OBSERVATIONAL STUDY ON PHARMACOINVASIVE STRATEGY AND PRIMARY ANGIOPLASTY IN A TERTIARY CARE CENTER IN ACUTE MYOCARDIAL INFARCTION PATIENTS

A dissertation submitted in partial fulfillment of

D.M. (Cardiology) Examination of the Tamil Nadu

Dr. M.G.R. UNIVERSITY, Chennai

to be held in 2015

CONTENTS

CERTIFICATE

This is to certify that the dissertation entitled

"Observational study on Pharmacoinvasive Strategy & Primary Angioplasty in a Tertiary Care Centre in Acute Myocardial Infarction Patients."

is a bonafide work done by
ANOOP GEORGE ALEX
Christian Medical College, Vellore, Tamil Nadu

in partial fulfillment of the University rules and regulations for award of
DM – Branch II Cardiology
Under my guidance and supervision during
the academic year 2012 - 15

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Dear Dr. Anoop George Alex,

I enclose the following documents:-

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Could you please sign the agreement and send it to Dr. Nihal Thomas, Addl. Vice Principal (Research), so that the grant money can be released.

With best wishes,

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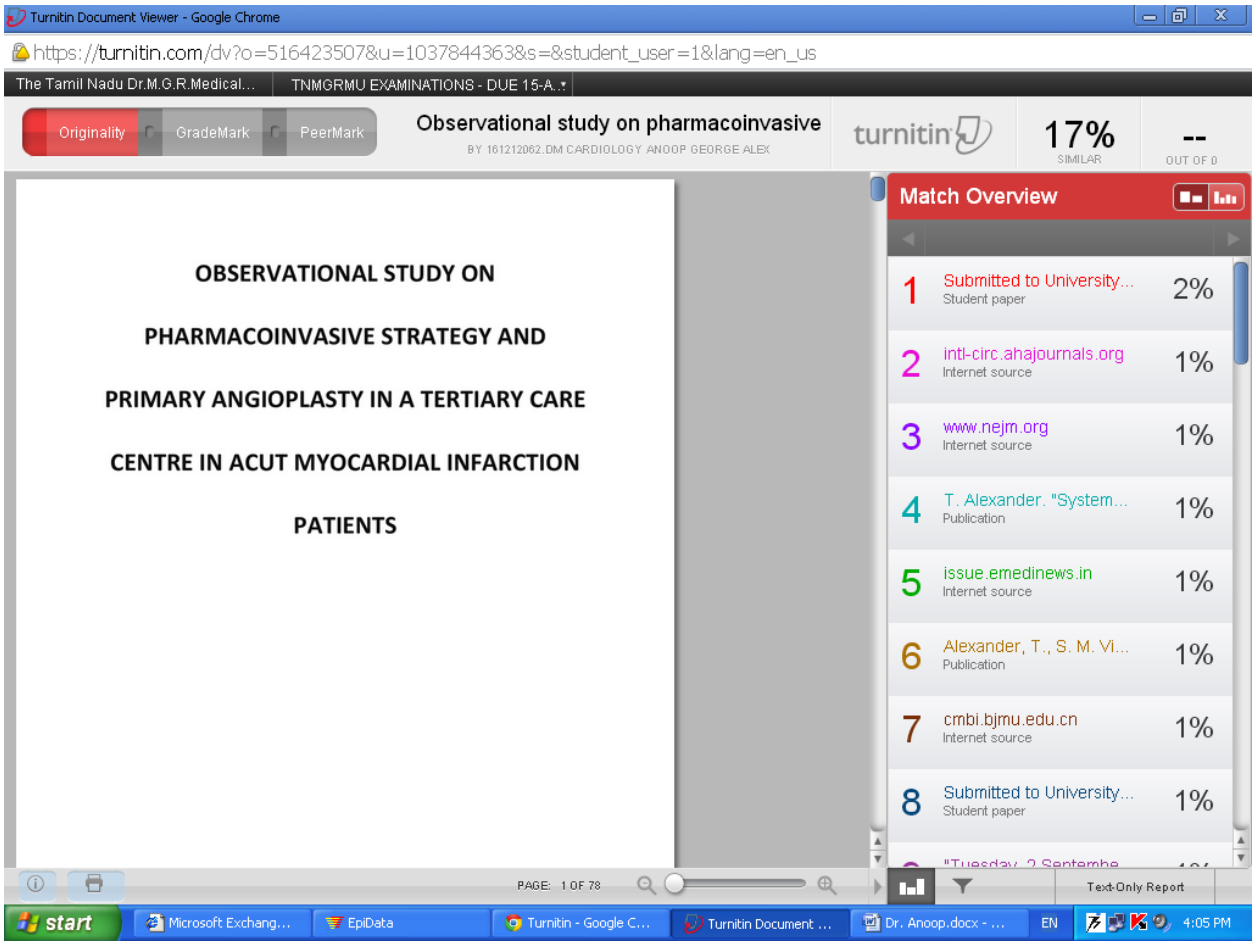
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ABSTRACT

Aims & Objective:

To compare the efficacy of Pharmacoinvasive therapy and Primary Angioplasty in STEMI patients. The objective was to to study whether the incidence of composite end points (mortality, cardiogenic shock and re-myocardial infarction) in Pharmacoinvasive strategy is non inferior to Primary Angioplasty in patients with STEMI.

Background:

In STEMI patients the treatment of choice is primary PCI. (ACC/AHA Class IA recommendation). But in real world situations, timely PCI remains a challenge especially in developing countries like India where the number of centres performing PCI are few. Timely transfer to such centres also remains a huge challenge considering the dearth of emergency ambulance services and the state of our road infrastructure.

Pharmacoinvasive strategy refers to routine angiography with a view to revascularize the infarct related vessel 3-24 hours after fibrinolysis. ACC/AHA has given a class IIa recommendation, while European society of Cardiology has given a class I recommendation for this strategy. We will look whether pharmacoinvasive strategy is non inferior to primary PCI (as proved in various trials) in a setting like ours where streptokinase is used as a fibrinolytic agent as compared to the western countries where these trials were conducted using tenecteplase as a fibrinolytic agent.

Methods:

137 patients were included in this study. All patients admitted with a diagnosis of STEMI at our centre, within a window period of 24 hours over a period of 9 months, who underwent PCI or pharmacoinvasive therapy was included in this study. Primary end points (death within 30 days, re-MI within 30 days, and cardiogenic shock) and secondary endpoints (arrhythmias, bleedingmanifestations, ischemic stroke, ejection fraction, mechanical complications, duration of hospital stay) were looked into.

Results:

Though the hypothesis was non-inferiority, analysis of composite of primary endpoint and mortality outcome suggested equivalence. There was no significant difference between the secondary outcomes between the two groups. Use of thrombus aspiration device and in turn the thrombus burden was significantly lower in the pharmacoinvasive group than in primary PCI arm. Pharmacoinvasive arm had significantly greater number of patients who presented with Anterior wall MI.

Conclusion:

This is a small study, which showed that pharmacoinvasive therapy is as effective as primary PCI in the setting of STEMI. It can also be further inferred that, pharmacoinvasive strategy using streptokinase, is an effective alternative to tenecteplase, in our setting with no associated increase in adverse events.

ABBREVIATIONS

- STEMI – ST Elevation Myocardial Infarction
- PCI - Percutaneous Coronary Intervention
- ACS – Acute Coronary Syndrome
- CCU – Coronary Care Unit
- CPU – Chest Pain Unit
- CAD - Coronary Artery Disease
- MR - Mitral Regurgitation
- VSR - Ventricular Septal Rupture
- CVD – Cardio Vascular Disease

AIMS &OBJECTIVE

AIM:

To compare the efficacy of Pharmacoinvasive therapy and Primary Angioplasty in STEMI patients.

OBJECTIVE:

To study whether the incidence of composite end points (mortality, cardiogenic shock and re-myocardial infarction) in Pharmacoinvasive strategy is non inferior to Primary Angioplasty in patients with acute MI.

LITERATURE REVIEW

Introduction

Amongst the spectrum of non-communicable diseases in India, Coronary artery disease (CAD) is the commonest, estimated to affect over 65 million people ,by the year 2015(1). Acute coronary syndrome (ACS) is comprised of Non ST elevation Myocardial Infarction (NSTEMI) and Unstable angina,(UA) and ST elevation MI (STEMI). One of the gravest complications of Coronary artery disease is STEMI which involves complete disruption of blood flow in the culprit vessel. Timely and complete restoration of blood flow in the culprit vessel helps to reduce the size of the infarct, thereby preserve viable myocardium and LV function and improve the rate of survival.(2)

Coronary reperfusion in the setting of STEMI is established mainly by these two modalities

1) Pharmacological (Fibrinolysis)

2) Mechanical (Primary PCI)

The goal of treatment is to reduce the ischemic time which refers to the period between the symptom onset and administration of reperfusion strategy which will result in restoration of antegrade blood in the culprit vessel.

Pharmacological strategy:

Fibrinolytic therapy assumed significance in the acute coronary syndrome management, as thrombus played a central role in the genesis of acute coronary occlusion.

GISSI-1 and ISIS-2 trials established that Streptokinase as a fibrinolytic agent improved mortality in the setting of ST elevation MI. Fibrin specific plasminogen activators (Tenecteplase, Reteplase) showed enhanced TIMI III flow rates and decreased re-occlusion rates as compared to streptokinase which contributed to improved 30 day mortality rates as shown in GUSTO -1 trial. (As compared to STK, administration of tissue plasminogen activators resulted in reduction of disabling stroke /death in nine out of one thousand patients treated, with a 0.2% absolute excess in incidence of intracranial haemorrhage)

Prehospital Fibrinolysis:

Meta analysis of six randomized trials compared in hospital fibrinolytic therapy and pre hospital fibrinolysis, for acute myocardial infarction patients they showed that the all cause mortality rates were lower in the pre hospital fibrinolysis group. CAPTIM trial, comparing the prehospital firinolysis group and primary PCI group, did not show any significant difference in the incidence of primary end point between them. (10) One explanation to this phenomenon was that with passage of time the thrombus will get more organized and will be more resistant to fibrinolytic therapy. Studies have shown that fibrinolysis is as beneficial as Primary PCI if administered within a window period of 3 hrs.

Primary PCI:

It was in 1979, that Rentrop and colleagues reported their experience with balloon angioplasty to open infarct related occluded arteries in 7 patients. The trials that followed comparing fibrinolysis with primary PCI showed encouraging results with significant decrease in mortality figures among the patients subjected to primary PCI. (3,4) These trials are of limited utility to current practice for the following reasons:

1) In these trials, balloon angioplasty was done and stenting was not done as against the current practice of stenting being done in most of primary PCI procedures.
2) There has been a qualitative jump in the fibrinolytic regimen, subsequently the use of which improved the TIMI flow rates and resulted in reduction of restenosis. These new generation fibrinolyitics were not used in these trials.
3) Similarly there has been considerable progress in the antiplatelet and anticoagulation regimen in the subsequent years that has improved the outcomes in the PCI group.

Clinical efficacy of primary PCI with balloon angioplasty in acute STEMI is limited by the risks of early reclusion and late restenosis, providing the rationale for the use of intracoronary stents. DANAMI-2,AIR PAMI,STOPAMI-1&2, PRAGUE-2, STAT are a few of the randomized trials which have demonstrated a better outcome with primary PCI and stenting compared to fibrinolysis (4-7).These trials used bare metals stents along with PCI. But the trials that followed, comparing drug eluting with bare metal stents pointed to

decreased target vessel revascularisation, cardiac death or MI and decreased rate of stent thrombosis, in the drug eluting stent group.(8-9)

In STEMI patients the treatment of choice is primary PCI. (ACC/AHA Class I A recommendation). But in real world situations, timely PCI remains a challenge especially in developing countries like India where the number of centres performing PCI are few. Timely transfer to such centres also remains a huge challenge considering the dearth of emergency ambulance services and the state of our road infrastructure. In such a scenario Pharmacoinvasive strategy assumes significance

Pharmacoinvasive strategy:

Primary PCI though , the preferred treatment in STEMI is not implemented on a routine basis to STEMI patients due to the practical difficulties encountered in doing so, due to various reasons that will be subsequently discussed. This coupled with the benefits of early pre hospital fibrinolysis and the importance of time to reperfusion, irrespective of the treatment strategy used served as the basis for development of a unified approach in management of STEMI patients.

Pharmacoinvasive strategy refers to routine angiography with a view to revascularize the culprit vessel 3-24 hours after fibrinolysis. Various trials have tried to compare Pharmacoinvasive strategies with existing modalities of reperfusion (11-14). These trials namely GRACIA-1, CRESS-in-AMI, TRANSFER-AMI, STREAM,STEP-AMI TRIAL- have highlighted the advantages of this therapeutic strategy.

In patients presenting with STEMI, for whom Primary PCI cannot be done with less than ninety minutes of door to balloon time or less than one twenty minutes of First medical contact to device time, pharmacoinvasive treatment strategy seems to be an ideal alternative. ACC/AHA has given a class IIa recommendation, while European society of Cardiology has given a class I recommendation for this strategy.

In our centre where we have a dedicated chest pain unit we offer primary PCI in all patients presenting with STEMI. If more than 90 minute's delay in door to balloon time is anticipated (to arrange finances for primary PCI or to produce the Tamil Nadu Government Insurance scheme) we thrombolyse these patients with Streptokinase and do coronary angiogram once the finances or insurance card is ready.

Here we plan to do an observational study on Pharmacoinvasive strategy as compared to primary PCI in tertiary care centre like ours situated in rural India. **We will look whether pharmacoinvasive strategy is non inferior to primary PCI (as proved in trials mentioned above) in a setting like ours where Streptokinase is used as a fibrinolytic agent as compared to the western countries where these trials were conducted using Tenecteplase as fibrinolytic agent.**

Global Burden:

Cardiovascular disease (CVD) accounts for 30% of all deaths in the world (15, 16). The World Health Organization (WHO) noted that CVD is not limited by manmade boundaries. CVD is the leading cause of death in developing countries and is not only a phenomenon that is confined to the developed world. 80% of CVD deaths occur in middle and low-income countries (17)

When compared to Chinese or European's, the propensity among South Asian's to have adverse clinical events is higher, even after adjusting for the degree of atherosclerosis and all other known risk factors. One hypothesis promulgated to reason out this phenomenon was that among south Asians there is a higher chance for plaque rupture and thrombotic events.

Amongst the spectrum of non-communicable diseases in India, Coronary artery disease (CAD) is the commonest, estimated to affect over 65 million people ,by the year 2015(1). In the last 40 years, the incidence of CVD in India has multiplied 4 times. It's estimated that, Indians will constitute almost 60% of patients with CVD in the whole world by 2020. (18)

Acute coronary syndrome (ACS) is comprised of Non ST elevation Myocardial Infarction (NSTEMI) and Unstable angina, (UA) and ST elevation MI (STEMI)ST elevation Myocardial Infarction (STEMI).In the industrialized world STEMI constitutes one of the biggest public health and its incidence is on the rise in developing countries.

The proportion of patients with ACS events diagnosed to have STEMI varies across observational studies – from 29% to 47%. This does not include Silent MI cases. The burden of MI in developing countries may be approaching that now afflicting developed countries.

The incidence of CVD in India may be underestimated due to the following factors

- Few mortality studies from India
- Filling of death certificates is either not done or is incomplete, in majority of the cases
- Absence of a centralized death registry specifically for CVD.

However, according to the World Bank and WHO estimates, there has been the rise in CVD deaths in India was proportional to its expanding population base. It is estimated that a large proportion of disability adjusted life years (DALY) lost in India could be attributable to CVD (19)

Declining Infant mortality with improving trends in life expectancy (which was 41 years from 1951 to 1961, expected to reach 72 years by 2030) and per capita income in India is expected to increase the prevalence of CVD in India in the times to come. (20)

UNIVERSAL DEFINITION & TYPES OF MI:

The existing criteria for definition of MI (2000) were refined by the Joint Task Force of the European Society of Cardiology, the American Heart Association, the World Heart Federation American College of Cardiology Foundation and (ESC/AHA/WHF/ACC) in 2007. Acute Myocardial Infarction was defined as an "event consequent to the death of cardiac myocytes (myocardial necrosis) that is caused by ischemia (as opposed to other etiologies such as myocarditis or trauma)" (21). The third universal definition of MI released in 2012 by the ESC/ACCF/AHA/WHF was almost the same as above.

According to the third universal definition, any one of the following criteria meets the diagnosis of MI:

Detection of a rise and/or fall of cardiac biomarker values (preferably cardiac troponin [cTn] with at least one value above the 99th percentile upper reference limit [URL]) and with at least one of the following:

- Symptoms of ischemia
- Electrocardiogram showing pathological Q waves.
- ST-T changes which is new or presumably new or new onset LBBB.
- Recognition an intracoronary thrombus by autopsy or angiography.
- Imaging evidence showing new regional wall motion abnormality or new loss of viable myocardium.

- Cardiac death associated with symptoms of myocardial ischemia and presumed new Left bundle branch block or new ischemic ECG changes, death occurring before cardiac biomarkers could be taken or before it increased in the system.
- Percutaneous coronary intervention related MI was defined by rise of biomarker values (cTn is preferred) greater than 5 x 99th percentile URL) in patients with normal baseline values (greater than 99th percentile URL) or a rise of values >20 percent if the baseline values are elevated but stable or falling. In addition, either (i) ST-T changes which is new or presumably new or new onset LBBB, (ii) symptoms of myocardial ischemia, (iii) angiographic loss of patency of a major coronary artery or a side branch or persistent slow- or no-flow or embolization, or (iv) Imaging evidence showing new regional wall motion abnormality or new loss of viable myocardium.
- Myocardial infarction secondary to stent thrombosis when detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and/or fall of cardiac biomarkers with at least one value above the 99th percentile
- Coronary artery bypass graft surgery - associated MI was defined by elevation of cardiac biomarker values greater than 10 x 99th percentile URL in patients with normal baseline cTn values. In addition, either (i) Electrocardiogram showing pathological new Q waves or new Left bundle branch block, (ii) angiographic documented new graft or native coronary artery occlusion, or (iii) imaging evidence showing new regional wall motion abnormality or new loss of viable myocardium.

