case. Intensive care and total length of hospital stay reduction would be the optimum strategy for achieving these goals (LOS). It is crucial to keep track of daily weights, hydration levels, and vital signs including orthostatic blood pressure monitoring and daily evaluation of symptoms and indications. Renal function and electrolytes should be monitored daily in the laboratory..

Diagnostic tests should consist of it hasn't been done recently, an echocardiography. assessment of myocardial If there is a suspicion of ischemia, it can be necessary to a catalyst for decompensation sodium restriction in the diet (2 g daily)and limiting fluid intake (2 L per day) may be helpful to alleviate congestion, Despite the fact that sodium and hydration restriction are useful in this situation has come under increasing scrutiny. The elevated danger of the reduced blood flow in HF exacerbates venous thrombosis. AHF patients mobility and

venous thromboembolism in hospitals. In all patients unless there is a clear exception, prophylaxis is recommended contraindication..

POST DISCHARGE MANAGEMENT:

The high rates of readmission seen in AHF are likely caused by early recurrence of HF symptoms and indications that point to deteriorating volume overload and/or neurohormonal activation. Therefore, prompt interventions might make it possible to intervene in order to stop the growth of volume overload and new admissions. Rehospitalizations for HF at least in part seem to be avoidable.

Additionally, a number of studies have looked into the advantages of post-discharge assistance, particularly patient-centered discharge instructions, transition coaches, follow-up phone calls, and early physician follow-up, though the impact of these studies' findings on outcomes has been inconsistent. The ideal time to schedule a follow-up consultation is 7 to 10 days after discharge, while patients with high-risk characteristics may benefit from a shorter wait time (less than a week).

DEVICES FOR MONITORING AND MANAGING HEART FAILURE

Patients with ventricular dyssynchrony and heart failure can be treated with cardiac resynchronization therapy. Substantial data demonstrates that CRT is safe and effective, with patients displaying significant improvement in both clinical symptoms and several measures of functional status, exercise capacity, and outcomes. Now, recommendations for CRT are based on both morphology and QRS duration. ICD implantation for preventative purposes in HF patients has also been shown to be beneficial. Implantable hemodynamic monitoring devices enhance the clinician's capacity to prevent HF decompensation episodes and may enhance the disease's natural course..

VENTRICULAR DYSSYNCHRONY:

THE TARGET OF CARDIACRESYNCHRONIZATION THERAPY

There are a number of conduction anomalies that are frequently observed in patients with chronic heart failure. These include ventricular conduction anomalies, such as bundle branch blockages, which change the time and rhythm of ventricular contraction, further debilitating the already failing heart mechanically. These ventricular conduction delays result in paradoxical septal wall motion, decreased left ventricular (LV) contractility, prolonged mitral regurgitation, and poor

ventricular filling. Ventricular dyssynchrony is the collective term for several mechanical signs of abnormal ventricular conduction. A prolonged QRS duration on the surface electrocardiogram, often greater than 120 milliseconds, has been used to define ventricular dyssynchrony (ECG). According to this criteria, ventricular dyssynchrony affects around one-third of patients with systolic HF. Ventricular dyssynchrony has also been linked to a higher death rate in HF patients, in addition to limiting the ability of the failing heart to expel blood. The implantation of pacing leads into the right and left ventricles has made it possible to treat ventricular dyssynchrony with pacing therapy. The term cardiac resynchronization treatment is now used to describe this type of pacing therapy. Small observational studies investigating the acute effects of CRT on hemodynamics and other measures of cardiac performance were conducted in the mid-1990s as a result of positive single-case experiences with CRT. These studies offered more evidence in favor of the usage of CRT. A number of uncontrolled or unblinded trials to further analyse the immediate and long-term effects of CRT on clinical status in HF patients quickly followed. These trials' outcomes were equally positive, with patients showing steady, sustained improvements in their ability to tolerate physical activity, overall well-being, and functional class according to the New York Heart Association (NYHA). The positive effects of CRT on functional status and outcomes were also supported by large-scale

randomised controlled trials (RCTs), which provided the original justification for this treatment. The indications for CRT have been both widened and constrained by more recent trials.

INDICATIONS FOR PROPHYLACTIC ICD

IMPLANTATION IN HEART FAILURE PATIENTS:

Strong (level I) recommendations for preventive ICDs in patients with HF and decreased EF were made in the 2013 ACC/AHA heart failure guidelines. With LVEF of 35% or less, NYHA Class II or III symptoms, and chronic guideline-directed medical therapy (GDMT), ICD therapy is advised for the primary prevention of SCD in some patients with nonischemic dilated cardiomyopathy or ischemic heart disease who have a reasonable expectation of meaningful survival for more than one year (level of evidence A). Patients in NYHA Class I who have ischemic heart disease and are at least 40 days post-MI and have LVEF of 35% or less and have a realistic expectation of meaningful survival for more than a year are also advised to receive ICD therapy. In light of these suggestions, it's noteworthy that a recent study demonstrated the value of ICD programming in reducing unwarranted shocks and enhancing patient outcomes. This trial showed that, in comparison to conventional programming, programming of ICD therapies

for tachyarrhythmias of 200 beats per minute or higher, or with a protracted delay in therapy for tachyarrhythmias of 170 beats per minute or higher, was associated with decreases in ineffective therapy and all-cause mortality over an average follow-up of 1.4 years.

SURGICAL INTERVENTION IN HEART FAILURE:

The possible surgical intervention in heart failure are

- Coronary artery revascularization
- Left ventricular reconstruction
- Cardiac transplantation

AIMS AND OBJECTIVES:

- To assess the usefulness of ECG as predictor of LV systolic dysfunction
- .To compare the patients who don't have that ECG finding in NSTEACS and estimate it's usefulness

INCLUSION CRITERIA:

- Age above 18 yrs
- New onset myocardial injury with ECG changes of ST depression , T wave inversion

EXCLUSION CRITERIA:

- STEMI
- Known coronary artery disease
- Bundle branch block
- Pacemakers implanted patients

STUDY CENTRE:

Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai

DURATION OF THE STUDY:

6 months

STUDY DESIGN:

Case control study

SAMPLE SIZE:

140 (70CASES +70CONTROLS)

DATA COLLECTION METHODS:

1. The patients enrolled in the study will have a detailed history and examination documented after obtaining oral and written consent
2. All patients presenting with anginal chest pain to emergency room are done ECG and the patients with ST depression and T wave inversion are included
3. They are subjected to echocardiography and their systolic function is measured in ejection fraction

SAMPLE SIZE AND SAMPLING METHOD:

Consecutive patients of MI satisfying the inclusion criteria will be included. Assuming mean left ventricular ejection fraction in MI patients with fQRS and MI patients without fQRS as 41.66 % (SD: ±11.45) and 50.21% (SD:± 10.47) respectively, alpha error of 5% and 90% power, 30

72 MI patients with fQRS and 30 MI patients without fQRS will be required for the study (OpenEpi software).

DATA COLLECTION:

The data of each patients was collected in a specifically prepared proforma and includes relevant medical history, ECG findings on Admission and serial ECGs, and ejection fraction assessed by Echocardiogramme within 48 hours of onset of symptoms.

STATISTICAL METHODS:

Data were entered in Microsoft Excel spreadsheet and analysed in SPSS software. Continuous variables like age and ejection fraction were expressed as mean (Standard Deviation). Association between fQRS and Ejection fraction was tested by comparing ejection fraction in Acute MI patients having fQRS(cases) with that of Acute MI patients not having fQRS(controls),by Univariate analysis , done with paired t test and Pearson product moment correlation coefficient. A chi squared test was used to analyze the probability of differences in frequency distributions between the groups and $p<0.05$ was taken to be statistically significant in all calculations. Occurrence of adverse cardiac events(ventricular tachycardia/ventricular fibrillation,sudden cardiac death) following Acute MI among the two groups will be expressed as percentage.

DATA ANALYSIS

The results of the study are shown in tables as below. The baseline characteristics observed are as follows

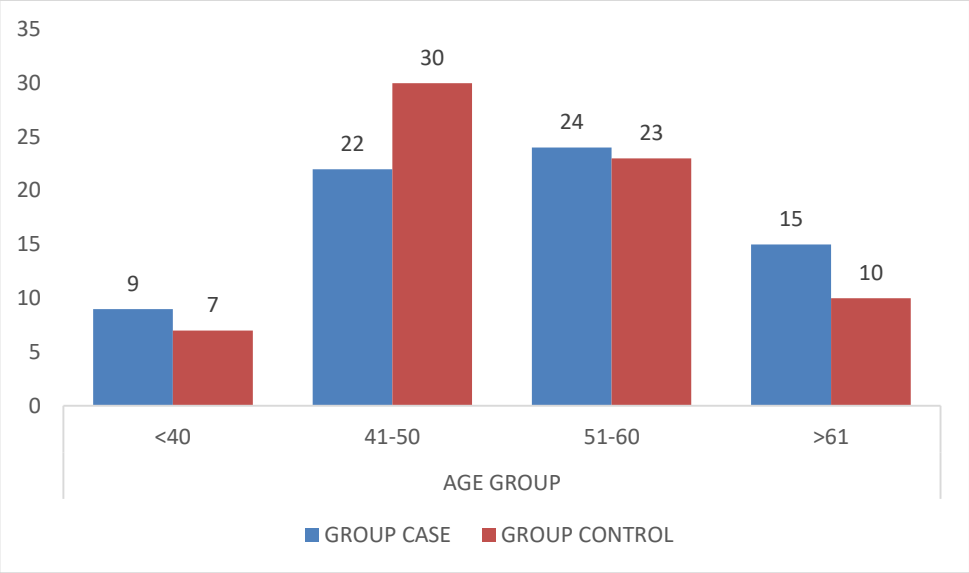
Number of cases studied are-140

AGE

Table1: Table comparing age between cases and controls

			AGE GROUP				Total	P value
			<40	41-50	51-60	>61		
GROUP	CASE	Count	9	22	24	15	70	0.475
		% within GROUP	12.9%	31.4%	34.3%	21.4%	100.0%	
	CONTROL	Count	7	30	23	10	70	
		% within GROUP	10.0%	42.9%	32.9%	14.3%	100.0%	
Total		Count	16	52	47	25	140	

Fig.17



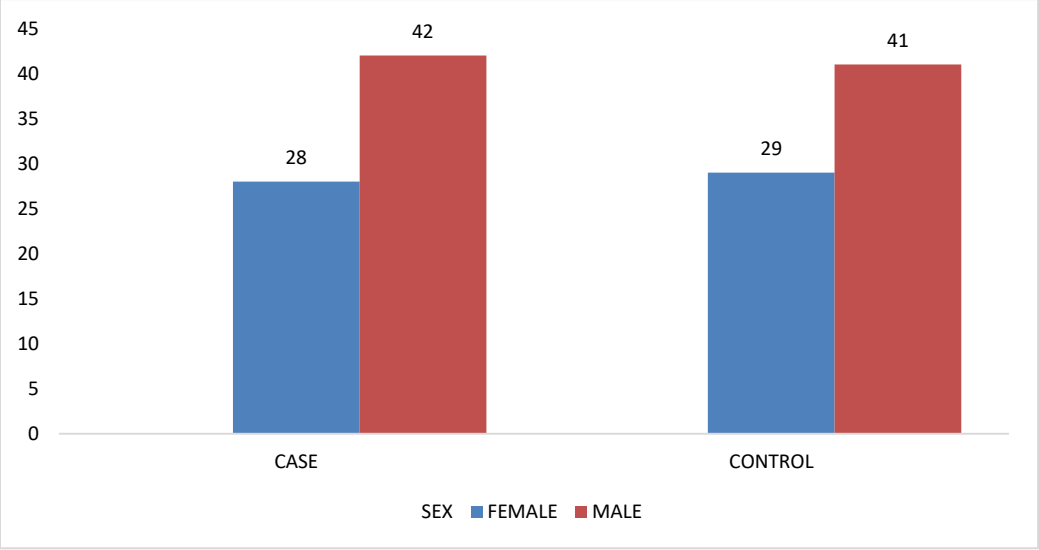
In our study the highest percentage of patients are in the age group of 40-50.yrs, There is no statistical significance among the age of distributin among cases and controls.

Sex :

Table2 Table comparing sex between cases and controls

			SEX		Total	P value
			FEMALE	MALE		
GROUP	CASE	Count	28	42	70	0.863
		% within GROUP	40.0%	60.0%	100.0%	
	CONTROL	Count	29	41	70	
		% within GROUP	41.4%	58.6%	100.0%	
Total		Count	57	83	140	
		% within GROUP	40.7%	59.3%	100.0%	

Fig18



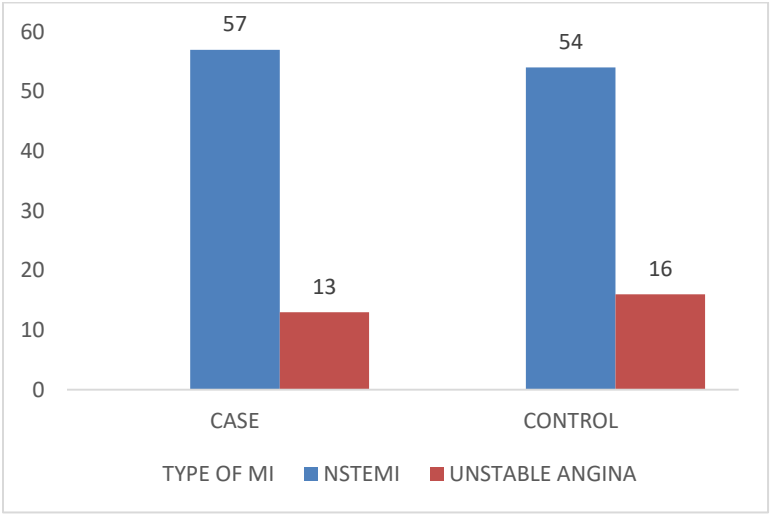
In our study cases males are 42% and females are 28% and there is no statistical significance among the cases and controls.