A clinical classification developed by the joint task force, based on the assumed cause of myocardial ischemia is as follows.

Type 1 (spontaneous MI): MI secondary to a pathologic process like rupture, erosion, dissection or fissuring affecting the wall of the coronary artery resulting in intraluminal thrombus.

- Type 2 (MI caused by imbalance secondary to ischemia): Myocardial infarction secondary to oxygen demand supply mismatch. (e.g.,coronaryendothelial dysfunction, coronary artery embolus, coronary artery spasm,tachy/brady-arrhythmias, respiratory failure, hypotension, or hypertension, anemia).
- Type 3 (MI resulting in mortality prior to the availability of biomarkers): Sudden cardiac death prior to the appearance of biomarkers in blood or the blood samples could be drawn for analysis.
- Type 4a (Myocardial infarction related to PCI)
- Type 4b (Myocardial infarction related to stent thrombosis)
- Type 5 (Myocardial infarction related to CABG)

PATHOGENESIS:

The following terms must be understood in some detail before the description of the pathogenesis of atherosclerosis.

Intimal thickening and intimal xanthoma — The pre-existing lesions occurring secondary to blood flow, also described as adaptive intimal thickening,(observed at branch points soon after birth) and intimal xanthoma ("fatty streaks") are considered to be the earliest manifestations of atherosclerotic disease. Studies have shown that intimal thickening are likely precursors for symptomatic atherosclerotic disease. This is so because the location of these lesions in children and adults were similar. Few plaques which may begin as fatty-streaks, are known to regress with time. (22). Histologically, inflammatory cells are not present in intimal thickening. Intimal thickening consists mainly of proteoglycan-collagen matrix and smooth muscle cells. Branch points of vessels may show fatty streaks, which occur due to the accumulation of macrophages within the intima and they regress with time (23).

Pathologic intimal thickening — Extracellular lipid pools consisting of proteoglycans without necrotic debris characterizes the stage between intimal thickening/initial xanthoma and more advanced fibroatheromas. This stage is called pathologic intimal thickening (24). Pathologic intimal thickening can be a precursor to plaque erosion and the development of complications attributed to luminal thrombi.

Fibrous cap atheroma — Fibrous cap atheroma (fibroatheroma) constitutes one of the first stages of lesions in the coronaries (25). It is characterized by a necrotic core rich in lipids, encapsulated by fibrous tissue. Necrosis with macrophage infiltration in the region of lipid pool, constitutes one of the first stages. This is followed by focal loss of proteoglycans or collagen. The accumulation of free cholesterol is not readily apparent. In late stages of fibroatheromas, accumulation of cellular debris, rise in levels of free cholesterol, and almost complete absence of extracellular matrix are seen. The fibrous cap atheroma may progress to a lesion causing significant luminal stenosis following episodes of hemorrhage with or without calcification and surface disruption.

Thin-cap fibroatheroma (vulnerable plaque): The thin-cap fibroatheroma or the vulnerable plaque, as it is commonly mentioned as, consists of a large necrotic core (encompassing almost 25 percent of plaque area) containing a thin fibrous cap separating the lumen and the necrotic. The fibrous cap measured < 65 μm in thickness. The fibrous cap predominantly consists of macrophage infiltrates and also T-lymphocytes, though to a lesser extent. Typically, the fibrous cap has scarce smooth muscle cells or are devoid of those. This lesion is considered a prelude to rupture (26)

The vast majority of thin-cap fibroatheromas (over 80 percent) in sudden coronary death victims have <75 percent cross-sectional luminal-narrowing (or <50 percent diameter stenosis) (27).

Most acute coronary syndromes result from the loss of integrity of the protective covering over an atherosclerotic plaque. Following mechanisms have been put forth to explain this transformation.

- Plaque rupture
- Plaque erosion or
- Calcified nodule.

The loss of the protective endothelial covering allows blood to come in contact with the highly thrombogenic contents of collagen and/or necrotic core of the plaque and allows luminal thrombosis to occur. Other mechanisms can cause acute coronary syndromes, including a supply-demand mismatch. (28,29)

Autopsy studies in sudden cardiac death and myocardial infarction patients have shown that when intra luminal thrombi are identified, the underlying pathology 55 to 75 percent of the time is plaque rupture, 25 to 40 percent of the time is plaque erosion and calcified nodules in 2-7% of patients. (30)

The precursor lesion of plaque rupture has been designated as thin-cap fibro atheroma, which is a descriptive term that was traditionally called a vulnerable plaque. (as described earlier). In around 70% of cases acute coronary thrombi occurs secondary to plaque rupture. Lesions that have large necrotic cores with a thin disrupted fibrous cap characterize it. Once necrotic core comes in contact with blood in the lumen, it will result in the cascade of thrombus formation.

While the precise pathogenic mechanism of plaque rupture is unclear, the following theories have been promulgated.

- Metalloproteinase causing destruction of extracellular matrix within the fibrous cap
- Macrophage apoptosis and the influence of local rheological forces

There was marked elevation of C-reactive protein in patients with plaque rupture, suggesting an underlying systemic inflammatory process that progressively leads to cap thinning and rupture. High Myeloperoxidase levels were seen in the patients with erosion, which was thought to trigger thrombus formation at the site of an eroded plaque expressing hyaluronan. Finally, among patients who did not exhibit features of fissure or erosion of the culprit plaque, cystatin c levels were higher, suggesting underlying functional mechanisms in the microcirculation or in epicardial coronary arteries.

Its interesting to note that plaque rupture, which is thought to be the cause of acute coronary syndromes in around 70% cases, occur in lesions with <50 percent diameter stenosis. Plaque progressions leading to obstructive lesions are a result of repeated ruptures, which may or may not be silent.

PSYCHOSOCIAL FACTORS IN MI:

A circadian variation was observed in the frequency of sudden cardiac death, MI and myocardial ischemia, which was characterized by a morning peak. (31) This may suggest the role of sympathetic nervous system, in triggering cardiac events. Acute coronary syndromes have been attributed to acute and chronic emotional distress, stressful life events, and disasters such as earthquakes. In addition, studies have found that personality type; depression, anxiety, and anger were risk factors for cardiovascular disease.

There are several hypothesized mechanisms by which emotional stress might trigger an acute MI. One such hypothesis points to the role of mental stress causing increase in heart rate, vascular tone, blood pressure, platelet aggregability (32). Mental stress or anger produces transient pressure surges or vasoconstriction, which can disrupt a vulnerable atherosclerotic plaque. Initiating the cascade of thrombosis resulting in an acute coronary syndrome. If disruption is major it will result in STEMI, leading to complete cessation of ante grade blood flow. If the disruption is minor, it may lead to non-occlusive thrombosis. In this setting, the patient may be asymptomatic or develop NSTEMI or unstable angina.

Depression and MI:

Depression is a risk factor for cardiovascular disease, both for cardiovascular patients and among previously healthy individuals. Several potential mechanisms have been proposed which include hypothalamic-pituitary-adrenal axis dysfunction, inflammatory and pro-thrombotic changes, decreased omega-3 fatty acid levels, diet related factors, reduced heart rate variability and adverse behavior including medication non-adherence, smoking and physical inactivity (33). Major depression develops in almost 20 percent of patients after MI, and is associated with poor compliance with recommendations for post MI therapy. In addition, a case-control study found that patients who suffered an MI and had no history of psychiatric illness were more than three times as likely to commit suicide within one month of the MI, compared with individuals who had no history of MI or psychiatric illness.

Spontaneous remission of depressive symptoms occurs in approximately one-half of cases of post-MI depression; the other cases either persist or remit and relapse within one year. Psychosocial interventions for treatment of depressive symptoms have shown little benefit for cardiovascular outcomes.

Seasonal pattern — A seasonal pattern was observed in mortality secondary to myocardial infarction, with more fatal events occurring in the winter than the summer (20 to 30 percent variation) (34). However, in diabetics or those taking beta blockers or aspirin such a seasonal pattern was not seen suggesting an important role for the sympathetic nervous system (35)

STEMI – MANAGEMENT:

It is estimated that 25 to 35% of patients suffering myocardial infarction, succumb, most often from ventricular fibrillation, before getting adequate medical attention (36). The prognosis is considerably better for those patients who reach a medical facility. Fibrinolysis or PCI, which constitute the initial management of STEMI, has resulted in the decline in mortality rates among patients with myocardial infarction with ST-segment elevation. The rate of in-hospital mortality was 5.7% among those receiving reperfusion therapy, as compared with 14.8% among those who were eligible for but did not receive such therapy as shown in an analysis by the National Registry of Myocardial Infarction (37). According to ACC/ AHA, short term mortality rates of patients with STEMI ranges from 5-6% during initial hospitalization and from 7-18% at 1 year

One of the gravest complications of Coronary artery disease is STEMI, which involves complete disruption of blood flow in the culprit vessel. Timely and complete restoration of blood flow in the culprit vessel helps to reduce the size of the infarct, thereby preserve viable myocardium and LV function and improve the rate of survival.(2)One of the key challenges in clinical practice is to shorten the time from symptom to reperfusion and choosing the optimal reperfusion strategy.

Coronary reperfusion in the setting of STEMI is established mainly by these two modalities

1) Pharmacological (Fibrinolysis)

2) Mechanical (Primary PCI)

The goal of treatment is to reduce the ischemic time which refers to the period between the symptom onset and administration of reperfusion strategy which will result in restoration of antegrade blood in the culprit vessel.

PHARMACOLOGICAL STRATEGY:

Fibrinolytic therapy assumed significance in the acute coronary syndrome management, as thrombus played a central role in the genesis of acute coronary occlusion. Fibrinolysis restores blood flow to the ischaemic myocardium by recanalising the thrombotic occlusion, thereby limiting the infarct size. The greatest potential for long term improvement in survival was observed in those patients in whom fibrinolytic therapy was administered within 1-2 hours of symptom onset.

Among myocardial infarction patients (either STEMI/ LBBB), in comparison to conservative management, fibrinolytic therapy improved left ventricular systolic function and survival. Rate of death among patients receiving fibrinolysis was 9.6% at 35 days, in a pooled analysis of nine large trials, as compared to 11.5% among control subjects. (38)
Mechanism of Action: All fibrinolytic agents exert their effect by converting proenzyme plasminogen to active form plasmin.

First Generation

Streptokinase:

GISSI-1 and ISIS-2 trials established that Streptokinase as a fibrinolytic agent which improved mortality in the setting of ST elevation MI. Streptokinase is isolated from bacteria and hence is antigenic. Potential disadvantages also include need for IV infusion, low fibrin specificity, shorter half life, risk of anaphylactic reaction and hemorrhage.

Second Generation

Tissue plasminogen activator is a weak plasminogen activator in the absence of fibrin. But in the presence of fibrin they substantially increase their activity on plasminogen. This happens so because of the arrangement of t-PA and plasminogen in fibrin. Fibrin provides a scaffold in which t-PA and plasminogen are held in a particular way. Catalytic efficiency of t-PA is increased due to this peculiar arrangement.

Alteplase:

Is a second generation fibrinolytic and produces only mild systemic fibrinogen depletion. It is administered over 90 minutes. Though it has fibrin specificity, its use is limited due to the need for IV infusion.

Third Generation:

Third generation agents have increased fibirin specificity, increased resistance to inhibition by plasminogen activators and longer half life.

Reteplase:

Which is a third generation agent is highly fibrin specific and is administered as a double bolus, each dose consisting of 10 units given over two minutes, 30 minutes apart.

Tenecteplase:

Most recently approved for the treatment of STEMI. The most important advantage is that it can be administered as a single bolus over five seconds. Moreover it has high fibrin specificity and is highly resistant to inactivation by plasminogen activator inhibitor, which are all features which contributes to its efficacy.

Fibrin specific plasminogen activators (Tenecteplase, Reteplase) showed enhanced TIMI III flow rates and decreased re-occlusion rates as compared to streptokinase which contributed to improved 30 day mortality rates as shown in GUSTO -1 trial. (As compared to STK, administration of tissue plasminogen activators resulted in reduction of disabling stroke /death in nine out of one thousand patients treated, with a 0.2% absolute excess in incidence of intracranial haemorrhage)

Micro vascular damage in reperfused myocardium leads to haemorragic infarct. Microvascular damage occurs due to platelet microemboli and thrombi. The incidence of haemorragic infarcts is more in patients undergoing fibrinolysis (as fibrinolysis increases the chance of micro embolisation of platelet aggregates due to exposure to clot bound thrombin which is a potent agonist of platelets.) as compared to catheter based reperfusion strategies.

Effect of Fibrinolysis in mortality:

Studies have shown that there is 18% reduction in short term mortality. Overall mortality reduction was to the tune of 25%. LATE and EMERAS trials showed that the mortality benefit extended to those patients undergoing fibrinolysis with a window period of 6-12 hours.. The greatest mortality benefit was observed in patients undergoing fibrinolysis in the first 1-2 hours after the onset of the symptom.

In STEMI patient's maximum benefit of fibrinolysis is obtained if it's started within 30 minutes of symptom onset. This so because, the efficacy of fibrinolysis declines as the time from onset of coronary occlusion to therapy increases. Earlier the institution of thrombolysis, greater will be the benefit. Even out of this 30-minute window period significant benefit is seen. Studies have shown increased survival benefit when fibrinolytic agents were administered within the first four hours after the onset of symptoms (38) and particularly within the first 70 minutes (39).

A mortality benefit is less likely with fibrinolytic therapy at 13 to 18 hours. The benefit of lysis may extend to patients presenting 12 hours after symptom onset and possibly up to 24 hours, if patient has stuttering or ongoing chest pain [40]. Benefits of late reperfusion is thought to be secondary to the presence of a patent infarct related vessel, leading to better ventricular healing, decreased expansion of the infarct, and greater electrical stability.

Limitations of Fibrinolysis:

Despite the above mentioned facts thrombolysis has the following limitations

- Even after timely administration, thrombolysis may not occur in around 15% of patients
- In those for whom successful thrombolysis occur, approximately one fourth of them may have reocclusion of infarct related artery resulting in re-MI
- Thrombolysis may not be a feasible treatment option in all patients due to several contraindications to thrombolysis.
- Increased risk of ICH with fibrinolysis especially in elderly.

Prehospital Fibrinolysis. Meta analysis of six randomized trials that compared in hospital fibrinolytic therapy and pre hospital fibrinolysis, for acute myocardial infarction patients they showed that the all cause mortality rates were lower in the pre hospital fibrinolysis group. (10). One explanation to this phenomenon was that with passage of time the thrombus will get more organized and will be more resistant to fibrinolytic therapy. It's interesting to note here that in the post hoc analysis of CAPTIM Trial, there was a marked trend toward reduction in mortality with fibrinolysis (2.2 versus 5.7 percent) among patients whose treatment assignment was made less than two hours after symptom onset. Other Studies have also shown that fibrinolysis is as beneficial as Primary PCI if administered within a window period of 3 hrs(41)

A meta-analysis of six randomized trials (6434 patients) comparing pre hospital to in-hospital fibrinolysis showed the following benefits (42)

- A shorter time to fibrinolysis (104 versus 162 minutes).
- Reduced all-cause hospital mortality (odds ratio 0.83, 95% CI 0.70-0.98).
- An absolute risk reduction of almost 2 percent that translated into one life saved for every 62 patients treated with prehospital fibrinolysis.

Prehospital and very early fibrinolysis have been compared to primary percutaneous coronary intervention (PCI), but the evidence to support one modality over the other is weak

PRIMARY PCI:

Primary PCI in Acute MI patients is the gold standard of treatment as it establishes a higher percentage of complete and lasting reperfusion. Primary PCI restores angiographically normal flow in the previously occluded artery in more than 90% of patients,(43,44) whereas fibrinolytic therapy does so in only 50 to 60% of such patients.

In a meta analysis of 23 randomized controlled comparisons of primary PCI (involving 3872 patients) and fibrinolytic therapy (3867 patients), rate of death at 4-6 weeks after treatment was significantly lower among those who underwent primary PCI (7% vs 9%)(48)

The concept of catheter based reperfusion for STEMI was introduced in 1979 when Rentrop and colleagues reported pilot experience with balloon angioplasty to open occluded infarct artery in 7 patients. The early trials comparing primary PCI with balloon angioplasty to fibrinolysis showed a significant reduction in mortality with the former (3,4). These trials are of limited utility to current practice for the following reasons:

- Stenting is performed in almost all patients who undergo PCI
- Current generation fibrinolytic agents were not used in many of the trials and
- Anticoagulation and antiplatelet protocols differed significantly from contemporary practice.

The clinical efficacy of primary PCI with balloon angioplasty in acute STEMI was limited by the risks of early reocclusion and late restenosis, providing the rationale for the use of intracoronary stents. Several randomized trials (DANAMI-2, PRAGUE-2, AIR PAMI, STAT, STOPAMI-1, and STOPAMI-2) have demonstrated a better outcome with primary PCI and stenting compared to fibrinolysis (4-7).These trials used bare metals stents along with PCI. But the trials that followed which compared drug eluting stents with bare metal stents showed decreased target vessel revascularisation, cardiac death or MI and decreased rate of stent thronmbosis. (8-9)

Primary PCI is the treatment of choice in patients presenting with STEMI. (ACC/AHA Class I A recommendation). Current guidelines for the treatment of STEMI, recommend a door to balloon time of 90 minutes or less for patients undergoing primary PCI. But in real world situations especially in a developing country like ours timely PCI remains a challenge due to the following factors

- Low levels of literacy, has resulted in decreased awareness about the disease and its early signs and symptoms Due to lack of awareness it takes a long time for the patient to ask for help.
- Number of centres performing PCI are few.
- Timely transfer to such centres also remains a huge challenge considering the dearth of emergency ambulance services and the state of our road infrastructure.