Type of myocardial infarction :

Table3 Table comparing MI between cases and controls

			TYPE OF MI		Total	P value
			NSTEMI	UNSTABLE ANGINA		
GROUP	CASE	Count	57	13	70	0.532
		% within GROUP	81.4%	18.6%	100.0%	
	CONTROL	Count	54	16	70	
		% within GROUP	77.1%	22.9%	100.0%	
Total		Count	111	29	140	
		% within GROUP	79.3%	20.7%	100.0%	

Fig19



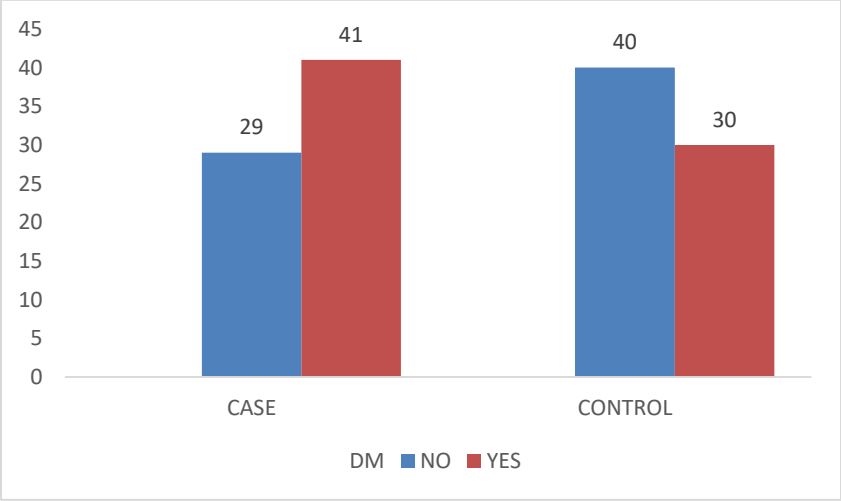
In both the cases and control NSTEMI and Unstable angina are equally distributed. In this study the number of NSTEMI cases are more than the Unstable angina. There is no statistical significance with P valure of 0.5

DIABETES :

Table4 Table comparing diabetes between cases and controls

			DM		Total	P value
			NO	YES		
GROUP	CASE	Count	29	41	70	0.063
		% within GROUP	41.4%	58.6%	100.0%	
	CONTROL	Count	40	30	70	
		% within GROUP	57.1%	42.9%	100.0%	
Total		Count	69	71	140	
		% within GROUP	49.3%	50.7%	100.0%	

Fig20



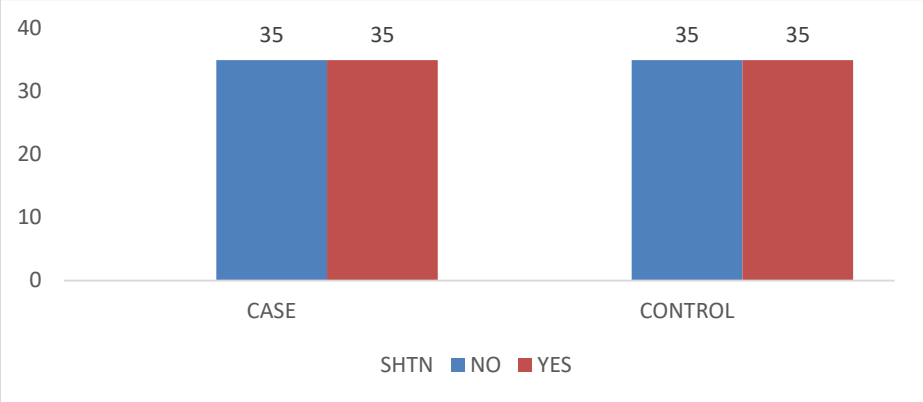
In our study case groups, DM are present in 58.6% and in control groups are 42.9%. There is no statistical significance with a p value (0.06

SYSTEMIC HYPERTENSION:

Table5 Table comparing hypertension between cases and controls

			SHTN		Total	P value
			NO	YES		
GROUP	CASE	Count	35	35	70	1.000
		% within GROUP	50.0%	50.0%	100.0%	
	CONTROL	Count	35	35	70	
		% within GROUP	50.0%	50.0%	100.0%	
Total		Count	70	70	140	
		% within GROUP	50.0%	50.0%	100.0%	

Fig21



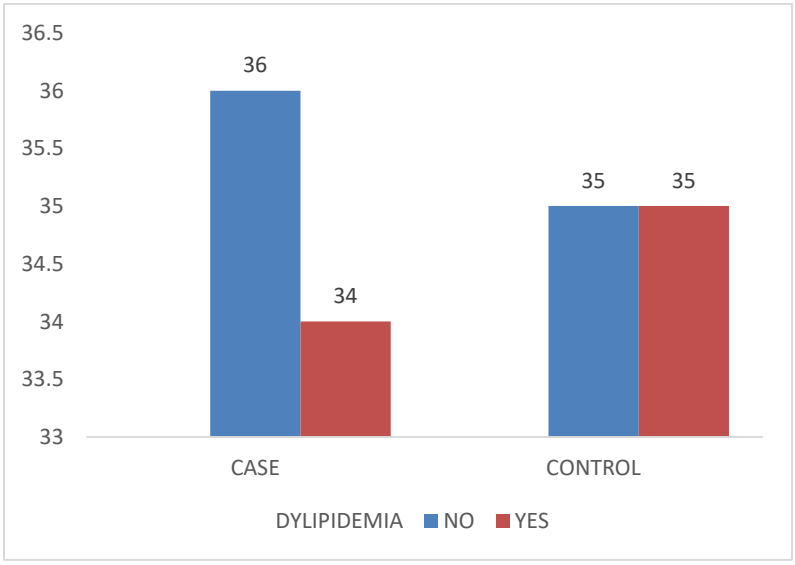
In this study number of hypertension cases between cases and control are same and is not statistically signifiant.. Hence no corrélation with fQRS.