- Delay within a tertiary care contributed by multiple factors like arranging finances obtaining consent, availability of PCI lab in busy hours and the availability of round the clock manpower etc.
- Inadequate private and public health insurance programmes has resulted in very low disposable incomes for most of the families toward their health expenses. Hence arranging finance on an emergency basis for a relatively costly intervention like primary PCI is a 'luxury' that very few people in a developing country like ours, is endowed with.

Pharmacoinvasive treatment assumes significance as a treatment strategy in STEMI patients in such a scenario.

PHARMACOINVASIVE STRATEGY:

Early presentation, rapid diagnosis and early reperfusion in remain the pillars of success in STEMI management. Expedition of each of these factors remains a big challenge and requires policy decisions, the results of which will take years to manifest. But among these, one factor which may have the greatest bearing on the final outcome and for which the treating physician and his team have a big role to play is that of instituting rapid reperfusion strategy.

Moreover it's time to look beyond the conventional goals in the management of STEMI in order to attain further mortality benefit in STEMI patients. The following example illustrates this concept. Studies have shown that though the door-to-balloon times have improved significantly for patients undergoing primary PCI for ST-segment elevation myocardial infarction, it has not reflected in the improvement in mortality, pointing to the fact that the focus should be on additional strategies to reduce in hospital mortality in these patients.(49)

As mentioned above Fibrinolytic therapy and Primary PCI are two commonly used reperfusion strategies which are conventionally viewed as mutually exclusive alternative therapeutic modalities. However great deal of recently acquired clinical evidence support the view that the two in combination are synergistic. The practical difficulties in implementing primary PCI in routine practice along with the evidence of benefit of pre hospital fibrinolysis (especially if administered early) and the overarching importance of time to reperfusion, regardless of the strategy used, serve as the foundation for development of a unified approach to management of patients with STEMI.

This Synergistic or unified approach is referred to as Pharmaco- invasive therapy.(50-52)

"Pharmacoinvasive strategy refers to routine angiography with a view to revascularization of infarct related artery 3-24 hours after fibrinolysis.

Relevance of Pharmaco-invasive approach

Early firinolytic therapy has documented benefits of 65, 37, 26 and 29 lives saved per 1000 treated patients in 0-1 hour, 1-2 hour, 2-3 hour respectively. This is so because "Time" is a crucial factor in STEMI care. The risk of 1 year mortality was increased by 7.5% for each 30 minute delay in treatment(53,54), confirming to the age old adage of time is muscle.

Prompt fibrinolytic treatment improves the likelihood of aborted myocardial infarction and the greatest incidence occurred in patients who underwent lysis within 1 hour of symptom onset with a sharp drop off after 3 hours.(55)

A delay in undergoing primary PCI results in reduced benefit of the invasive procedure Nallamothu et al. in his published studies highlighted the fact that mortality benefit associated with Primary PCI was lost if PCI-related delay exceeded 60 min.(56), pointing to the theoretical assumption of early fibrinolytic therapy compensating for PCI related delay.

CREATE and Kerala ACS Registry shows that there is significant delay in patient presentation, and initiation of timely reperfusion. Pharmaco-invasive approach helps to shorten the time to reperfusion of infarct related artery by initiation of lysis. PCI in 3-24 hours helps to consolidate the initial reperfusion process and prevent reocclusion of the infarct related artery.

Various trials have tried to compare Pharmacoinvasive strategies with existing modalities of reperfusion (11-14)

GRACIA-1: Included 500 patients randomized to either delayed PCI (6-24hrs after fibrinolysis) or to an ischemia guided conservative approach. The former approach was associated with a reduction in mortality, reinfarction and revascularization rates at one year including favourable trends for mortality and reinfarction

CARESS – in – AMI: Demonstrated that strategy of routine angiogram and revascularization after PCI was better than recue only angioplasty after fibrinolysis

TRANSFER – AMI Trial: Compared pharmacoinvasive strategy with standard treatment (including Rescue PCI if required or if required or delayed angiography). The results showed that there was no difference in mortality between the two groups but the pharmacoinvasive strategy was associated with fewer ischaemic complications than the standard treatment group.

STREAM TRIAL: Showed that prehospital fibrinolysis coupled with timely angiography was non inferior to primary PCI in patients presenting with STEMI within 3 hours after symptom onset and who were unable to undergo primary PCI within 1 hour. The earlier administration of fibrinolytic therapy was associated with lower rates of cardiogenic shock and congestive heart failure, more aborted myocardial infarction, and higher patency rates on the initial coronary angiogram, with no differences in reinfarction rates or death.

The initial study was done comparing the composite of primary end points at 30 days. After that the results of a follow up study at one year also showed similar mortality rates between pharmacoinvasive arm and primary PCI arm.

WEST Study: showed that Parmacoinvasive strategy produced results equivalent to Primary PCI.

STEP AMI Trial (57): is the first Indian Trial, a prospective observational, multicentric study evaluating the efficacy and safety of Pharmaco-invasive strategy versus standard primary PCI in 200 patients with acute myocardial infarction within 12 hours of symptom onset .In an interim presentation ahead of publication, STEP P-AMI investigators concluded that pharmaco- invasive strategy was safe and was associated with comparable outcomes as primary PCI at 1 year. Moreover it was associated with more than 80% infarct related artery patency even when the total ischaemic time was about 4 hours. Significant portion of patients did not require stenting. Lower thrombus burden and better TIMI flow was also observed in patients in the pharaco-invasive arm. There was no increase in bleeding risk in patients in pharmaco-invasive arm.

Apart from these various other trials like SIAM III, CAPITAL –AMI, NORDISTEMI also compared pharmaco invasive approach to existing modalities of reperfusion. The hypothesis of Pharmaco-invasive approach is also backed by Metaanalysis (58). Compared to routine treatment after fibrinolysis, early PCI within 24 hrs of fibrinolysis, significantly reduced the composite end point of death, re-infarction

and ischemia within 30 days. The occurrence of severe bleeding episodes was not increased in pharmacoinvasive arm, which may be due to the increased time interval between lysis and coronary angiogram in pharmacoinvasive arm.

All these trials adds more evidence to support the conclusion that a fibrinolytic strategy including early coronary angiography is an excellent therapy for STEMI when primary PCI is not readily available, especially in patients with potentially large myocardial infarct size or when early treatment is possible.

Contemporary Trends in the management of STEMI in India

National registry data from 89 cities suggest that Indian patients with STEMI frequently fail to receive an adequate reperfusion therapy and to a greater extent than comparable patients in the developed countries. (59) For example, reperfusion therapy with fibrinolysis is received by less than 60% of Indian patients with STEMI and those that undergo it often do so after great delays. (59) Furthermore, a few patients go on to early invasive evaluations and less than 10% receive percutaneous coronary intervention (PCI) during their hospitalization despite growing support for this type of pharmacoinvasive approach.

The data about the contemporary trends in STEMI patients in India comes from CREATE Registry and KERALA ACS Registry.

Some of the salient features of **CREATE Registry** are as follows

- Median time from onset of symptom to hospital arrival in STEMI patients was 300 minutes as compared to 140-170 minutes in developed countries.
- Door to needle time was 50 minutes as compared to 32-40 minutes in developed countries.
- Mode of transport for the majority was through private transportation. Only 5% of patients utilized ambulance.
- Approximately 59% received fibrinolytic therapy and only 9% underwent percutaneous coronary intervention during hospitalization, suggesting substantial room for improvement in the use of acute reperfusion therapy for STEMI patients in INDIA

Kerala ACS Registry: documented that

- STEMI constituted the largest chunk of ACS admissions and had the highest in-hospital mortality and non-fatal event rates.
- Patients with STEMI were more likely to present with a window period of more than 6 hours.
- Thrombolysis was used in 41% of STEMI patients
- Only nearly half of STEMI patients received some modality of reperfusion therapy, in comparison to registries in western countries where about 30% of eligible patients did not receive reperfusion therapy.

Thus Pharmacoinvasive strategy seems to be an ideal alternative to Primary PCI in patients presenting with STEMI in whom Primary PCI cannot be done with a door to balloon time of 90 minutes or First medical contact to device time less than or equal to 120 minutes. ACC/AHA has given a class IIa recommendation while European society of Cardiology has given a class I recommendation for this strategy.

In our centre, where we have a dedicated chest pain unit, we offer primary PCI in all patients presenting with STEMI. If a delay of more than 90 minute s (Door to balloon time) is anticipated (for the patient to arrange finances for primary PCI or to produce the Tamil Nadu Government Insurance scheme or for the relatives to take an informed decision in consultation with the family members) we thrombolyse these patients with Streptokinase and do coronary angiogram with the view to revascularize the infarct related artery, once the finances / insurance card is ready .or the decision is made.

In this observational study on Pharmacoinvasive strategy as compared to primary PCI in tertiary care centre like ours situated in rural India, we will look whether pharmacoinvasive strategy is non inferior to primary PCI (as proved in trials mentioned above) in a setting like ours where Streptokinase is used as a fibrinolytic agent as compared to the western countries where these trials were conducted using Tenecteplase as fibrinolytic agent.

MATERIALS & METHODS

a. **Setting:** Coronary Care Unit and Chest Pain Unit of the department of Cardiology.

b. **Participants:**

Inclusion criteria:

1) All patients admitted with a diagnosis of ST elevation myocardial infarction in the CPU/CCU of CMC, Vellore.

2) Within a window period of 24 hours.

3) Over a period of 9 months,

4) Underwent PCI or pharmacoinvasive therapy.

Exclusion criteria:

1) Previous CABG/PCI

2) Contraindication for lysis

3) Severe renal impairment (eGFR <30 ml/min)

4) Patients lysed with Tenecteplase

5) Consent not given by patient/relatives.

Primary PCI arm:

All patients willing for primary PCI and can arrange finances for the same on an urgent basis (<24 hours) will be included in the primary PCI arm.

Pharmacoinvasive arm:

All patients either

- Referred from an outside centre after thrombolysis and who undergo PCI or
- who were initially thrombolysed in our centre

Who subsequently underwent coronary angiogram in our centre with a view to undergo revascularisation of infarct related artery is included in the pharmacoinvasive therapy arm.

c. **Variables:**

The primary end points include

- Death within 30 days
- Re-Myocardial infarction within 30 days, and
- Cardiogenic shock.

The secondary end points are

- Arrhythmias
- Bleeding manifestations (intracranial haemorrhage, gastrointestinal bleeding, mucosal bleeding)
- Ischemic stroke,
- Ejection fraction (measured by ECHO),
- Mechanical complications (MR,VSR, ventricular free wall rupture),
- Duration of hospital stay

Potential confounding factors affecting primary and secondary end points including age, sex, co morbidities, smoking, alcohol, total ischemic time, door to balloon time, mode of transport to our hospital etc will also be looked into.

d. **Data Sources/measurement:**

Data of ST elevation MI patients admitted in CCU and CPU who will be enrolled for this study will be obtained by

- History and Clinical examination
- ECG, ECHO
- Routine blood investigations

Several trials (CARESS-in-AMI, TRANSFER AMI, WEST study, STREAM) have addressed the potential benefit of pharmacoinvasive strategy. In all these trials Tenecteplase was used as the fibrinolytic agent. Due to cost constraints, streptokinase is the fibrinolytic agent that is commonly used in India and in our centre. **Till date, there are no trials comparing pharmacoinvasive strategy using streptokinase with primary angioplasty. Hence, we would like to see whether pharmacoinvasive strategy using streptokinase is also non inferior to primary angioplasty in patients admitted with ST elevation MI getting admitted within a window period of 24 hours, in this cohort study**

RESULTS & ANALYSIS

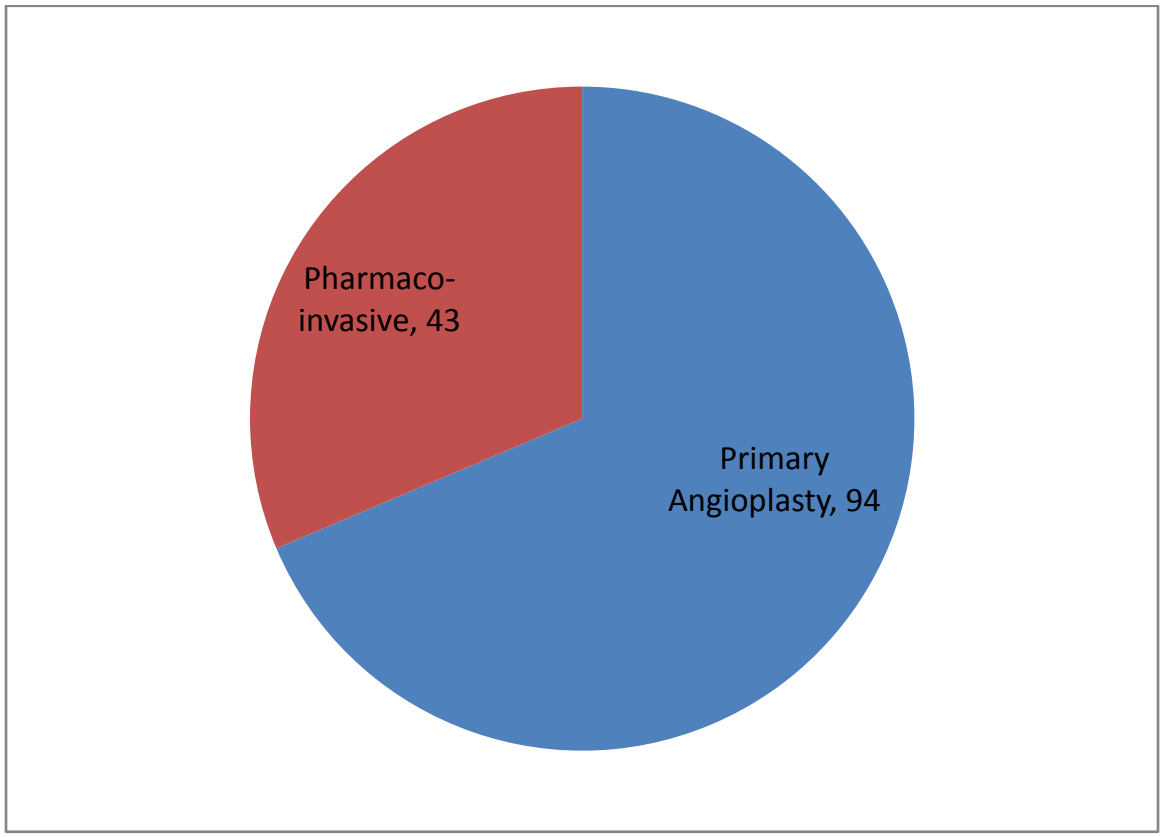

Figure 1: Distribution of STEMI patients in the two treatment arms.

Out of the 147 patients analyzed 94 were in the Primary PCI arm and 43 were in the Pharmacoinvasive arm. (Figure 1)

GENDER DISTRIBUTION

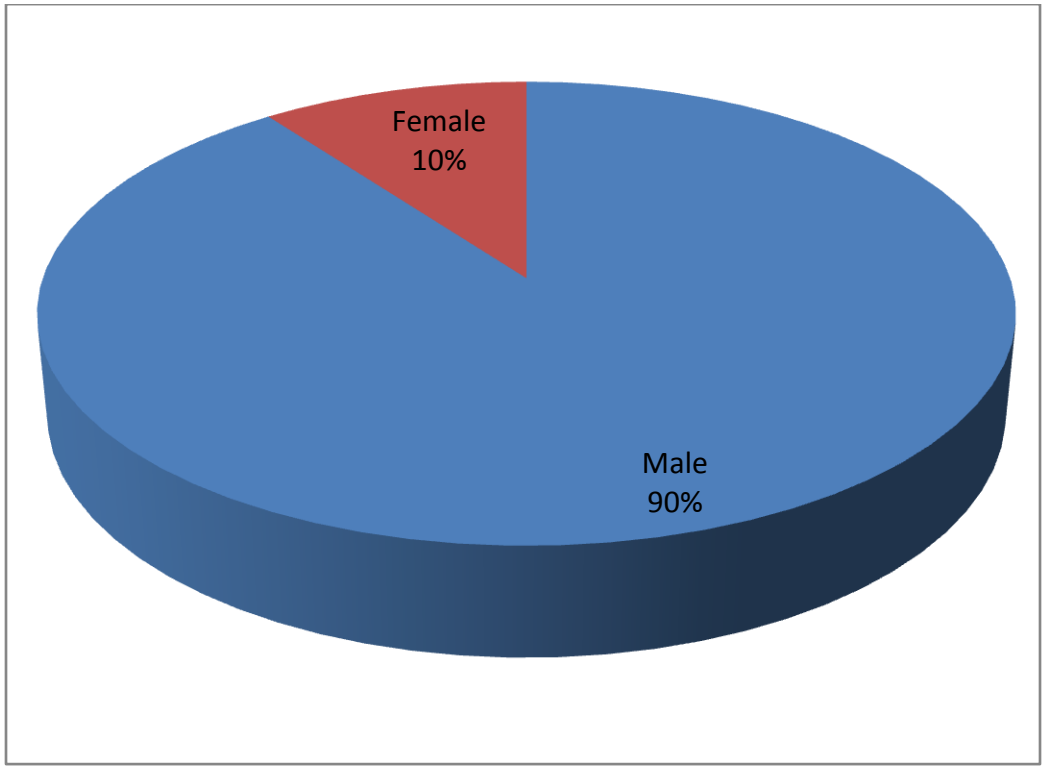

Figure 2: Gender Distribution among STEMI patients.

90% of patients who presented with STEMI were males while females constituted only the rest 10%.

AGE & GENDER DISTRIBUTION

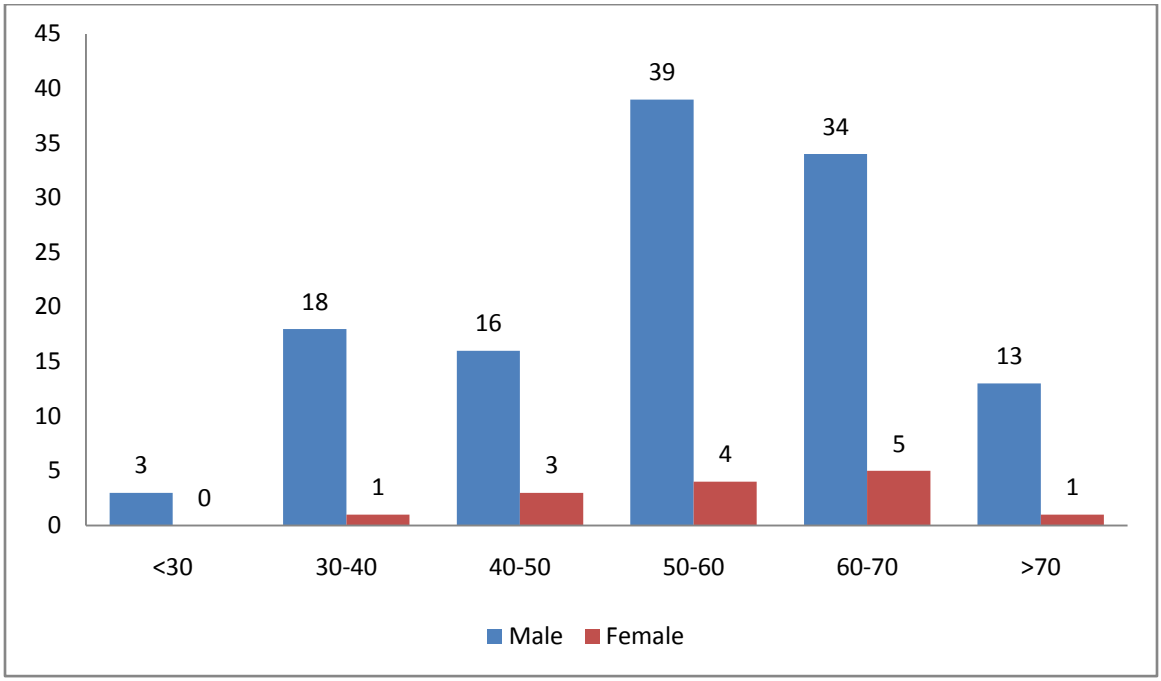

Figure 3: Age & Gender Distribution

As is evident from figure 3, males among the age group between 50-60 years, most commonly, presented with STEMI. Among the females, the most common age group of presentation was in the 60-70 year group.

PRESENTING SYMPTOMS

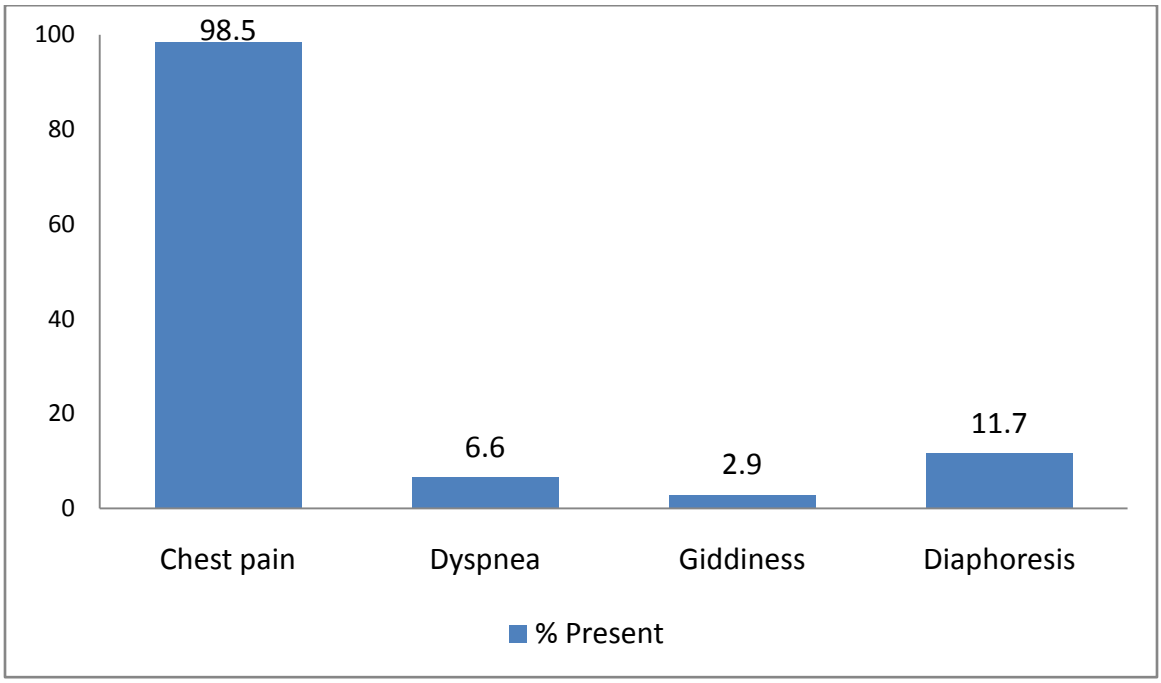

Figure 4: Symptoms at presentation.

98.5 % of patients with STEMI had chest pain as the presenting symptom. Diaphoresis was the next most common symptom at presentation constituting 11.7%. This was followed by dyspnea (6.6%) and giddiness (2.9%

CORONARY RIK FACTORS

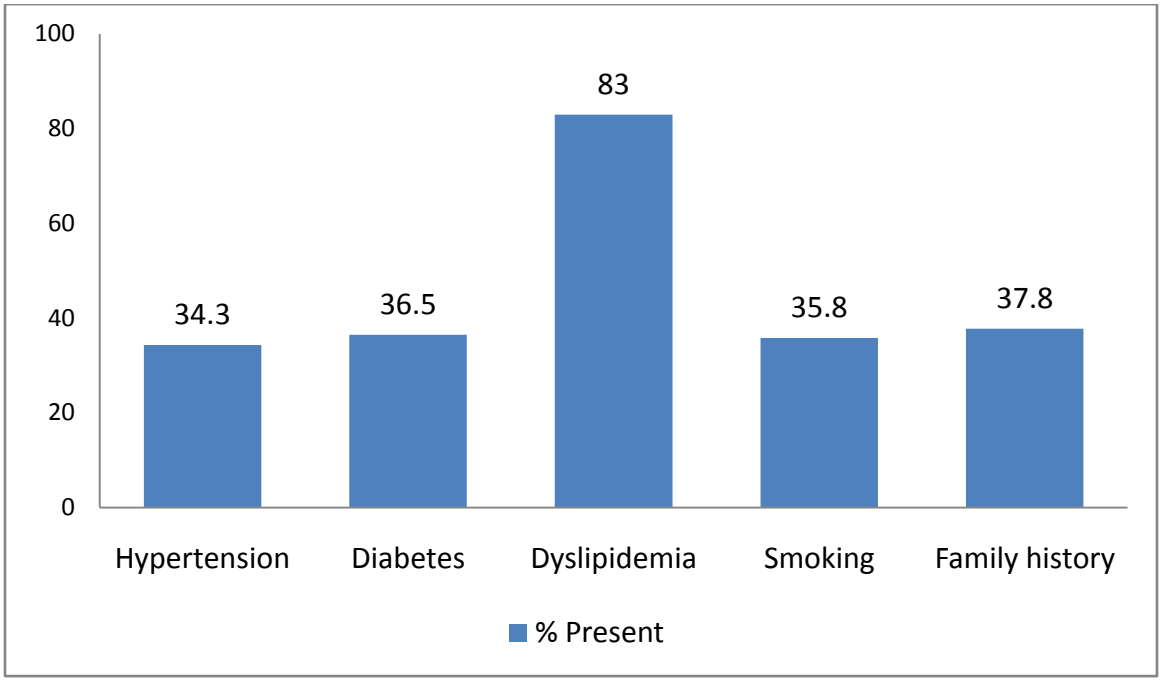

Figure 5: Risk factor profile among STEMI patients.

The most common coronary risk factor that was most common among the patients who presented with STEMI was dyslipidemia (83%), which was followed by family history of coronary artery disease (37.8%). This was closely followed by diabetes mellitus (36.5%), smoking (35.8%), and systemic hypertension (34.3%).

MODE OF TRANSPORT

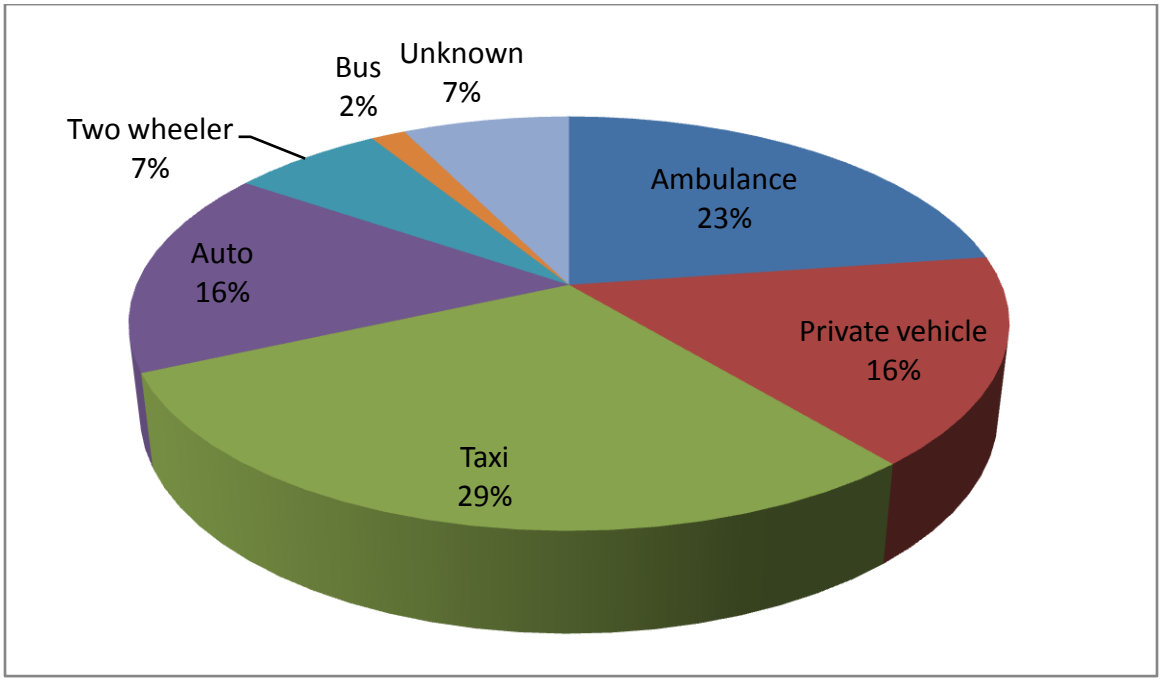

Figure 6: Mode of Transport

As is evident from Figure 6, majority of patients (29%) who presented with STEMI hired a taxi to reach our hospital. 23% of patients were transported in an ambulance while 16% of patients utilized their own private vehicle to come to our center. It's interesting to note that 16% of patients came with STEMI in an auto rickshaw and the mode of transport was a two-wheeler for 7% of patients. 2% of patients utilized bus transport to reach the hospital.

BASELINE CHARACTERESTICS

VARIABLE	PRIMARY ANGIOPLASTY (N=94)	PHARMACO-INVASIVE (N=43)
Age (Mean)	57	52
Sex		
Male	87 (93%)	36 (84%)
Female	7 (7%)	7 (16%)
Killip Class		
Class I	76 (81%)	33 (77%)
Class II	8 (8.5%)	3 (7%)
Class III	1 (1%)	2 (5%)
Class IV	9 (9.5%)	5 (11%)
Heart Rate	79	87
Systolic BP	119	121

Figure 7: Baseline characteristics

The mean age of patients in the primary PCI arm was 57 years (with a range of 23-85 years) and pharmacoinvasive arm was 52 years (with a range of 17-85 years).

Males constituted 93% of patients in primary PCI arm and 84% of patients in pharmacoinvasive arm. Female who presented with STEMI who underwent a revascularization procedure had pharmacoinvasive procedure more commonly than primary PCI.

The percentage of patients in Killips class I was81% in primary PCI arm and 77% in pharmacoinvasive arm. There was no significant difference in the distribution of patients in killips class IV between both the treatment arms (9.5% vs. 11%).

The mean heart rate was 79 per minute among patients in primary PCI arm and 87 per minute in pharmacoinvasive arm.

The mean systolic BP was 119mmHg in primary PCI arm and 121mmHg in pharmacoinvasive arm.

DISTRIBUTION OF CORONARY RISK FACTORS and LOCATION OF STEMI

VARIABLE	PRIMARY ANGIOPLASTY (N=94)	PHARMACO-INVASIVE (N=43)
HTN	34 (36%)	13 (30%)
Smoking	31 (33%)	18 (42%)
Diabetes	37 (39%)	13 (30%)
Family history of CAD	36 (38%)	9 (21%)
Diagnosis		
Anterior	51 (54%)	32 (74.5%)
Inferior	40 (42.5%)	11 (24.5%)
Others	3 (3.5%)	0

Figure 8: Distribution of Coronary Risk factors and location of STEMI

Among the coronary risk factors there was there was no significant difference, in the prevalence of the conventional coronary risk factors between the two arms, as is evident from figure 8.

74.5% of patients in the pharmacoinvasive arm presented with an Anterior wall MI while only 54% of patients in primary PCI arm had Anterior wall MI on presentation. The distribution of inferior wall MI among primary PCI patient was 42.5% whereas in pharmacoinvasive arm was 24.5%. There was a significant difference between the distributions of anterior wall MI between the two strategies (p-0.012)

ACCESS SITE and TIMI FLOW:

VARIABLE	PRIMARY ANGIOPLASTY (N=94)	PHARMACO-INVASIVE (N=43)
Access site		
Radial	91 (97%)	41 (95%)
Femoral	3 (3%)	2 (5%)
TIMI flow		
1	2 (2%)	2 (5%)
2	10 (11%)	5 (12.5%)
3	80 (87%)	33 (82.5%)

Figure 9: Access site and TIMI flow.

91% of patients in primary PCI arm and 95% of patients in Pharmacoinvasive arm underwent revascularization procedure through the radial route.

There was no significant difference between the TIMI flow rates among the treatment arms as is evident from Figure 9.

ANGIOGRAPHIC PROFILE

VARIABLE	PRIMARY ANGIOPLASTY (N=94)	PHARMACO-INVASIVE (N=43)
SVD	55 (58.5%)	28 (65%)
DVD	20 (21.5%)	9 (21%)
TVD	15 (16%)	3 (6.5%)
LM+TVD	2 (2%)	0
LM+DVD	1 (1%)	1 (2%)
LM+SVD	1(1%)	0
Recanalised vessel and not stented	0	3 (6.9%)
Use of thrombus aspiration	69 (73%)	9 (21%)

Figure 10: Angiographic profile of patients in both treatment arms

58.5% of patients in primary PCI arm and 65% of patients in pharmacoinvasive arm had single vessel disease in coronary angiography. The distribution of double vessel coronary artery disease was 21.5% and 21 % respectively in both the arms. 6.5% of patients in pharmacoinvasive arm and 16% of patients in primary PCI arm had triple vessel coronary artery disease (p-0.09). There was no significant difference between the occurrences of left main lesions in both the arms.

It is interesting to note that 6.9% of patients in pharmacoinvasive arm had recanalised vessel and was not stented, while in primary arm no patient had a recanalised vessel on coronary angiography.

Thrombus aspiration device was used in only 21% of patients who underwent pharmacoinvasive therapy as against73% of patients in primary PCI arm suggesting a significant reduction in thrombus burden with thrombolysis with streptokinase. (p <0.001)

LABORATARY VALUES

LAB VALUES	PRIMARY	PHARMACOINVASIVE
Creatinine	0.97	0.93
CKMB	83	89
Troponin T	1667	2672
Serum Sodium	133	135
Serum Potassium	3.8	3.7
Total cholesterol	165	167
Triglyceride	137	146
HDL	39	41
LDL	112	106
Hb1AC	7	6.7
TSH	2.2	1.3

Figure 19: Distribution of Lab values between both the treatment arms

The above table shows the mean values of different laboratory investigations between both the treatment arms. Most of the values were equally matched between both the arms. (P-0.06 for mean troponin values between n=both the arms)

TIME INTERVALS

VARIABLE	PRIMARY ANGIOPLASTY (N=94)	PHARMACO-INVASIVE (N=43)
Pain to door time	5.50 hrs	5.51 hrs
Door to balloon time (mins)	87 min	-
Lysis to CAG (hrs)	-	10.5 hrs
Total ischemic time (mins)	417 min	

Figure 11: Time intervals

Pain to door time is the time period between the onset of pain to the patients' arrival in the hospital

Door to Balloon Time (DBT) is the time in minutes between the arrival of the patient in the hospital and the first balloon inflation or device deployment in his culprit vessel to establish ante grade flow.

Lysis to CAG is the time span between thrombolysis and coronary angiogram in patients in the pharmacoinvasive arm.

Total Ischemic time refers to the time span between the onset of pain to effective restoration of ante grade flow in primary PCI.

The average pain to door for primary PCI patients was 5.50 hours while that of pharmacoinvasive group was 5.51 hours, indicating that the median time delay from onset of symptoms to first medical contact was similar in the two study groups.

The average door to balloon time in primary PCI arm was 87 minutes.