Dyslipidemia :

Table6 Table comparing dyslipidemia between cases and controls

			DYLIPIDEMIA		Total	P value
			NO	YES		
GROUP	CASE	Count	36	34	70	0.866
		% within GROUP	51.4%	48.6%	100.0%	
	CONTROL	Count	35	35	70	
		% within GROUP	50.0%	50.0%	100.0%	
Total		Count	71	69	140	
		% within GROUP	50.7%	49.3%	100.0%	

Fig22



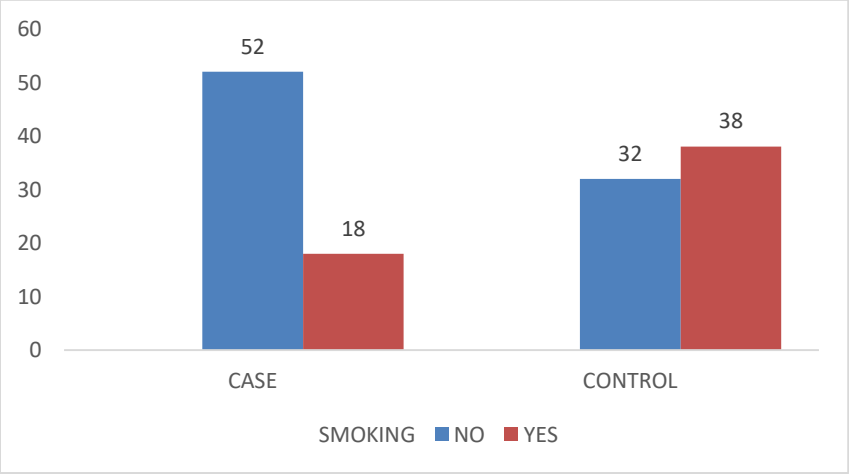
In this study the hypertension is not significantly correlated between the cases and controls

Smoking:

Table7 Table comparing smoking between cases and controls

			SMOKING		Total	P value
			NO	YES		
GROUP	CASE	Count	52	18	70	0.001
		% within GROUP	74.3%	25.7%	100.0%	
	CONTROL	Count	32	38	70	
		% within GROUP	45.7%	54.3%	100.0%	
Total		Count	84	56	140	
		% within GROUP	60.0%	40.0%	100.0%	

Fig23



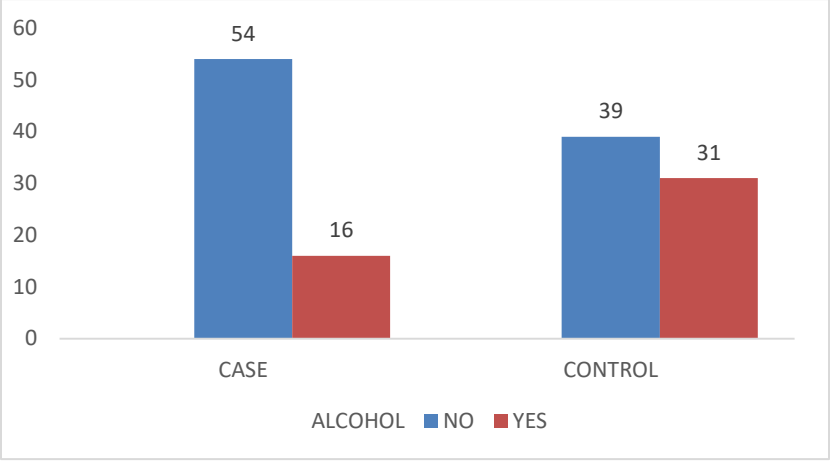
In this study cases shows smoking in 18% and in controls shows smoking in 38% of patients. The P value is (0.001) statistically significant . Thus smoking is associated significantly with fQRS.

ALCOHOL :

Table8

			ALCOHOL		Total	P value
			NO	YES		
GROUP	CASE	Count	54	16	70	0.007
		% within GROUP	77.1%	22.9%	100.0%	
	CONTROL	Count	39	31	70	
		% within GROUP	55.7%	44.3%	100.0%	
Total		Count	93	47	140	
		% within GROUP	66.4%	33.6%	100.0%	

Fig24



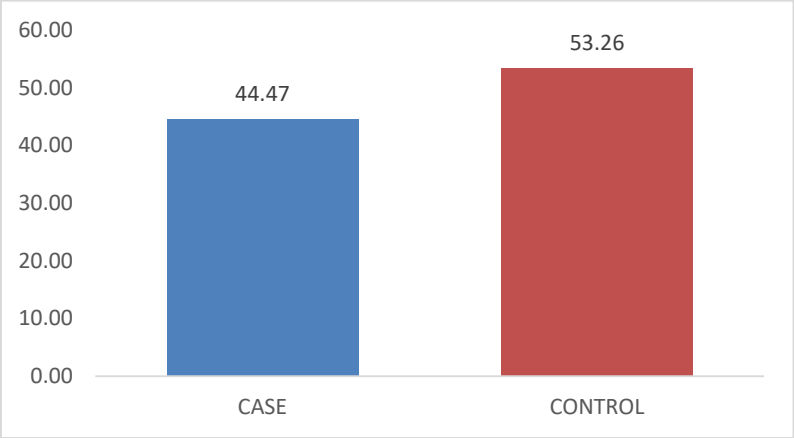
In this study cases shows alcohol in 16% and in controls shows smoking in 31% of patients. The P value is (0.001) statistically significant. Thus smoking is associated significantly with fQRS

EJECTION FRACTION

Table9 Table comparing ejection fraction between cases and controls :

GROUP		Mean	Std. Deviation	P value
EJECTION FRACTION	CASE	44.47	10.72	<0.0001
	CONTROL	53.26	8.89	

Fig25



The average ejection fraction among the cases were 44.7% and controls were 53.26%

There is significant différence between the cases and controls with a P value (<0.0001)

DISCUSSION :

In India and other developing nations, ischemic heart disease is the major cause of illness and mortality. Acute coronary syndrome (STEMI, non-STEMI, unstable)

A medical emergency is angina. In addition to detrimental lifestyle changes and a high prevalence of risk factors such diabetes mellitus, alcoholism, smoking, and hypertension, the incidence of acute coronary syndrome is rising daily. Numerous indicators, including related heart failure symptoms, the left ventricular ejection fraction, and elevated cardiac biomarkers, can be used to evaluate the prognosis in individuals with acute coronary syndrome.

In this case control study of relationship between fragmented QRS in an ECG and left ventricular ejection fraction patients presenting with NON STEACS consists of a group of 140 patients(cases 70+controls 70) who were admitted in intensive coronary care unit, Madras Medical College &Hospital, Chennai

In our study smoking appears to be correlated with fragmented QRS. The mean ejection fraction of cases are 44% and controls are 53% and the P valure is 0.0001.

CONCLUSION:

- There is a significant association between presence of fragmented QRS and the ejection fraction in the setting acute myocardial infarction
- This study shows no statistically significant relationship between presence or absence of fragmented QRS and sex distribution, diabetes, hypertension and alcoholism.