The mean time period between lysis and CAG in pharmacoinvasive arm was 10.5 hours

The total ischemic time counting the duration between onset of pain and ballooning in primary PCI patients and CAG in pharmacoinvasive patients was 417 minutes and 916 minutes respectively.

PRIMARY END POINTS

VARIABLE	PRIMARY ANGIOPLASTY (N=94)	PHARMACO-INVASIVE (N=43)
Primary end points	13 (14%)	3 (7%)
Mortality	5 (5%)	2 (4.5%)
Cardiogenic shock	12 (13%)	3 (7%)
Re MI	1 (1%)	0

Figure 12: Primary end point.

The primary end point (mortality due to any cause, cardiogenic shock and re MI) occurred in 13 out of the 94 patients (14%) in primary PCI group and3 out of 43 (7%) patients in pharmacoinvasive group

If we try to analyze the composite of primary end point further, we see that the mortality percentage in both the arms is almost similar (5 vs. 4.5%). Cardiogenic shock, post procedure occurred in 13% of patients in primary angioplasty group and 7% in pharmacoinvasive arm. Only one patient was admitted with a recurrent MI and that occurred in the primary PCI arm.

SECONDARY END POINTS

SECONDARY END POINTS	PRIMARY ANGIOPLASTY	PHARMACOINVASIVE
Ejection fraction (Mean)	44.3	42.4
Duration of hospital stay	3.83 days	3.62 days
Bleeding manifestations	1 (1%)	0
Ischemic stroke	0	1 (2.5%)
Arrhythmias	2 (2%)	0
Mechanical complications	3 (3%)	1 (2.5%)

Figure 13: Distribution of secondary end points.

There was no significant difference in the occurrence of secondary end points between the two groups. Ejection fraction in primary PCI arm was 44.3% while in pharmacoinvasive arm was 42.4%.

The duration of hospital stay was 3.83 days in primary PCI arm whereas inpharmcoinvasive arm was 3.26 days.

Bleeding manifestation in the form of groin hematoma occurred only in one patient who was in primary PCI arm. Ischemic stroke occurred in one patient who was in pharmacoinvasive arm.

Arrhythmia's occurred in 2 patients both of whom had undergone primary PCI procedure. One patient had recurrent VT requiring DC shock and the other had Ventricular tachycardia and fibrillation

Mechanical complication (Mitral Regurgitation) occurred in 3 patients in PCI arm and 1 patient in pharmacoinvasive arm.

MORTALITY OUTCOME

	Follow up at 30 days				**Total**	**Confidence Interval**
Strategy	**Death**	**%**	**Alive**	**%**		
pharmacoinvasive	2	4.7	41	95.3	43	(-9.09 to 7.75)
Primary	5	5.3	89	94.7	94	
Total	7	5.1	130	94.9	137	

Figure14: Mortality outcome

On analyzing the mortality outcome between the two groups, we see that mortality in pharmacoinvasive arm was 4.7%, whereas the mortality in primary PCI arm was 5.3%.

PRIMARY ENDPOINT

	Primary end point				Total	Confidence Interval
Strategy	**Event**	**%**	**Non-event**	**%**		
Pharmacoinvasive	3	7.0	40	93.0	43	(-18.88 to 5.17)
Primary	13	13.8	81	86.2	94	
Total	16	11.7	121	88.3	137	

Figure 15: Primary end point

In Primary endpoint (composite outcome), the Pharmacoinvasive procedure has nearly 50% less event as compared to Primary procedure

KAPLAN-MEIER CURVES FOR THE PRIMARY END POINT

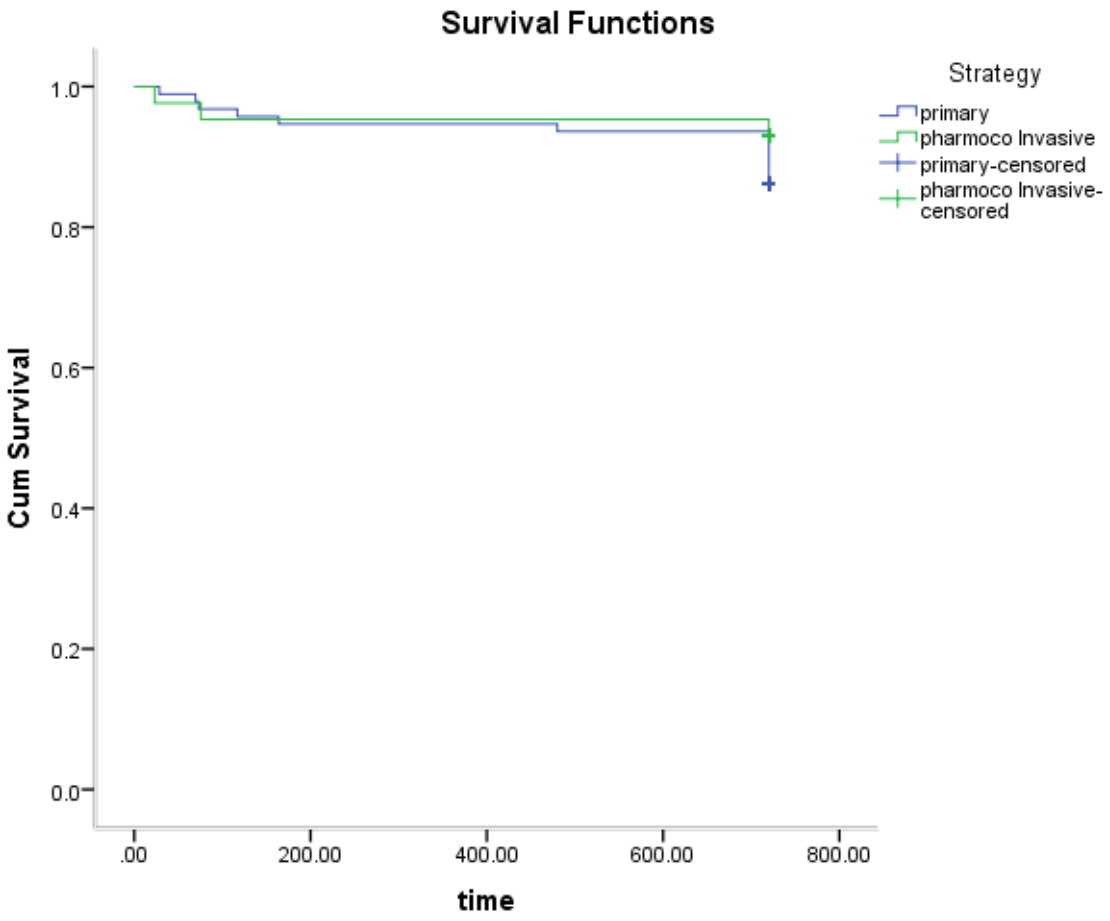

Figure 16: Kaplan – Meier Curve

The primary end point was a composite of death from any cause, shock, congestive heart failure, or re-infarction within 30 days

Relative risk and confidence intervals are provided for Inference

Variables	Primary events	Total	Pharmaco events	Total	Relative Risk	Lower limit	Higher limit
Age							
<75	12	90	3	40	1.778	0.531	5.956
>75	1	4	1	3	0.75	0.072	7.731
Hypertension							
Yes	11	60	3	27	1.65	0.5	5.44
No	2	34	1	13	0.765	0.0756	7.734
Infarct location							
Anterior	9	52	3	28	1.615	0.475	5.489
others	4	38	1	12	1.263	0.156	10.244
Killips Class							
Class I	8	68	1	32	3.765	0.491	28.837
others	5	13	2	8	1.538	0.386	6.135
Gender							
Male	11	87	3	36	1.517	0.45	5.119
Female	2	5	1	7	2.8	0.34	23.058
Smoking							
Smokers	10	63	3	21	1.111	0.337	3.659
Non-smokers	3	28	1	18	1.929	0.217	17.136
Systolic Blood Pressure							
<90	3	11	1	3	0.818	0.126	5.302
>90	10	79	2	37	2.342	0.54	10.156

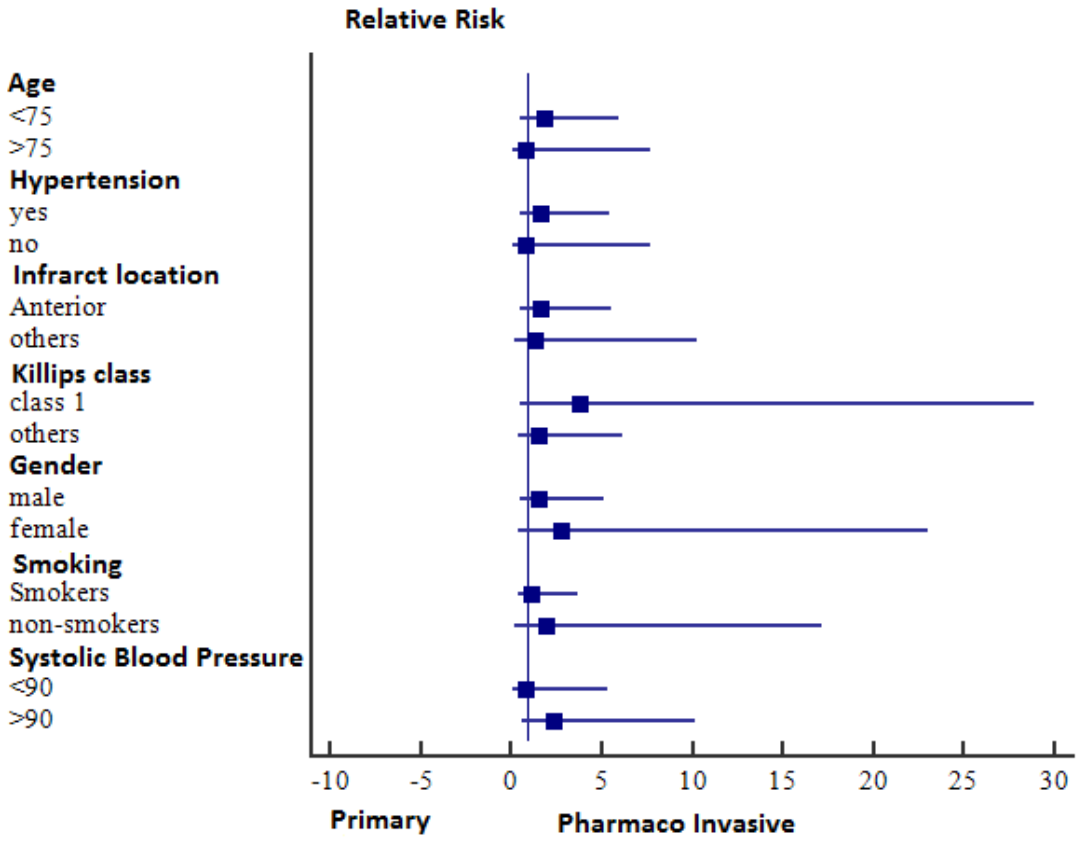

Figure 18:Shown are the rates of primary end point among patients undergoing pharmacoinvasive PCI or primary PCI

DISCUSSION

Introduction:

Coronary artery disease (CAD) is currently the most common, non-infectious disease in India and will affect over 65 million of its people by the year 2015(1). ST elevation Myocardial Infarction(STEMI), Non ST elevation Myocardial Infarction (NSTEMI) and Unstable angina, forms the entire spectrum of Acute coronary syndrome (ACS).One of the gravest complications of Coronary artery disease is STEMI which involves total occlusion of infarct related artery. Timely and complete restoration of flow in infarct related artery helps to reduce infarct size, preserve LV function and improve survival rates(2)

Coronary reperfusion in the setting of STEMI is established mainly by these two modalities

1) Pharmacological (Fibrinolysis)

2) Mechanical (Primary PCI)

Apart from these two treatment modalities, one strategy that has proved beneficial in this patient population is the Pharmacoinvasive strategy. Pharmacoinvasive strategy refers to routine angiography with a view to revascularization of infarct related artery 3-24 hours after fibrinolysis.

The goal of treatment is to reduce the total ischemic time which refers to the time period between the onset of symptoms and administration of reperfusion strategy which will result in restoration of coronary blood flow in the infarct related artery.

In our study, we plan to include all ST elevation MI patients admitted in CPU and CCU who were either taken up for primary angioplasty or who underwent pharmacoinvasive therapy over a period of 9 months. We plan to specifically look into the primary and secondary end points with respect to the above group of patients. The primary end points include death, re-Myocardial infarction and cardiogenic shock. The secondary end points are Arrhythmia's, Bleeding manifestations, Ischemic stroke, Ejection fraction, mechanical complications, duration of hospital stay and post infarction angina.

We will also look into the total ischemic time, door to balloon time, door to needle time, mode of transport to our hospital and demographic profile of patients involve in this study which are probable confounding factors affecting primary and secondary end points.

Before the detailed analysis of the findings of this study it is pertinent to have a detailed discussion with regards to the various modalities used in the treatment of STEMI patients. This includes its mechanism of action, merits, demerits, trials favoring the particular modality involved, and the mortality and morbidity benefit each of these treatment modality offers.

Pharmacological strategy:

Fibrinolytic therapy assumed significance in the setting of acute coronary syndrome given the central role of thrombus in the genesis of acute coronary occlusion. Fibrinolysis restores blood flow to the ischaemic myocardium by recanalising the thrombotic occlusion, thereby limiting the infarct size. Patients treated within the first 1-2 hours of the symptom onset have the greatest potential for long term improvement in survival with fibrinolysis.

Fibrinolytic therapy, in comparison to conservative management improved left ventricular systolic function and survival, in patients with myocardial infarction associated with either ST- segment elevation or left bundle-branch block. All fibrinolytic agents exert their effect by converting proenzyme plasminogen to active form plasmin.

Agents for thrombolysis could be broadly divided into

- First Generation : Streptokinase
- Second Generation: Tissue plasminogen Activator (Alteplase)
- Third Generation: Reteplase and Tenecteplase.

Fibrin specific plasminogen activators (Tenecteplase, Reteplase) showed enhanced TIMI III flow rates and decreased re-occlusion rates as compared to streptokinase which contributed to improved 30 day mortality rates as shown in GUSTO -1 trial. (As compared to STK t-PA led to reduction of death/disabling stroke in 9/1000 patients treated with a 0.2% absolute excess in incidence of intracranial haemorrhage)

Studies have shown that there is 18% reduction in short term mortality. Overall mortality reduction was to the tune of 25%. LATE and EMERAS trials showed that the mortality benefit extended to those patients undergoing fibrinolysis with a window period of 6-12 hours. The greatest mortality benefit was observed in patients undergoing fibrinolysis in the first 1-2 hours after the onset of the symptom.

Fibrinolytic therapy should be started within 30 minutes after the diagnosis of STEMI is made, as its efficacy declines as the time from onset of coronary occlusion to therapy increases. This recommendation is based on the following information.

The earlier reperfusion therapy can be given, the greater the benefit that can be achieved. The survival benefit is greatest when fibrinolytic agents are administered within the first four hours after the onset of symptoms (38) and particularly within the first 70 minutes (39). A mortality benefit is less likely with fibrinolytic therapy at 13 to 18 hours. There may be benefit in patients presenting 12 hours after symptom onset and possibly up to 24 hours if the patient has ongoing or stuttering chest pain [40]. Although most myocardial necrosis occurs early (within the first 90 to 180 minutes), the advantages of late reperfusion are presumably related to the presence of a patent infarct-related vessel, leading to improved ventricular healing, reduced infarct expansion, and greater electrical stability.

Despite the above mentioned facts thrombolysis has the following limitations

- Even after timely administration, thrombolysis may not occur in around 15% of patients
- In those for whom successful thrombolysis occur, approximately one fourth of them may have reocclusion of infarct related artery resulting in re-MI
- Thrombolysis may not be a feasible treatment option in all patients due to several contraindications to thrombolysis.
- Increased risk of ICH with fibrinolysis especially in elderly.

Prehospital Fibrinolysis:

Meta analysis of 6 randomized trials comparing pre hospital fibrinolysis and in hospital fibrinolytic therapy for acute MI showed that pre hospital fibrinolysis significantly reduced all cause hospital mortality rates. One explanation to this phenomenon was that with passage of time the thrombus will get more organized and will be more resistant to fibrinolytic therapy.

A meta-analysis of six randomized trials (6434 patients) comparing pre hospital to in-hospital fibrinolysis showed the following benefits (42)

- A shorter time to fibrinolysis (104 versus 162 minutes).
- Reduced all-cause hospital mortality (odds ratio 0.83, 95% CI 0.70-0.98).
- An absolute risk reduction of almost 2 percent that translated into one life saved for every 62 patients treated with pre-hospital fibrinolysis

PRIMARY PCI:

Primary PCI in Acute MI patients is the gold standard of treatment as it establishes a higher percentage of complete and lasting reperfusion. Primary PCI restores angiographically normal flow in the previously occluded artery in more than 90% of patients,(43,44) whereas fibrinolytic therapy does so in only 50 to 60% of such patients.

In a meta analysis of 23 randomized controlled comparisons of primary PCI (involving 3872 patients) and fibrinolytic therapy (3867 patients), rate of death at 4-6 weeks after treatment was significantly lower among those who underwent primary PCI (7% vs 9%)(48). Several randomized trials (DANAMI-2, PRAGUE-2, AIR PAMI, STAT, STOPAMI-1, and STOPAMI-2) have demonstrated a better outcome with primary PCI and stenting compared to fibrinolysis (4-7).These trials used bare metals stents along with PCI. But the trials that followed which compared drug eluting stents with bare metal stents showed decreased target vessel revascularisation, cardiac death or MI and decreased rate of stent thronmbosis.(.(8-9)

Primary PCI is the treatment of choice in patients presenting with STEMI. (ACC/AHA Class I A recommendation). Current guidelines for the treatment of STEMI, recommend a door to balloon time of 90 minutes or less for patients undergoing primary PCI. But in real world situations especially in a developing country like ours timely PCI remains a challenge due to the following factors

- Low levels of literacy, has resulted in decreased awareness about the disease and its early signs and symptoms due to lack of awareness it takes a long time for the patient to ask for help.
- Numbers of centres performing PCI are few.
- Timely transfer to such centres also remains a huge challenge considering the dearth of emergency ambulance services and the state of our road infrastructure.
- Delay within a tertiary care contributed by multiple factors like arranging finances obtaining consent, availability of PCI lab in busy hours and the availability of round the clock manpower etc.
- Inadequate private and public health insurance programs have resulted in very low disposable incomes for most of the families toward their health expenses. Hence arranging finances on an emergency basis for a relatively costly intervention like primary PCI is a ‘luxury’ that very few people in a developing country like ours is endowed with.

Pharmacoinvasive strategy assumes significance in such a scenario.

Pharmacoinvasive Strategy:

Fibrinolytic therapy and Primary PCI are two commonly used reperfusion strategies which are conventionally viewed as mutually exclusive alternative therapeutic modalities. However great deal of recently acquired clinical evidence support the view that the two in combination are synergistic. The practical difficulties in implementing primary PCI in routine practice along with the evidence of benefit of pre hospital fibrinolysis (especially if administered early) and the overarching importance of time to reperfusion, regardless of the strategy used, serve as the foundation for development of a unified approach to management of patients with STEMI.

This Synergistic or unified approach is referred to as Pharmaco- invasive therapy. (50-52) Pharmacoinvasive strategy refers to routine angiography with a view to revascularization of infarct related artery 3-24 hours after fibrinolysis.

Time is a crucial factor in STEMI care. The risk of 1 year mortality was increased by 7.5% for each 30 minute delay in treatment (53,54) , confirming to the age old adage of time is muscle.

Prompt fibrinolytic treatment improves the likelihood of aborted myocardial infarction and the greatest incidence occurred in patients who under A delay in undergoing primary PCI results in reduced benefit of the invasive procedure Nallamothu et al. in his published studies highlighted the fact that mortality benefit associated with Primary PCI was lost if PCI-related delay exceeded 60 min.(56), pointing to the theoretical assumption of early fibrinolytic therapy compensating for PCI related delay.

CREATE and Kerala ACS Registry shows that there is significant delay in patient presentation, and initiation of timely reperfusion. Pharmaco-invasive approach helps to shorten the time to reperfusion of infarct related artery by initiation of lysis. PCI in 3-24 hours helps to consolidate the initial reperfusion process and prevent reclusion of the infarct related artery.

Several trials (CARESS-in-AMI, TRANSFER AMI, WEST study, STREAM, STEP AMI etc.) have addressed the potential benefit of pharmacoinvasive strategy.

The aim of this study was to to compare the efficacy of Pharmacoinvasive therapy and Primary Angioplasty in STEMI patients. The main objective was to study whether the incidence of composite end points (mortality, cardiogenic shock and re myocardial infarction) in Pharmacoinvasive strategy is non inferior to Primary Angioplasty in patients with acute MI.

Unique feature of this study was that in all the trials mentioned above comparing pharmacoinvasive strategy with other modes of revascularisation tenecteplase was used as the fibrinolytic agent. Due to cost constraints, streptokinase is the fibrinolytic agent that is commonly used in India and in our centre. Till date, there are no trials comparing pharmacoinvasive strategy using streptokinase with primary angioplasty. Hence, we would like to see whether pharmacoinvasive strategy using streptokinase was also non inferior to primary angioplasty in patients admitted with ST elevation MI getting admitted within a window period of 24 hours.

Detailed interpretation of the findings

Out of 147 patients who underwent a revascularization for procedure, 94 patients had primary PCI while 43 patients underwent pharmacoinvasive therapy. As mentioned above primary PCI is the gold standard for STEMI management. Hence primary PCI as a preferred treatment option is given to all the patients on presentation with STEMI, which explains a greater proportion of patients in the primary PCI arm. Those patients who are not willing for the same at presentation (due to various reasons such as delay in arranging finances, not able to take a quick decision, not sure regarding the insurance card status etc.) undergo thrombolysis with streptokinase. Routine coronary angiogram, is done, in 3-24 hours after thrombolysis, if they are willing for the procedure and can arrange finances / insurance card for the same.

90% of patients who underwent the revascularization procedure were males and females constituted only 10% of patients. The following reasons could be attributed for this skewed distribution.

- STEMI is more common among males than females
- The wide spread gender discrimination prevalent in our society has mirrored in the preference of the best option among males and not for the female sex
- Insurance cover more common amongst the breadwinner in the family.

The incidence of MI among males was maximally seen in the 50-60 age group. This was followed by the60 -70 year age group and then by 30-40 age group, probably reflecting the trend seen in the last decade or two where CVD is becoming more common in the younger age group among the male population in India. Among females, the maximal incidence was in the 60-70 age group followed by 50-60 years and 40-50 years age groups.

98.5 % of patients with STEMI had chest pain as the presenting symptom. It is estimated that almost 30% of people with ACS present with atypical symptoms. Diaphoresis was the next most common symptom at presentation constituting 11.7%. This was followed by dyspnea (6.6%) and giddiness (2.9%).

The mode of transport: Majority of patients (29%) who presented with STEMI hired a taxi to reach our hospital. 23% of patients were transported in an ambulance while 16% of patients utilized their own private vehicle to come to our center. It's interesting to note that 16% of patients came with STEMI in an auto rickshaw and the mode of transport was a two-wheeler for 7% of patients. 2% of patients utilized bus transport to reach the hospital.

Mode of transport is an important cog in the full wheel of effective and timely reperfusion therapy. In many cases the delay in reaching the hospital can be the difference between life and death. Hence it's important to understand the fact that only 23% of patients with STEMI was transferred to our hospital in an ambulance. Many patients reached our center in auto rickshaw's, two wheelers and by bus. This is an area that requires urgent and serious thought by the health authorities and the government. As

mentioned above speedy and efficient transfer to the point of care goes a long way in the effective management of STEMI patients. The outcomes would be much better if more of these patients could be transferred to our center in time. Raising awareness about the warning signs of ACS and having an emergency number for the ambulance service equipped with paramedical team with life saving measures is the need of the hour.

Baseline characteristics: As mentioned above males outnumbered the females in those who underwent primary PCI and pharamacoinvasive procedure. The gender distribution between both the strategies was matched evenly. The distribution of patients in each of the Killip's class was similar. There was no significant difference between the mean heart rate and systolic blood pressure values in the patients in both arms.

There was no significant difference between the underlying coronary risk factors between the patients in primary PCI and pharmacoinvasive arm.

There was a significant difference between the locations of infarction in patients between the two arms. 54% of patients in primary PCI arm and 74.5% of patients in pharmacoinvasive arm initially presented with Anterior wall MI. (p-0.012)

91% of patients in primary PCI arm and 95% of patients in Pharmacoinvasive arm underwent revascularization procedure through the radial route.

There was no significant difference between the TIMI flow rates among the treatment arms

There was no significant difference in the angiographic profile in patients between both arms. The p value for the difference in distribution of patients with triple vessel disease on coronary angiography was 0.09.

It’s interesting to note that 6.9% of patients in pharmacoinvasive group had a recanalised vessel while none of the patients in the primary PCI arms had a recanalised vessel, on coronary angiography. This value is considerably lower than other studies where tenecteplase was used as a thrombolytic agent. In STEPP – AMI study the infarct-related artery patency at angiogram was 82.2% in pharmacoinvasive group as against 22.6% primary PCI group.

There was a significant difference between the two arms in the use of thrombus aspiration catheter (73% vs. 21%, p<0.001) this suggests a decrease in the thrombus burden in patients who was subjected to thrombolysis with streptokinase. Again to quote the figures from STEPP AMI, thrombus was present in 26.7% in pharmacoinvasive group versus 63.2% in primary PCI group.

There was no significant difference between the blood investigation results between both the treatment arms. Troponin values between the two arms were the only value that showed considerable difference (1667 vs. 2672) but was deemed statistically insignificant (p-0.06).The increased troponin values seen in pharmacoinvasive arm may be due to the fact that pharmacoinvasive arm had more anterior wall MI patients as compared to primary PCI arm.

The median time delay from onset of symptoms to first medical contact was similar in the two study groups (5.5 vs. 5.51 hrs). The door to balloon time in our center(87 mins) was within the specified limit as recommended by the ACC/AHA(90 mins). The mean duration between lysis and CAG was 10.5 hours.

PRIMARY END POINT

	Primary end point				Total	Confidence Interval
Strategy	**Event**	**%**	**Non-event**	**%**		
Pharmoco invasive	3	7.0	40	93.0	43	(-18.88 to 5.17)
Primary	13	13.8	81	86.2	94	
Total	16	11.7	121	88.3	137	

The primary end point (mortality at 30 days, re-MI, cardiogenic shock) occurred in 3 out of the 43 patients (7%) in pharmacoinvasive group and 13 out of the 94 patients (14%) in primary PCI group.

In Primary endpoint (composite outcome), the Pharmacoinvasive procedure has nearly 50% less event as compared to Primary procedure. Therefore, the Non Inferiority hypothesis does not arise here. However, as the 95% CI covers null hypothesis value 0, the above data suggests equivalence. If the above difference has to be shown statistically significant, then the study would have dealt with 240 and 490 subjects in Pharmacoinvasive and primary arms respectively. That is the power of study at the moment is 21%. The minimum required power is 80

MORTALITY OUTCOME

	Follow up at 30 days				Total	Confidence Interval
Strategy	**Death**	**%**	**Alive**	**%**		
Pharmoco Invasive	2	4.7	41	95.3	43	(-9.09 to 7.75)
Primary	5	5.3	89	94.7	94	
Total	7	5.1	130	94.9	137	

Though the hypothesis has been non-inferior for pharmacoinvasive procedure as compared to Primary procedure, the lower mortality of 4.7% in the pharmacoinvasive arm implies that pharmacoinvasive is nearly equal. Therefore, the non-inferiority concept does not arise here. And therefore, the above data suggests equivalence. This also suggests equivalence if the equivalence margin is 10% on either side, as the 95% CI covers null hypothesis value 0.

The distribution of individual components of the primary and secondary end points is as follows

VARIABLE	PRIMARY ANGIOPLASTY (N=94)	PHARMACO-INVASIVE (N=43)
Primary end points	13 (14%)	3 (7%)
Mortality	5 (5%)	2 (4.5%)
Cardiogenic shock	12 (13%)	3 (7%)
Re MI	1 (1%)	0
SECONDARY END POINTS	PRIMARY ANGIOPLASTY	PHARMACOINVASIVE
Ejection fraction (Mean)	44.3	42.4
Duration of hospital stay	3.83 days	3.62 days
Bleeding manifestations	1 (1%)	0
Ischemic stroke	0	1 (2.5%)
Arrhythmias	2 (2%)	0
Mechanical complications	3 (3%)	1 (2.5%)

The distribution of secondary endpoints was similar between the two study arms. There was no significant difference between the ejection fraction, duration of hospital stay occurrence of bleeding manifestations, ischemic stroke, arrhythmias or mechanical complication amongst the two groups. In the STREAM study where a similar comparison was attempted in patients arriving within 3 hours, there was a significant increase in intracranial hemorrhage in lysis group which became non significant after 50% reduction in the dose of tenecteplase among patients 75 years or older. This may suggest a lower trend for occurrence of bleeding episodes if streptokinase is used instead of tenecteplase.

It should be borne in mind that pharmacoinvasive arm had larger number of patients with Anterior wall MI and the mean troponin values in pharmacoinvasive arm was also higher (though not statistically significant**).** Yet the results point towards equal benefit.

CONCLUSION

The following conclusions can be drawn from this study.

1) In Primary endpoint (composite outcome), the pharmacoinvasive procedure has nearly 50% less event as compared to Primary procedure. Mortality in pharmacoinvasive arm was 4.7% lower as compared to primary PCI arm. Though the hypothesis was non-inferiority, it is not applicable in this study as the above data suggests **equivalence.**
2) There was no significant difference between the secondary outcomes between the two groups.
3) Use of thrombus aspiration device and in turn the thrombus burden was significantly lower in the pharmacoinvasive group than in primary PCI arm.
4) Streptokinase though inferior to tenecteplase as a thrombolytic agent, maybe an ideal alternative, for pharmacoinvasive procedure in a tertiary centre like ours, where a majority of patients cannot afford tenecteplase.

LIMITATIONS

Though this study showed pharmacoinvasive therapy using streptokinase to be as effective as primary PCI, this study has the following limitations

1) This was an observational study with all its associated drawbacks
2) The sample size was small. A study on larger cohort of patients is necessary to draw meaningful conclusions. (If the above difference has to be shown statistically significant, then the study should include 240 and 490 subjects in pharmacoinvasive and Primary arms respectively. The power of the study at the moment is 21%. The minimum required is 80%)

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Annexure 1

Informed consent form to participate in a research study

Study Title:To study the Observational Study on Pharmacoinvasive strategy and Primary Angioplasty in atertiary care centre in acute Myocardial Infarction patients.

Study Number:
Subject's Initials: _____ Subject's Name:_____
Date of Birth/Age:_____

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]
(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ]
(iii) I understand that the sponsor of the clinical trial, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published. [ ]
(iv) I agree not to restrict the use of any data or results that arise from this study Provided such a use is only for scientific purpose(s) [ ]
(v) I agree to take part in the above study. [ ]

(or Thumb Impression)

Signature of the Subject/
Representative: _____
Date:_____/_____/_____
Signatory's Name:_____

Signature of the Investigator: _____
Date: _____/_____/_____
Signatory's Name: _____

(or Thumb Impression)

Signature of the Witness:
Date: _____/_____/_____
Name of the Witness:_____

Annexure 2

RAW DATA COLLECTION SHEET

Proforma - Case no.

Date of admission:

Therapeutic Strategy employed:

NAME:

AGE : SEX: Ht. : Wt. : BMI :

HOSPITAL NO.:

Address :

Contact no. :

Risk factor	HTN	DM	DL	Current or Ex Smoker	Prior CAD/ACS /MI			Prior PTCA / CABG		CKD	PAD	CVA	Fam CAD
Yes/no													
Dur													

Presenting complaints:

1. Chest pain : Mins 2. Dyspnea : Min 3. Others:

Pain to door time (Window Period) :

Mode of Transport :

Exam,n : Pulse : BP : CVS : RS:

Killip,s class

Door to Balloon time : Min

Time period b/w lysis and CAG (for pharmacoinvasive group)

Access site: Radial / Femoral

Angiographic profile : CAD – SVD/DVD/TVD/ LM+TVD , Occlusion in : Proximal / Mid /Dital

Infarct related artery : LM/ LAD /RCA/RI/ LCx/ D1/D2/D3/Septal branch of LAD/ OM1/OM2/OM3/AM/RPDA/LPDA/ RPLB/LPLB.

Pre PCI TIMI flow :

Use of thrombus aspiration by export catheter(YES/NO) or Clearway catheter (YES/NO)

Stent:

Final TIMI flow:

Post procedure complications:Vascualr site bleeding / other bleeding manifestations/Stent thrombosis/ Ischaemic stroke/ICH.

Investigations :

Parameter	Hb.	Platelets	Creat	eGFR	CKMB	TrT/I	Na	K	Lipids CH/TG/HDL/LDL			AC	PC	Hb A1c	TSH	
At PCI																

ECHO : (before discharge)

1. LVEF

2. Any mechanical Complications

- Duration of hospital stay –

If patient expired in hospital cause of death –

Follow up at 30 days : Alive /Dead

If dead, probable cause of death :

Cardiogenic shock after procedure .