LIMITATIONS OF THE STUDY:

- A 12 lead ECG with a fragmented QRS requires a low pass filter setting of 100 or 150 Hz. With a filter setting of 40 or 60 Hz, fragmentation can be readily ignored.
- Its that a fragmented QRS is not a particular finding and should only be interpreted if there is sufficient clinical proof of myocardial scarring, such as in coronary artery disease or primary electrical anomalies like Brugada syndrome.

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ABBREVIATIONS USED

- ACS- ACUTE CORONARY SYNDROME
- STEMI- ST ELEVATION MYOCARDIAL INFARCTION.
- NSTEMI- NON ST ELEVATION MYOCARDIAL INFARCTION
- UA- UNSTABLE ANGINA
- SHT- SYSTEMIC HYPERTENSION
- DM- DIABETES MELLITUS
- LVD- LEFT VENTRICULAR DYSFUNCTION
- AWMI- ANTERIOR WALL MYOCARDIAL INFARCTION
- IWMI- INFERIOR WALL MYOCARDIAL INFARCTION
- RVMI- RIGHT VENTRICULAR MYOCARDIAL INFARCTION
- CPK-MB- CREATININE PHOSPHOKINASE MB
- CHD- CORONARY HEART DISEASE
- ACE- ANGOTENSION CONVERTING ENZYME
- PCI- PERCUTANEOUS CORONARY INTREVENTION
- ECG- ELECTROCARDIOGRAM
- HDL-HIGH DENSUTY LIPOPROTEIN
- LDL- LOW DENSITY LIPOPROTEIN
- UFH- UNFRACTIONATED HEPARIN
- LMWH- LOW MOLECULAR WEIGHT HEPARIN

PROFORMA

PROFORMA NO.

NAME: AGE/SEX:

CONTACT

COMORBIDITY:

- DIABETES
- HYPERTENSION

RISK FACTOR

- ALCOHOL
- SMOKING

DIAGNOSIS:

- NSTEMI / UNSTABEL ANGINA

ECHO : Ejection Fraction

PATIENT INFORMATION SHEET

We are conducting "**A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION ",** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

In this study we select the patients who are presenting with acute chest pain and there are asked about their risk factors and examination done. Patients are then done Ecg to determine the type of myocardial infarction. Patients with NSTEACS are selected and then done ECHO.

Both ECG and ECHO are noninvasive investigations, there is no chances of causing harm to the patients

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

தகவல் தாள்

சென்னை ராஜீவ் காந்தி அரசு பொது மருத்துவமனையில் அனுமதிக்கப்படும் நோயாளிகளிடையே : "**A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION**" குறித்து ஒரு ஆய்வை நாங்கள் மேற்கொண்டு வருகிறோம், அதற்காக உங்கள் மாதிரி எங்களுக்கு மதிப்புமிக்கதாக இருக்கும்.

நாங்கள் சில நோயாளிகள் தேர்வு செய்கிறோம், நீங்கள் தகுதியுள்ளவராகக் கண்டறியப்பட்டால், சில சோதனைகளைச் செய்ய உங்கள் இரத்த மாதிரிகளை மற்றும் ஊடுகதிர் பயன்படுத்துகிறோம். ஆராய்ச்சியில் நோயாளிகளின் தனியுரிமை ஆய்வு முழுவதும் பராமரிக்கப்படும். ஆராய்ச்சியின் விளைவாக ஏதேனும் வெளியீடு அல்லது விளக்கக்காட்சி ஏற்பட்டால், தனிப்பட்ட முறையில் அடையாளம் காணக்கூடிய தகவல்கள் எதுவும் பகிரப்படாது.

இந்த ஆய்வில் பங்கேற்பது தன்னார்வமானது. இந்த ஆய்வில் பங்கேற்க வேண்டுமா அல்லது எந்த நேரத்திலும் திரும்பப் பெறலாமா என்பதை நீங்கள் தீர்மானிக்க சுதந்திரமாக இருக்கிறீர்கள்; உங்கள் முடிவால் உங்களுக்கு உரிமையுள்ள எந்தவொரு நன்மைகளிலும் இடைவெளி ஏற்படாது.

புலனாய்வாளரின் கையொப்பம் பங்கேற்பாளரின் கையொப்பம்

PATIENT CONSENT FORM

Study Detail : " **A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION** "

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

Identification Number :

Patient may check (√) these boxes

- I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.
- I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.

- I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.
- I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.
- I hereby consent to participate in this study.
- I hereby give permission to undergo detailed clinical examination and blood investigations as required.

Signature of investigator Signature/Thumb impression of participant

Patient name and address:

நோயாளி ஒப்புதல் படிவம்

ஆய்வு விவரம் : "**A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION** "

ஆய்வு மையம்: ராஜீவ் காந்தி அரசு பொது மருத்துவமனை, சென்னை.

நோயாளியின் பெயர்:

நோயாளியின் வயது:

அடையாள எண் :

நோயாளி இந்த பெட்டிகளை (√) செய்யலாம்:-

- மேற்கண்ட ஆய்விற்கான நடைமுறையின் நோக்கத்தை நான் புரிந்து கொண்டேன் என்பதை உறுதிப்படுத்துகிறேன். கேள்வி கேட்க எனக்கு வாய்ப்பு உள்ளது, எனது முழு திருப்திக்கும் எனது எல்லா கேள்விகளுக்கும் சந்தேகங்களுக்கும் பதில் அளிக்கப்பட்டுள்ளது.
- ஆய்வில் நான் பங்கேற்பது தன்னார்வமானது என்பதையும், எனது சட்ட உரிமைகள் பாதிக்கப்படாமல், காரணமின்றி எந்த நேரத்திலும் திரும்பப் பெற எனக்கு சுதந்திரம் உள்ளது என்பதையும் நான் புரிந்துகொள்கிறேன்.
- மருத்துவ ஆய்வின் ஸ்பான்சர், ஸ்பான்சர் சார்பாக பணிபுரியும் மற்றவர்கள், நெறிமுறைக் குழு மற்றும் ஒழுங்குமுறை அதிகாரிகள் எனது சுகாதார பதிவுகளைப் பார்க்க எனது அனுமதி தேவையில்லை என்பதை நான் புரிந்துகொள்கிறேன், தற்போதைய ஆய்வு மற்றும் மேற்கொண்டுள்ள எந்தவொரு ஆராய்ச்சியையும் பொறுத்தவரை இது

தொடர்பாக, நான் ஆய்வில் இருந்து விலகினாலும் இந்த அணுகலை ஒப்புக்கொள்கிறேன். எவ்வாறாயினும், சட்டத்தின் கீழ் தேவைப்படாவிட்டால், மூன்றாம் தரப்பினருக்கு வெளியிடப்பட்ட அல்லது வெளியிடப்பட்ட எந்தவொரு தகவலிலும் எனது அடையாளம் வெளிப்படுத்தப்படாது என்பதை நான் புரிந்துகொள்கிறேன். இந்த ஆய்வில் எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் பயன்பாட்டை கட்டுப்படுத்த வேண்டாம் என்று நான் ஒப்புக்கொள்கிறேன்.