3 Re –MI/ Rest angina or AOE / heart failure / Stent thrombosis with symptom class for Angina /Dysponea .

Arrythmia's after procedure : (specify the rhythm abnormality with the treatment offered)

Annexure 3

MASTER DATA SHEET

Sl No	Name	Strategy	Age	Sex	Hospital NO	HTN	Diabetes	Dyslipidemia	Smoking	Prioir CAD	Prior PTCA /CABG	CKD	PAD	RHD	CVA	Family h/o CAD	Chest pain	Dyspnea	Giddines	Sweating
1	Manohara Redd	1	65	1	920379f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
2	Janaki Raman	2	53	1	922295f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
3	Saraswathamma	2	66	2	922272f	1	2	2	2	2	2	2	2	2	2	2	1	1	2	2
4	Sivarajan	1	72	1	922271f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
5	Devan.R	1	60	1	922264f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
6	Nadana Sabapat	1	23	1	581891d	2	2	2	2	2	2	2	2	2	2	1	1	1	1	1
7	Kannan	1	70	1	638049	1	1	2	1	2	2	2	2	2	1	2	1	2	2	2
8	Haseena Begam	1	45	2	921458d	2	2	1	2	2	2	2	2	2	2	1	1	1	2	2
9	raji	2	75	1	831188d	2	1	2	2	1	2	2	2	2	2	2	1	2	2	2
10	Ashok Raj	2	50	1	585738a	2	2	2	2	2	2	2	2	1	2	2	1	2	2	2
11	Raghuraman.V.V	1	60	1	277803c	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
12	Krishnamurthy	2	40	1	922047f	2	2	1	1	2	2	2	2	2	2	2	1	2	2	2
13	Jabanullah.M	2	49	1	904575f	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2
14	Rajendran.S	2	57	1	920880f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
15	K.Seshiaha	2	57	1	920873f	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2
16	Wiiliam Joseph	2	48	1	888240f	1	1	1	1	2	2	2	2	2	2	1	1	2	2	2
17	Nirmala.N	1	55	2	920831f	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2
18	Rajendran.K	1	51	1	920414F	1	1	1	2	2	2	2	2	2	2	2	1	2	2	2
19	Surya	2	17	1	920377F	2	2	2	2	2	2	2	2	2	2	1	1	1	2	1
20	Durairaj.S	1	68	1	920411f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
21	Perumal	1	70	1	920435f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
22	Chandran	1	59	1	510313b	1	1	2	2	2	2	2	2	2	2	2	2	1	2	2
23	Newton Amos	1	59	1	920351f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
24	Udayakumar	2	39	1	920080f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
25	Rajamani.A	1	55	1	680185b	2	2	1	2	2	2	2	2	2	2	1	1	2	2	2
26	Ambu	1	70	1	920118f	2	1	2	1	2	2	2	2	2	2	2	1	2	2	2
27	Bharathi.V	1	63	1	917609f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
28	Duraichi.V	1	54	2	476970b	1	2	2	2	2	2	2	2	2	2	1	1	2	2	2
29	Sathyanarayana	1	57	1	790577c	1	1	1	2	2	2	2	2	2	2	1	1	2	2	2
30	Govindaraj	1	70	1	917355f	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2
31	Dhanapathi.M	1	36	1	698465c	2	1	2	2	2	2	2	2	2	2	1	1	2	2	2
32	Sigamani.A	2	60	1	915898f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
33	Paradesi	1	68	1	320377c	2	1	2	2	2	2	2	2	2	2	1	1	2	2	2
34	Thiyagesan.T	1	64	1	915367f	1	1	1	2	2	2	2	2	2	2	1	1	2	2	2
35	Iyanagar.S.N	1	53	1	743100d	2	1	1	2	2	2	2	2	2	2	1	1	2	2	2
36	Markose	1	73	1	917410f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
37	Anwaruddin	1	73	1	197934c	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
38	Mohan.K	1	45	1	914996f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
39	Baba.S.M	1	33	1	912784F	2	2	1	2	2	2	2	2	2	2	2	1	1	2	1
40	Ramakrishnan	1	68	1	701256A	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2
41	Sathyamoorthy	1	39	1	912910F	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2
42	Ravi	1	53	1	914679F	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2
43	Gopi.E	1	57	1	902659f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
44	Chnadrakumari.	1	59	1	904069F	1	1	2	2	2	2	2	2	2	2	1	1	2	2	2
45	Kuppa Achari	1	64	1	184994D	1	1	1	2	2	2	2	2	2	2	1	1	2	2	2
46	Shankar	1	58	1	909982F	2	1	1	1	2	2	2	2	2	2	2	1	2	2	1
47	Jaleel Khan	1	58	1	704692f	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2
48	Shankar	1	33	1	909131F	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
49	Somasekar.T	1	66	1	907809F	2	1	2	2	1	1	2	2	2	2	2	1	2	2	2
50	Mohan.K	1	36	1	907719f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
51	Shanmugam	1	39	1	907758f	1	1	1	1	2	2	2	2	2	2	2	1	2	2	2
52	Kotteswaran.S	1	66	1	972180d	1	2	2	2	2	2	2	2	2	2	1	1	2	2	1
53	Kamala Kannan	1	53	1	912127f	2	1	1	2	2	2	2	2	2	2	2	1	2	2	1
54	Appaswamy	1	73	1	080976B	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
55	Saravathulla.V	1	53	1	904067f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
56	Loganathan	1	58	1	902921f	2	1	2	2	2	2	2	2	2	2	1	1	2	2	2
57	Prakasam Chetty	1	67	1	902852f	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2
58	Rasheed	1	76	1	20016	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2
59	Govinda Achari	1	85	1	394763A	1	1	2	2	2	2	1	2	2	2	2	1	2	2	2
60	Rajamani.V	1	70	1	926182f	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2
61	Loganathan.R	1	60	1	947606c	1	1	2	1	1	2	2	2	2	2	2	1	2	2	2
62	Shanmugam.c	1	70	1	497277c	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2
63	Muniammal	2	65	2	923934f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
64	Shankar.K	1	44	1	926058f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
65	Gopal.N.M	1	72	1	926071f	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2
66	Radhabahia	1	70	1	923760f	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2

Pain to door time (hr)	Mode of transport	Pulse	BP	CVS	RS	Killips class	Diagnosis	Door to ballon time(mins)	Time period bw lysis and CAG	Access site	Angiographic profile	Infarct related artery	Use of thrombus aspiration	Stent	Final TIMI flow	HB	platelets	Creatinine	C
3.5	0	86	90/70	1	1	1	2	60	.	1	2	3	1	1	3	13	2.24	0.7	.
4	1	86	100/70	1	1	1	1	.	17	1	1	1	2	2	3	19	1.28	0.8	.
14	3	80	100/60	1	1	1	2	.	8	1	5	3	2	0	0	11	.	1.14	
4	2	74	130/80	1	1	1	2	75	.	1	3	3	1	3	2	15	1.79	1.1	
11	0	74	120/70	1	1	1	1	240		1	2	1	1	4	3	14	4.28	0.96	
1	2	74	110/70	1	1	1	1	80		1	1	1	1	5	3	17	1.69	0.87	
4	4	60	80/60	1	2	4	2	50		1	3	3	1	1	3	13	1.7	1.2	
4	2	74	120/70	1	1	1	2	85		1	1	3	1	1	3	12	3.19	0.6	
3	3	74	160/90	1	2	2	2		11	1	2	3	1	4	3	17	3.03	1.37	
3	0	84	130/80	1	2	3	1		3.5	1	1	1	1	9	3	13	2.94	0.89	
2	4	74	130/80	1	1	1	1	70		1	5	1	1	4	3	16	3.49	0.84	
5	3	78	100/80	1	1	1	1		10	1	1	1	2	2	3	15	3.37	1	
8	3	74	140/100	1	1	1	1		13	1	1	1	2	7	3	15	1.27	0.8	
4	3	74	130/80	1	1	1	2		5	1	1	3	2	11	3	15	3.24	0.8	
2.5	1	74	130/82	1	1	1	1		7.5	1	2	1	2	6	3	14	2.7	0.52	
9	1	100	170/100	1	1	1	1		12	1	1	1	2	12	2	17	1.62	0.8	
4	3	74	120/70	1	1	1	3	90		1	1	1	2	11	3	14	3.19	0.49	
4	1	74	110/70	1	1	1	1	80		1	1	1	1	5	3	13	1.38	0.74	
5.5	1	94	80/58	1	1	4	1		6	2	1	1	1	11	0	13	3.05	0.53	
5	3	64	120/80	1	1	1	2	150		1	2	3	2	5	2	12	2.08	0.9	
3	3	84	140/80	1	1	1	1	70		1	1	1	1	6	3	7.2	3.14	1.2	
9	2	126	160/100	1	2	3	2	80		1	3	3	2	11		12	4.3	1.07	
3	2	74	110/70	1	1	1	1	55		1	3	1	2	4	3	17	2.85	0.92	
3	2	86	120/70	1	1	1	1		11	1	1	1	2	5	3	10	2.93	0.85	
1	5	64	140/80	1	1	1	2	98		1	2	2	2	5	3	14		0.8	
5	2	74	130/80	1	1	1	2	80		1	1	3	2	8	3	15	3.65	0.68	
2.5	2	45	100/60	1	1	1	2	88		1	4	3	1	8	3	12	2.9	0.9	
2	1	74	130/80	1	1	1	2	80		1	2	3	2	5	3	14	3.06	0.6	
2.5	1	100	80/60	1	1	4	1	80		1	1	1	1	8	2	16	3.26	1.2	
6	3	74	80/50	1	1	4	2	140		1	2	2	1	13,2	3	14	4.08	1.02	
4	4	108	124/70	1	1	1	1	90		1	1	1	1	8	3			0.72	
12	1	88	110/90	1	1	1	1		18	1	1	1	2	5	3	15	2.13	0.82	
1	1	84	140/88	1	1	1	1	79		1	2	1	2	5	3	16	2.79	1.06	
3	4	120	90/60	1	1	1	1	98		1	1	1	1	8	3	15	2.1	0.96	
12	3	108	110/70	1	1	1	1	82		1	2	1	2	3	3	13	2.8	1.14	
6	0	80	140/90	1	1	1	2	82		1	1	3	1	13	3	12	2.1	0.7	
4	0	98	80/60	1	1	4	4	98		1	1	1	2	5	3	9.5	2.4	1.06	
2	3	86	80/60	1	1	4	1	88		1	1	1	1	4	2	20	1.17	1.62	
2.5	1	0	0	1	1	4	1	90		1	1	1	3	5	3	17	2.43	0.7	
9	5	74	130/70	1	1	1	2	100		1	1	3	2	3	3	12	2.6	1.17	
4.5	2	74	100/60	1	1	1	1	80		1	1	1	1	5	3	15	2.6	0.89	
7	4	104	70/50	1	1	4	1	75		1	1	1	1	2	2	14	2.01	1.04	
2	4	90	100/60	1	1	1	1	76		1	1	1	1	4	3	16	2.23	1.05	
7	3	88	150/90	1	1	1	1	80		1	1	1	2	5	2	15	1.9	1.18	
5.5	2	74	110/80	1	1	1	1	70		1	1	1	1	8	3	14	2.53	`.06	
2	3	74	130/80	1	2	2	1	80		1	1	2	1	3	3	19	2.3	1.23	
4	2	70	130/90	1	1	1	1	110		1	1	1	1	5	3	16	2.92	0.85	
4	3	92	130/90	1	1	1	1	90		1	1	1	1	5	3	15	1.4	0.82	
3	1	94	130/80	1	1	1	1	80		1	1	1	1	11	3	14	2.04	1.34	
3	3	72	100/80	1	1	1	1	75		1	1	1	1	4	3	18	1.9	0.7	
2	3	74	150/90	1	1	1	2	55		1	2	3	1	6	3	16	2.15	1.4	
2	4	104	140/80	1	1	1	1	70		1	1	1	1	8	3	15	2.8	0.9	
11.5	1	80	160/100	1	1	1	1	85		1	1	1	1	5	3	16	3.3	0.82	
6	3	74	130/80	1	2	2	1	92		1	1	1	1	14	3	11	1.8	1.45	
2	2	74	100/70	1	1	1	1	80		1	2	1	2	5	3	14	1.8	1.02	
8.5	3	86	100/70	1	2	2	1	75		1	3	1	1	7	1	14	3.86	1.24	
5	2	84	130/70	1	1	1	1	72		1	1	1	2	3	3	16	1.86	0.7	
2	4	86	150/80	1	1	1	2	55		1	1	3	2	1	3	11	2.03	1.16	
8	1	86	130/90	1	1	1	2	75		1	2	3	1	14	3	16	2.54	1.28	
5	3	86	120/70	1	1	1	2	90		2	2	3	1	6	3	11	3.86	0.7	
7	0	86	140/90	1	1	1	2	75		1	4	2	1	4	3	13	1.73	0.7	
5.5	0	74	130/80	1	1	1	2	86		1	3	2	1	5	3	15	2.7	1.2	
5	5	86	120/70	1	1	1	1		18	1	1	1	2	5	3	12	1.08	0.53	
10	3	80	120/70	1	1	1	2	110		1	1	3	1	4	3	13	2.25	0.6	
4.5	1	86	130/80	1	1	1	1	90		1	6	1	1	2	3	13	2.59	0.8	
4	0	86	140/90	1	1	1	2	78		1	1	2	1	5	2	14	2	0.5	

Creatinine	CKMB	TrT	sodium	Potassium	Cholesterol	TG	HDL	LDL	HB1ac	TSH	ECHO (EF)	Mechanical complication	Duration of hospital stay	Cardiogenic shock	Other complications	Died in hospital	Follow up at 30 days	Cause of death						
																					Time b/w admission & death			
0.7	.	30	135	3.5	155	50	40	111	6.2		47.6	2	3	2	4	2	1	0						
0.8	.	.	132	3.7	184	110	30	139	6	1.28	43.3	2	3	2	4	2	1	0						
1.14	41	1328	136	2.6	162	144	31	99	6.1	0.8	46.9	2	3	2	4	2	1	0						
1.1	8.2	32	135	3.8	124	68	32	86	6.1		54	2	3	2	4	2	1	0						
0.96	62	441	130	3.7	165	108	27	127	6		47.4	2	3	2	4	2	1	0						
0.87			138	3.6	106	77	18	76		0.95	49.1	2	4	2	4	2	1	0						
1.2			135	3.7							25	MR	3	1	1,2	1	2	Multiorga	CHECK			28.5hrs		
0.6		39	130	4.1	201	175	33	141	6.1	4.3	53.1	2	3	2	4	2	1	0						
1.37				4.7	227	81	58	166	7.5		54.8	2	3	2	5	2	1	0						
0.89	500	10000	139	3.2	280	142	41	173	5.2	0.9	22	2	4	1	1,5	1	2	Multiorgan failure				75 hrs 45 mins		
0.84		176	135	3.3	154	137	35	114	0.78		42.3	2	4	2	4	2	1	0						
1	104	309.4	130	3.9	275	148	42	205	5.8		40.7	2	3	2	4	2	1	0						
0.8	16	422	138	4.2	134	164	38	72	6.8		46	2	2	2	4	2	1	0						
0.8	5.6	39.8	134	3.6	117	82	39	74	4.6	1.34	57	2	3	2	4	2	1	0						
0.52		1879	135	4.1	212	1009	33	169	5.8	3.9	44.2	2	2	2	4	2	1	0						
0.8	105	5596	139	2.6	127	144	61	45	7	0.42	40	2	3	2	4	2	1	0						
0.49	2.4	16	139	3.3	218	110	47	148	7.9	4.4	51.9	2	3	2	4	2	1	0						
0.74	3.4	65.7	139	3.3	139	242	19	98	6.9		47.8	2	3	2	4	2	1	0	No rResidual lesions. ectatic vessels					
0.53		418	137	3.8	89	79	28	51			25	1	1	1	1,2	1	2	2				23hrs 40 mins		
0.9				3.9	118	197	30	72	5.5		46.1	2	4	2	4	2	1	0						
1.2			138	3.4	166	398	32	80	5.1	3.5	48.7	2	4	2	4	2	1	0						
1.07	92	4107	114	4.3	145	51	51	87	10.3	3.04	45.2	2	20	2	4	2	1	0	Couldn't wire					
0.92		43.64	140	3.1	243	123	46	184	5.1	2.8	47.3	2	4	1	4	2	1	0						
0.85	2.7	39			164	131	35	117	7.1	1.6	40.6	2	4	2	4	2	1	0						
0.8		30.6	138	4.3	271	376	37	180		1.35	54.2	2	2	2	4	2	1	0						
0.68	4	27.74	134	3.8	174	259	18	125	8.3		55	2	4	2	4	2	1	0						
0.9		213	142	2.8	140	99	35	110	5.5	2.75	32.4	1	4	2	4	2	1	0						
0.6	3.7	40.6	139	3.6					5.3		44.6	2	4	2	4	2	1	0						
1.2	3.8	10.4	136	3.2	195	141	36	128	7.9	3.6	30.3	2	9	2	4	2	1	0						
1.02	12	51.4	137	4.2	180	108	31	131	5.7	0.5	47	2	3	2	4	2	1	0						
0.72	23	397.5	126	3.8	136	126	30	87	11.2	0.65	41	2	3	2	4	2	1	0						
0.82	92.69	563	125	3.9						0.78	42	2	4	2	4	2	1	0						
1.06	10	93	137	3.9	120	93	43	63	6.6		39	2	3	2	4	2	1	0						
0.96	5.96	97.32	134	2.9	184	134	43	129	9.3	1.68	33	2	4	2	4	2	1	0						
1.14	300	3934		4.6	195	32	165	164	7.3	12.6	40.2	2	3	2	4	2	1	0						
0.7	300	2122	130	4.1	152	81	35	119	7.7	0.95	59.6	2	4	2	4	2	1	0						
1.06	300	10000	134	3.3	97	107	33	56	5.4	2.09	54	2	4	2	4	2	1	0						
1.62	2.02	76.4			242	176	38	170	7.9		25	2	3	1	1,6	1	2	3				73hrs 40mins		
0.7		71.38	130	3.1	245	153	49	178		1.69	47	2	4	2	4	2	1	0						
1.17		2425	137	4.1	187	167	39	128	6	0.95	52	2	3	2	4	2	1	0						
0.89		1395	140	3.3	182	63	45	109	6.3	3.2	37	2	4	2	4	2	1	0						
1.04		83.7	133	3.9	128	123	24	82	7	1.5	35.1	2	3	2	4	2	1	0						
1.05	46.2	8156	132	4.3	193	180	37	129	6.6		46	2	3	1	1,3	1	2	4	Stent Throml	Coronary Ect	Type I	164hrs		
1.18	12.46	112.4	131	4.4	158	127	41	99	10.6		37	2	4	2	4	2	1	0						
`.06	13	3070	135	3.8	228	146	38	165	8	1.8	31	2	3	2	4	2	1	0						
1.23	6.61	25.12	135	4.6	175	252	29	106	8.3	1.7	37.4	2	4	2	4	2	1	0						
0.85	15.3	1771	134	3.5	168	130	33	122	9.4	0.4	47.1	2	3	2	4	2	1	0	Coronary Ect	Type I				
0.82	32	297	132	3.9	149	150	30	100	5.6	1.7	44.7	2	3	2	4	2	1	0						
1.34		10000	135	3.7	111	42	36	74	8.8		38	2	5	2	4	2	1	0	Very Late stent thrombosis. Not stented as III flow established with POBA					
0.7		19.4	141	3.5	174	123	46	123	5.6		38	2	3	2	4	2	1	0						
1.4			140	3.6	191	374	46	117	7		55	2	3	2	4	2	1	0						
0.9	179.8	4375	138	3.7	190	135	31	135	5.5	3.5	34.4	2	3	2	4	2	1	0						
0.82		70	135	3.8	240	120	46	152	9.1	2.4	42	2	4	2	4	2	1	0						
1.45	29.15	707	135	3.3	131	73	32	79	5.8		45.5	2	3	2	7	2	1	0	Had ReMI d/t Acute stent thrombosis					
1.02	2.76	35.18	138	3.6	130	217	31	70			42.4	2	5	2	4	2	1	0						
1.24	51.6	2215	125	3.5	202	132	43	142	13.5		36	3	4	1	1	1	2	3				69.5hrs		
0.7	21.9	154.5	131	5.9	174	78	30	132	5.4		39	2	3	2	4	2	1	0						
1.16		123	136	3.6	108	70	33	72	7.3		56.5	2	3	2	4	2	1	0						
1.28	15.9	122.8	133	4.5	206	298	35	145	7.1	6.023	51.5	2	5	2	5	2	1	0						
0.7			131	4	162	145	29	125	10.3	2.7	55	2	3	2	4	2	1	0						
0.7			131	3.7	91	158	30	42	7.3	0.39	45.9	2	8	2	4	2	1	0						
1.2			139	4.6	188	100	43	140	6.2	0.78	44	2	4	1	4	2	1	0	Had VT after procedure					
0.53		188.8	137	3.5	115	46	39	62	5.5		49	2	3	2	4	2	1	0						
0.6	96.7	435.5	126	3.8	144	101	51	90	5.2	2.6	49	2	3	2	4	2	1	0						
0.8	300	3268	136	3.7	142	46	35	109		0.54	41	2	3	2	4	2	1	0						
0.5	13.1	79	140	3.4	149	96	35	95	5.1	4.15	42	2	4	2	4	2	1	0						