- மேற்கண்ட ஆய்வில் பங்கேற்கவும், ஆய்வின் போது கொடுக்கப்பட்ட அறிவுறுத்தல்களுக்கு இணங்கவும், ஆய்வுக் குழுவுடன் உண்மையுடன் ஒத்துழைக்கவும், எனது உடல்நலம் அல்லது நல்வாழ்வில் ஏதேனும் சரிவு ஏற்பட்டால் அல்லது எதிர்பாராத அல்லது ஏதேனும் ஏற்பட்டால் உடனடியாக ஆய்வு ஊழியர்களுக்கு அறிவிக்கவும் ஒப்புக்கொள்கிறேன். அசாதாரண அறிகுறிகள்.
- இந்த ஆய்வில் பங்கேற்க நான் இதன்மூலம் ஒப்புக்கொள்கிறேன்.
- தேவைக்கேற்ப விரிவான மருத்துவ பரிசோதனை மற்றும் இரத்த விசாரணைகளை மேற்கொள்ள நான் இதன்மூலம் அனுமதி அளிக்கிறேன்.

புலனாய்வாளரின் கையொப்பம் பங்கேற்பாளரின் கையொப்பம் / கட்டைவிரல் எண்ணம்

நோயாளியின் பெயர் மற்றும் முகவர

INSTITUTE ETHICAL COMMITTEE APPROVAL:

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

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EC Reg. No(DHR).EC/NEW/INST/2021/1618
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.N.NAVEENKUMAR,
MD Internal Medicine Post Graduate student,
Institute of Internal Medicine,
Madras Medical College,
Chennai-600003.

Dear Dr. N.NAVEENKUMAR,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION"- NO.15072022.** The following members of Ethics Committee were present in the meeting held on **06.07.2022** conducted at Madras Medical College, Chennai 3.

1. Prof.P.V.Jayashankar,MS Orth.,D.Orth.,M.Ch Orth (Liverpool) :Chairperson
2. Prof.N.Gopalakrishnan,MD.,DM., FRCP, Director, Inst.of Nephrology,MMC,Ch. : Member Secretary
3. Prof. K.M.Sudha, Prof. Inst. of Pharmacology,MMC,Ch-3 : Member
4. Prof. Alagarsamy Jamila ,MD, Vice Principal, Stanley Medical College, Chennai : Member
5. Prof.Meena Suresh, MD.,DGO.,Prof.of Obst & Gynaec, IOG,Chennai : Member
6. Prof.S.Lakshmi, Prof. of Paediatrics ICH Chennai :Member
7. Tmt.Arnold Saulina, MA.,MSW., :Social Scientist
8. Thiru S.Govindasamy, BA.,BL,High Court,Chennai : Lawyer
9. Thiru K.Ranjith, Ch- 91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003.

PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled of the "**A STUDY ON FRAGMENTED QRS AS A PREDICTOR OF LV DYSFUNCTION IN NONSTELEVATION MYOCARDIAL INFARCTION"** by the candidate **Dr. NAVEENKUMAR N** with registration Number **200120100519** for the award of **DOCTOR OF MEDICINE** in the branch of **GENERAL MEDICINE**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 2**%**percentageplagiarism in the dissertation.

PROF. Dr.P. MALARVIZHI M.D.,
Guide,
Professor of Internal Medicine,
Madras Medical College,
Chennai – 600 003.

PROFESSOR
INSTITUTE OF INTERNAL MEDICINE
MMC & RGGGH, CHENNAI-600 003

PLAGIARISM CERTIFICATE

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SA	**dissertation vivek.docx** Document dissertation vivek.docx (D121789871)	3
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SA	**Guidelines-Acute and Chronic-HF-FT.pdf** Document Guidelines-Acute and Chronic-HF-FT.pdf (D12824405)	1

MASTER CHART:

S.N	NAME	GROU	AGE		SEX	TYPE OF MI	DM	SHT	DYLIPI MIA	SMOKII	ALCO I	EJECTI FRACTI
1	ramu	1	56	3	MALE	NSTEMI	YES	YES	YES	NO	NO	40
2	malliga	1	40	1	FEMALE	NSTEMI	NO	YES	NO	NO	NO	32
3	padmaban	1	55	3	MALE	NSTEMI	NO	NO	YES	YES	YES	45
4	patti	1	33	1	MALE	NSTEMI	NO	NO	YES	YES	NO	55
5	venkatachalam	1	48	2	MALE	NSTEMI	YES	NO	YES	YES	YES	28
6	anjappar	1	60	3	MALE	UNSTABLE ANGINA	YES	YES	NO	NO	NO	60
7	venda	1	55	3	FEMALE	NSTEMI	YES	NO	YES	NO	NO	55
8	ponniah	1	65	4	MALE	NSTEMI	YES	NO	NO	YES	NO	45
9	asokan	1	39	1	MALE	NSTEMI	NO	YES	NO	YES	YES	50
10	rajavelu	1	48	2	MALE	NSTEMI	NO	YES	YES	YES	NO	41
11	subburayan	1	70	4	MALE	UNSTABLE ANGINA	YES	NO	YES	NO	NO	37
12	pavalammal	1	47	2	FEMALE	NSTEMI	NO	NO	YES	YES	YES	60
13	kamatchi	1	52	3	FEMALE	NSTEMI	NO	YES	YES	NO	NO	58
14	philip	1	36	1	MALE	NSTEMI	YES	NO	NO	YES	NO	39
15	mohammed	1	57	3	MALE	NSTEMI	YES	NO	YES	NO	NO	33
16	panjali	1	48	2	FEMALE	UNSTABLE ANGINA	YES	NO	NO	NO	NO	55
17	anitha	1	50	2	FEMALE	UNSTABLE ANGINA	YES	YES	NO	NO	NO	40
18	muniyammal	1	49	2	FEMALE	NSTEMI	NO	NO	NO	NO	NO	28
19	sreedevi	1	56	3	FEMALE	UNSTABLE ANGINA	YES	YES	NO	NO	NO	47
20	satish kumar	1	61	4	MALE	NSTEMI	YES	YES	YES	YES	NO	41
21	shaqlin	1	47	2	MALE	NSTEMI	YES	NO	NO	YES	NO	58
22	gopal	1	52	3	MALE	NSTEMI	YES	YES	NO	NO	YES	40
23	ammaniammal	1	40	1	MALE	NSTEMI	NO	YES	YES	NO	NO	60
24	manivel	1	39	1	MALE	NSTEMI	YES	YES	NO	NO	NO	40
25	rajapandian	1	48	2	MALE	NSTEMI	YES	NO	NO	NO	NO	50
26	saravanan	1	52	3	MALE	UNSTABLE ANGINA	NO	NO	YES	YES	NO	46
27	varadarajan	1	67	4	MALE	NSTEMI	NO	YES	YES	NO	YES	53
28	indrani	1	49	2	FEMALE	NSTEMI	NO	YES	NO	NO	NO	49
29	kaliyammal	1	55	3	FEMALE	NSTEMI	NO	YES	YES	NO	NO	34
30	murugan	1	58	3	MALE	NSTEMI	NO	NO	YES	YES	NO	53
31	thalapakatti	1	62	4	MALE	NSTEMI	NO	YES	NO	YES	NO	54
32	rajendran	1	56	3	MALE	NSTEMI	YES	YES	NO	NO	YES	46
33	mohanasunadram	1	48	2	MALE	NSTEMI	YES	NO	NO	NO	YES	35
34	kaja moideen	1	35	1	MALE	NSTEMI	YES	NO	NO	NO	NO	63
35	sekar	1	44	2	MALE	NSTEMI	YES	YES	YES	NO	YES	64
36	fathima bee	1	49	2	FEMALE	UNSTABLE ANGINA	YES	YES	NO	NO	NO	50
37	kolandaiyammal	1	66	4	FEMALE	UNSTABLE ANGINA	YES	NO	YES	NO	NO	43
38	vadivukarasi	1	53	3	FEMALE	NSTEMI	YES	NO	YES	NO	NO	47
39	amudha	1	54	3	FEMALE	UNSTABLE ANGINA	NO	YES	YES	NO	NO	39
40	amarnath	1	48	2	MALE	NSTEMI	NO	NO	YES	NO	YES	35
41	christy	1	58	3	FEMALE	NSTEMI	NO	YES	NO	YES	NO	29
42	ramakrishnan	1	37	1	MALE	NSTEMI	YES	YES	NO	NO	YES	32
43	manirathnam	1	49	2	MALE	NSTEMI	YES	YES	NO	NO	NO	29
44	nagappan	1	62	4	MALE	NSTEMI	YES	YES	NO	NO	NO	40
45	rathinammmal	1	57	3	FEMALE	NSTEMI	YES	NO	NO	NO	NO	58
46	lingeshwaran	1	54	3	MALE	NSTEMI	YES	NO	YES	YES	NO	37
47	babu	1	43	2	MALE	UNSTABLE ANGINA	NO	YES	YES	NO	YES	28
48	chinnaponnu	1	44	2	FEMALE	NSTEMI	YES	NO	NO	NO	NO	35
49	yogambal	1	63	4	FEMALE	NSTEMI	NO	NO	NO	NO	NO	62
50	duraisamy	1	71	4	MALE	UNSTABLE ANGINA	NO	NO	NO	YES	NO	45
51	thulasi	1	66	4	FEMALE	NSTEMI	YES	YES	YES	NO	NO	44
52	ganapathyraja	1	42	2	MALE	NSTABLE ANGIN	YES	NO	YES	NO	YES	51
53	gurumoorthy	1	53	3	MALE	NSTEMI	YES	YES	YES	NO	YES	24
54	rajathy	1	58	3	FEMALE	NSTEMI	NO	NO	YES	NO	NO	30
55	uma	1	68	4	FEMALE	NSTEMI	NO	NO	NO	NO	NO	56
56	anjuthan	1	59	3	MALE	NSTEMI	YES	NO	NO	YES	NO	68
57	benazir	1	44	2	FEMALE	NSTEMI	NO	YES	NO	NO	NO	47
58	kathavarayan	1	71	4	FEMALE	NSTEMI	YES	NO	NO	YES	YES	50
59	nathan	1	45	2	MALE	NSTEMI	NO	YES	YES	NO	NO	46
60	veerammal	1	62	4	FEMALE	NSTEMI	NO	NO	YES	NO	NO	42
61	usharani	1	43	2	FEMALE	NSTABLE ANGIN	YES	YES	NO	NO	NO	33
62	oliver	1	35	1	MALE	NSTEMI	NO	YES	YES	NO	NO	50
63	sitalaxmi	1	53	3	FEMALE	NSTEMI	NO	NO	YES	NO	NO	41
64	danalaxmi	1	57	3	FEMALE	NSTEMI	NO	NO	YES	NO	NO	43

55	uma	1	68	4	FEMALE	NSTEMI	NO	NO	NO	NO	NO	56
56	anjuthan	1	59	3	MALE	NSTEMI	YES	NO	NO	YES	NO	68
57	benazir	1	44	2	FEMALE	NSTEMI	NO	YES	NO	NO	NO	47
58	kathavarayan	1	71	4	FEMALE	NSTEMI	YES	NO	NO	YES	YES	50
59	nathan	1	45	2	MALE	NSTEMI	NO	YES	YES	NO	NO	46
60	veerammal	1	62	4	FEMALE	NSTEMI	NO	NO	YES	NO	NO	42
61	usharani	1	43	2	FEMALE	NSTABLE ANGIN	YES	YES	NO	NO	NO	33
62	oliver	1	35	1	MALE	NSTEMI	NO	YES	YES	NO	NO	50
63	sitalaxmi	1	53	3	FEMALE	NSTEMI	NO	NO	YES	NO	NO	41
64	danalaxmi	1	57	3	FEMALE	NSTEMI	NO	NO	YES	NO	NO	43
65	baskar	1	47	2	MALE	NSTEMI	YES	YES	NO	NO	NO	48
66	jonathan	1	63	4	MALE	NSTEMI	YES	YES	NO	NO	NO	63
67	rafiq	1	80	4	MALE	NSTEMI	YES	NO	YES	NO	NO	37
68	vinoth kumar	1	59	3	MALE	NSTEMI	YES	YES	NO	NO	YES	25
69	shivaji	1	57	3	MALE	NSTEMI	YES	NO	YES	NO	NO	35
70	kusbboo	1	48	2	FEMALE	NSTEMI	YES	YES	NO	NO	NO	37
71	ramamoorthy	2	53	3	MALE	NSTEMI	YES	NO	YES	YES	NO	50
72	pappaya	2	49	2	FEMALE	NSTEMI	NO	YES	NO	YES	NO	56
73	raja	2	57	3	MALE	NSTABLE ANGIN	YES	NO	YES	YES	NO	49
74	rathinavel	2	62	4	MALE	NSTEMI	NO	NO	YES	NO	YES	57
75	pandiyan	2	58	3	MALE	NSTEMI	NO	NO	YES	YES	YES	62
76	jit goel	2	60	3	MALE	NSTEMI	YES	NO	NO	YES	YES	57
77	suren	2	48	2	MALE	NSTEMI	YES	YES	YES	YES	NO	38
78	hariom agarwal	2	50	2	MALE	NSTEMI	NO	YES	YES	YES	NO	48
79	rasathi	2	63	4	FEMALE	NSTABLE ANGIN	YES	NO	NO	NO	NO	59
80	yuvarani	2	61	4	FEMALE	NSTEMI	NO	YES	YES	NO	NO	68
81	kanammal	2	53	3	FEMALE	NSTEMI	YES	YES	YES	NO	NO	62
82	periyar	2	47	2	MALE	NSTEMI	NO	NO	YES	YES	YES	54
83	bharathi	2	49	2	FEMALE	NSTEMI	NO	YES	YES	NO	YES	60
84	janammal	2	47	2	FEMALE	NSTEMI	YES	NO	NO	NO	NO	51
85	ruthammal	2	53	3	FEMALE	NSTABLE ANGIN	NO	NO	YES	NO	NO	43
86	manoranjan	2	48	2	MALE	NSTEMI	YES	YES	NO	YES	YES	40
87	edwin	2	54	3	MALE	NSTABLE ANGIN	NO	NO	NO	YES	YES	55
88	pinky	2	60	3	FEMALE	NSTEMI	YES	NO	NO	NO	YES	63
89	renu	2	50	2	FEMALE	NSTABLE ANGIN	NO	YES	NO	NO	NO	57
90	vasanthi	2	60	3	FEMALE	NSTEMI	YES	NO	YES	NO	NO	50
91	preetham	2	46	2	MALE	NSTEMI	NO	YES	NO	YES	YES	35
92	chandramouli	2	48	2	MALE	NSTEMI	YES	YES	NO	YES	NO	47
93	sadasivam	2	52	3	MALE	NSTEMI	NO	NO	YES	NO	NO	53
94	kannuupaiyan	2	37	1	MALE	NSTABLE ANGIN	YES	YES	NO	YES	NO	56
95	chinrasu	2	40	1	MALE	NSTEMI	YES	YES	NO	YES	YES	54
96	ravi	2	56	3	MALE	NSTABLE ANGIN	YES	YES	YES	NO	YES	65
97	duraisamy	2	50	2	MALE	NSTEMI	YES	NO	YES	YES	YES	68
98	prabu	2	57	3	MALE	NSTEMI	YES	YES	NO	YES	NO	53
99	kalvathy	2	60	3	FEMALE	NSTEMI	NO	YES	YES	NO	NO	56
100	manimegalia	2	37	1	FEMALE	NSTEMI	YES	NO	YES	NO	NO	56
101	nadiya	2	48	2	FEMALE	NSTABLE ANGIN	NO	YES	NO	NO	NO	40
102	uma	2	57	3	FEMALE	NSTEMI	YES	YES	NO	NO	NO	57
103	babu	2	43	2	MALE	NSTEMI	NO	NO	NO	YES	YES	58
104	daniel	2	67	4	FEMALE	NSTEMI	NO	NO	NO	YES	NO	62

105	ali baba	2	70	4	MALE	NSTEMI	YES	YES	YES	NO	NO	32
106	ganesh balaji	2	58	3	MALE	NSTABLE ANGIN	NO	YES	NO	YES	YES	40
107	madusudahanan	2	43	2	MALE	NSTABLE ANGIN	YES	NO	YES	YES	YES	56
108	revathi	2	58	3	FEMALE	NSTEMI	NO	NO	YES	NO	NO	57
109	joseph sen	2	61	4	MALE	NSTABLE ANGIN	YES	YES	YES	YES	YES	58
110	inniyan	2	58	3	MALE	NSTEMI	NO	NO	YES	YES	YES	51
111	manimozhi	2	43	2	FEMALE	NSTEMI	YES	YES	NO	NO	NO	54
112	vembuli	2	38	1	MALE	NSTEMI	NO	YES	NO	YES	NO	62
113	vijaya	2	46	2	FEMALE	NSTEMI	YES	YES	NO	NO	NO	68
114	kupammal	2	42	2	FEMALE	NSTEMI	NO	YES	NO	YES	NO	51
115	vanitha	2	53	3	FEMALE	NSTEMI	YES	NO	NO	NO	NO	57
116	jesudas	2	51	3	MALE	NSTABLE ANGIN	NO	NO	YES	YES	YES	30
117	karupannan	2	49	2	MALE	NSTEMI	NO	YES	YES	YES	YES	35
118	latha	2	39	1	FEMALE	NSTEMI	NO	NO	NO	NO	NO	63
119	anbalagan	2	47	2	MALE	NSTEMI	YES	NO	NO	YES	YES	56
120	banumathi	2	67	4	FEMALE	NSTABLE ANGIN	NO	NO	NO	NO	NO	49
121	yeshwanth	2	55	3	MALE	NSTEMI	NO	YES	YES	YES	YES	51
122	ashiq	2	43	2	MALE	NSTEMI	NO	NO	YES	NO	YES	58
123	dawood	2	68	4	MALE	NSTEMI	YES	YES	YES	YES	NO	62
124	sivagami	2	41	2	FEMALE	NSTEMI	NO	NO	YES	NO	NO	58
125	sree chitra	2	51	3	FEMALE	NSTABLE ANGIN	NO	NO	NO	NO	NO	50
126	manorammal	2	37	1	FEMALE	NSTEMI	YES	NO	NO	NO	NO	48
127	sivamalar	2	45	2	FEMALE	NSTEMI	NO	YES	NO	NO	NO	53
128	yesupatham	2	58	3	MALE	NSTEMI	NO	NO	NO	YES	YES	56
129	antony vijay	2	47	2	MALE	NSTEMI	YES	YES	YES	YES	YES	60
130	sundar	2	50	2	MALE	NSTEMI	NO	NO	YES	NO	YES	37
131	jack	2	57	3	MALE	NSTABLE ANGIN	NO	YES	NO	YES	YES	51
132	velumani	2	63	4	MALE	NSTEMI	NO	YES	YES	YES	YES	57
133	gnanasezhian	2	47	2	MALE	NSTEMI	NO	NO	YES	YES	YES	33
134	vardammal	2	49	2	FEMALE	NSTEMI	YES	NO	YES	NO	NO	64
135	senguttuvan	2	68	4	MALE	NSTEMI	NO	YES	NO	YES	YES	61
136	ganesan	2	44	2	MALE	NSTABLE ANGIN	NO	YES	NO	YES	YES	53
137	kuppan	2	38	1	MALE	NSTEMI	NO	NO	YES	YES	NO	64
138	elizabeth	2	48	2	FEMALE	NSTEMI	YES	YES	NO	NO	NO	53
139	sudhamoorthy	2	48	2	FEMALE	NSTEMI	NO	NO	YES	NO	NO	47
140	rakesh	2	50	2	MALE	NSTEMI	NO	YES	NO	YES	YES	54