67	Kallappa	1	62	1	923672f	2	2	2	1	2	2	2	2	2	2	2	1	5	2	2
68	Murugan	1	63	1	923450f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
69	Nagalingam.M	2	60	1	923471f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
70	Vediyappan.P	2	75	1	923331f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
71	Rajendran.R	1	57	1	838324d	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2
72	Kasi	1	57	1	570730d	2	2	1	1	1	1	2	2	2	2	2	1	2	2	2
73	Raghunathan	1	55	1	923201f	1	1	2	1	1	2	2	2	2	2	2	1	2	2	2
74	Indira	1	37	1	923001f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
75	Noor Mohamme	2	31	1	922944f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
76	Indira	1	37	2	923001f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
77	Kannaiah.C.G	1	70	1	922973f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	1
78	Ranmesh.R	2	40	1	922524f	2	2	1	1	2	2	2	2	2	2	2	1	2	2	2
79	Mogiliah.K	1	61	1	487888b	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
80	Yuvaraj	2	49	1	704828f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	1
81	Papathiammal	1	77	1	922286f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
82	Kumar.B	1	47	1	751963f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
83	Narasimhan.P.G	1	58	1	753047f	1	1	1	1	2	2	2	2	2	2	2	1	2	2	2
84	Kumar.D	1	44	1	753193f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
85	Rajendran.R	1	57	1	751958f	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2
86	Subramaniayam	1	34	1	751786f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
87	Rajagopal	1	68	1	672975b	2	2	2	1	2	2	2	2	2	2	2	1	2	2	1
88	Sundaram.S	1	64	1	751510f	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2
89	Subramabi.J.K	1	65	1	751298f	1	1	2	2	2	2	2	2	2	2	2	1	2	2	1
90	Pandurangan.V	1	65	1	751206f	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2
91	Govindamma	1	65	2	751213f	2	2	1	2	2	2	2	2	2	2	1	1	2	2	2
92	Mohankumar.V	1	38	1	751176f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
93	Perumal swamy	1	61	1	750852f	1	1	2	2	1	2	2	2	2	1	2	1	2	2	2
94	Salima Bee	1	55	2	750847f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1
95	Muniswamy	1	65	1	750762f	2	2	2	1	2	2	1	2	2	2	2	1	2	2	1
96	Kumaresan	1	50	1	750073f	1	1	1	2	2	2	2	2	2	2	1	1	2	2	2
97	Kuppusamy.S	1	56	1	726959f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
98	Aravindh	1	37	1	726872f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
99	Vijaya kumar.D	1	42	1	753365f	2	1	2	1	2	2	2	2	2	2	2	1	2	2	2
100	Mohammad Bas	1	52	1	173953b	1	1	2	2	2	2	2	2	2	2	1	1	2	2	2
101	Pichandi.C	1	61	1	901584f	1	2	2	2	2	2	2	2	2	2	1	1	2	2	2
102	Ramachandran	1	56	1	063020d	2	2	2	1	2	2	2	2	2	2	1	1	2	1	2
103	Sundari	1	76	2	901430f	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2
104	Shanmugam V	1	70	1	834570f	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2
105	Naryan.N	1	65	1	754820f	1	1	2	1	2	2	2	2	2	2	1	1	2	2	2
106	901425f	1	37	1	901425f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
107	Elumalai	1	58	1	901770f	2	1	2	2	2	2	2	2	2	2	1	2	2	1	1
108	Anjanayelu.B1	1	70	1	901873f	1	1	1	1	2	2	2	2	2	2	2	1	2	2	2
109	Subramani.E	1	59	1	780396f	1	1	1	1	2	2	2	2	2	2	2	1	2	2	2
110	Narayanamurthy	1	41	1	902117f	2	2	2	2	2	2	2	2	2	2	1	1	2	2	2
111	Javaha Basha	1	40	1	902146f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
112	Natarajan.C	1	51	1	902438f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
113	Sethu	1	55	1	902489f	2	2	2	1	2	2	2	2	2	2	1	1	2	2	2
114	Mala	2	50	2	912738f	2	1	2	2	2	2	2	2	2	2	1	1	2	2	2
115	Abdul Rabani	2	44	1	043536d	1	2	2	1	2	2	2	2	2	2	2	1	2	2	2
116	Violet Jamuna	2	43	2	552259c	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2
117	Govindaswamy	2	85	1	914518f	1	1	1	2	2	2	2	2	2	2	1	1	2	2	2
118	Duraiswamy	2	52	1	904987f	1	1	2	1	2	2	2	2	2	2	2	1	2	2	2
119	Prithvirajan	2	40	1	904708f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
120	Devaraj	2	37	1	901840f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
121	Suresh Babu.R	2	47	1	904868f	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2
122	Ambiga	2	66	2	904770f	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2
123	Subramani.S	2	65	1	753069f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	1
124	Krishnamurthy.C	2	62	1	984562D	1	1	2	2	2	2	2	2	2	2	2	1	2	2	2
125	Sundaravelu.A	1	53	1	700288a	1	1	2	1	2	2	2	2	2	2	2	1	2	2	2
126	Kirubhanandan.	2	42	1	750702f	1	1	1	2	2	2	2	2	2	2	2	1	2	2	2
127	Amsaveni	2	53	2	716719f	1	2	2	2	2	2	2	2	2	2	2	1	1	2	1
128	Rajesh.N	2	36	1	740956f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
129	Partheeban.K	2	42	1	348111f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
130	Kanchan Nair	2	77	1	740919f	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
131	Pichandi	2	53	1	750449f	2	2	2		2	2	2	2	2	2	2	1	2	2	2
132	Farroque basha	2	47	1	726946f	2	2	2	1	2	2	2	2	2	2	1	1	1	2	2
133	Prakash.R	2	28	1	901471f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
134	Mohan.S	2	50	1	754207f	2	2	2	1	2	2	2	2	2	2	2	1	2	2	2
135	Sekar.N	2	52	1	840867f	2	2	2	2	2	2	2	2	2	2	1	1	1	2	2
136	Bhaskaran.K	2	54	1	902195f	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2
137	Thiyagaraj.N	2	65	1	048159f	1	1	2	2	2	2	2	2	2	2	1	1	2	2	2
138	Natarajan.K.S	2	76	1	902187f	2	2	2	1	2	2	2	2	2	2	2	1	2	1	2
139	Gouse Bee	2	70	2	103356A	1	1	1	2	2	2	2	2	2	2	2	1	2	2	1

5	0	96	80/60	1	1	4	2	76		1	3	3	1	15	2	15	1.62	1.12
2	1	74	150/100	1	1	1	2	70		1	2	3	1	4	3	11	2.17	0.8
2	2	68	130/80	1	1	1	2		5.5	1	3	3	2	5	3	15	1.6	0.95
1	0	74	180/80	1	1	1	1		20	1	7		2			12	2.83	0.9
4.5	1	86	120/70	1	1	1	2	85		1	3	3	1	11	3	11	2.26	0.26
2.5	1	84	110/70	1	2	2	1	72		1	2	1	1	5	3	21	1.95	1.41
12	2	74	150/80	1	1	1	5	75		1	3	3	1	14	3	16	1.05	0.9
6.5	4	84	90/70	1	1	1	1	180		1	1	1	2	16	3	13	2.25	0.5
12	1	94	100/82	1	1	1	1		12.5	1	1	1	2	2	3	15	2.7	0.7
6.5	4	84	90/70	1	1	1	1	100		1	1	1	2	16	3	13	2.25	0.5
3	3	74	110/82	1	1	1	1	60		1	1	1	1	6	2	14	1.65	1.36
3	2	130	80/70	1	1	4	1		23	1	1	1	1	8	2	16	2.37	0.86
14	4	74	130/70	1	1	1	1	75		1	1	1	1	4	3	14	2.14	0.82
7.5	3	84	110/82	1	1	1	1		11.5	1	1	1	2	5,2	3	15	2.67	1.18
4	1	46	110/70	1	1	1	2	130		2	1	3	1	4,2	3	9.7	2.53	1.7
1.5	4	80	120/70	1	1	1	1	65		1	1	1	1	8	3	12	3.78	0.8
15	7	80	140/90	1	1	1	1	80		1	2	1	2	7		15	2.28	1.06
8	1	80	140/90	1	2	2	1	125		1	1	1	1	5	3	16	2.93	1.08
8.5	3	80	150/100	1	1	1	1	70		1	1	1	1	5	3	18	2.69	1.15
8.5	3	82	120/80	1	1	1	1	60		1	1	1	1	13	3	15	2.7	0.87
5.5	3	86	120/80	1	1	1	2	120		1	1	3	1	8	1	12	3.23	0.9
7	2	96	120/60	1	2	2	2	120		1	1	3	2	15	3	13	2.9	0.89
8	3	68	90/60	1	1	1	2	135		1	3	3	1	15	3	16	2.01	1.09
2.5		92	110/80	1	1	1	1	80		1	1	1	1	8	3	15	2.56	0.9
3	4	68	100/80	1	1	1	2	75		1	3	3	1	5	3	12	2.5	0.81
2		80	130/880	1	1	1	1	125		1	1	1	1	5	3	15	2.7	1.04
3.5	3	68	100/60	1	1	1	2	96		1	2	3	1	8	3	16		0.78
8	3	100	120/80	1	1	1	1	100		1	1	1	1	17	3	12	4.9	0.9
18.5		70	140/80	1	2	2	1	45		1	1	1	1	15	3	11	2.4	2.22
3	2	110	120/70	1	1	1	2	50		1	1	3	2	5	3	16	2.5	1
17.5	3	84	110/60	1	1	1	1	85		1	1	1	1	13	3	13	3.3	1.16
5.5	2	108	90/60	1	1	1	1	80		1	1	1	2	15	3			0.96
23	4	66	120/70	1	1	1	2	95		1	2	3	1	17	3	13	2.66	0.8
7	4	92	120/70	1	1	1	2	92		1	3	3	1	5	3	11	2.5	1.65
15	1	80	110/70	1	1	1	2	80		1	3	3	1	8	3	16	2.46	0.98
9	3	38	70/50	1	1	4	2	80		1	3	3	1	15	3	13	2.5	1.2
10	3	86	120/80	1	1	1	1	86		1	2	1	1	3	3	12	2.4	0.86
3	3	80	140/90	1	1	1	2	90		1	1	3	1	8	3	13	2.8	1.1
1	4	42	130/80	1	1	1	2	65		2	1	3	1	8	3	13	3	1.24
5	1	86	140/90	1	1	1	2	80		1	1	3	1	5	3	18	2.6	1
14	4	60	90/80	1	1	1	2	70		1	2	3	1	4	3	14	3.2	1.2
2	2	74	110/80	1	1	1	1	98		1	1	1	2	4	3	12	2.19	1.23
2	3	92	130/90	1	1	1	1	80		1	1	1	2	5	3	13	3.7	0.4
4	1	74	130/80	1	1	1	2	85		1	2	3	1	18	3	15	2.6	0.9
4	4	74	100/80	1	1	1	1	150		1	1	1	1	15	3	13	2.4	1.13
6	3	74	130/80	1	2	2	1	72		1	3	1	1	5	2	18	2.6	0.95
4	3	86	140/90	1	1	1	1	76		1	1	1	2	8	3	14		0.9
6	1	74	80/60	1	1	4	1		4	1	1	1	2	2	2	14	3.03	0.58
4	4	52	80/50	1	1	4	2		3.5	1	1	3	1	7	3	17	3.3	1.07
3	4	64	110/80	1	1	1	2		3.45	1	2	3	2	7	3	11		0.63
2	3	86	120/70	1	1	1	1		22	1	1	1	2	5	3	14	2.8	0.98
4	3	224	100/60	1	1	4	2		23	2	2	3	2	5	3	14	2.83	1.43
1.5	2	110	160/100	1	1	1	1		12	1	1	1	2	13	3	16		1.1
7	1	74	100/70	1	1	1	1		5	1	1	1	2	5	3	14	2.9	1.24
9	3	76	130/80	1	1	1	1		12	1	1	1	2	6	3	14	2.14	0.95
4	5	140	120/70	2	2	3	1		10	1	3	1	2	5	2	11	3.5	0.84
10	5	56	140/100	1	1	1	2		8	1	1	2	2	17	3	12	1.01	1.04
6	3	35	100/70	1	1	1	2		4.45	1	3	3	2	17	3	12	1.8	1.37
6	1	74	150/100	1	1	1	2	60		1	2	3	2	5	3	12		0.6
11		82	110/80	1	1	1	1		7.5	1	1	1	2	5	3	16	3.5	0.9
10	2	96	130/90	1	2	2	1		5	1	1	1	1	17	3	9.4	1.5	1.16
3.5		72	110/80	1	1	1	1		7	1	1	1	2	7		14	1.6	0.93
4	7	92	140/80	1	1	1	1		8	1	1	1	2	7	3	18		1.2
4.5	2	80	130/80	1	1	1	1		12	1	2	1	2	8	3	12	2.3	0.7
3.5	1	72	100/80	1	1	1	1		4	1	2	1	2	7	3	14	2.27	0.8
2	1	108	140/90	1	2	2	1		10	1	2	1	1	13	3	15	2.08	1.14
8	3	86	120/70	1	1	1	1		20	1	1	1	2	7		19	3.4	0.8
3	1	74	130/80	1	1	1	1		4	1	1	1	1	7	3	12		1.1
3	1	80	110/80	1	1	1	1		4	1	1	1	2	7	3	14	3.5	0.9
9	1	120	130/80	1	1	1	1		15	1	1	1	2	4	3	14	1.63	1.01
8	4	74	140/80	1	1	1	2		14	1	2	3	1	5	3	13	2.07	0.67
3.5	4	74	110/70	1	1	1	2		10	1	2	3	2	4	3	12	1.85	1.69
4	4	96	160/90	1	1	1	1		11	1	3	1		8	2	11	3.14	0.6

240		136	3.9	155	106	27	117	5.4	3.71	53.2	2	8	2	4	2	1	0				
	21.38	136	3.7	156	140	32	103	6.6	0.7	55.9	2	3	2	4	2	1	0				
	1876	136	3.8	105	80	33	66	5.3	0.52	56	2	3	2	4	2	1	0				
	180	123	4.3	139	78	53	80	6.3		35.3	2	3	2	4	2	1	0	not stented .	Discharged at request		
		150	4.4	173	191	34	123	6.2	0.49	55	2	6	2	4	2	1	0				
4.2	147	135	3.8	188	320	40	109	6	3.13	52.5	2	5	2	4	2	1	0				
56	912	131	3.8	104	70	23	71	12.2	2.4	51.6	2	6	2	4	2	1	0				
249	976	131	3.9	129	144	44	67	5.2	0.12	41.3	2	3	2	4	2	1	0				
161	8744	134	3.8	163	54	42	102	6		45.9	2	5	2	4	2	1	0				
229	976	131	3.9	129	114	44	67	5.2	0.12	41.3	2	3	2	4	2	1	0				
4.6	93.3							5.8	0.57	33	2	3	1	2	1	2	2	Recurrent VT, Cardiogenic shock			117.5hrs
15.14		133	3.9	268	341	63	162	5.7	0.97	35.4	2	3	2	4	2	1	0				
112	972	131	3	160	133	27	124	7.1	3.04	46.6	2	3	2	4	2	1	0				
19.7	154.2	134	3.3	166	197	30	110			45.6	2	2	2	4	2	1	0				
		136	5.2	131	114	29	94	8.6	1.8	51.8	1	5	2	4	2	1	0				
237	8896	137	3.5	159	141	39	96	7.5		40.4	2	3	2	4	2	1	0				
294	10000			204	165	45	131	11.3		37.3	2	2	2	4	2	1	0				
500	9500	109	4.3	144	83	46	93	7.1		38.2	2	4	1	4	2	1	0				
21.59	7148	133	4.2	240	292	31	163			37.5	2	3	2	4	2	1	0				
48.7	443	135	3.5	177	60	39	125	5		47	2	3	2	4	2	1	0				
23	142	131	4.3	118	86	23	76	6.5		42	2	5	2	2	2	1	0	VT,VF during procedure dc shocked. Not stented due			
2	834	135	4.1	116	60	27	79			46	2	3	2	4	2	1	0				
190	863	135	3.7	186	137	37	122	9		51.7	2	3	1	4	2	1	0				
16	245	137	4.8	171	119	36	125	6		36.5	2	4	2	4	2	1	0				
2.35	32	140	3.9	248	190	36	182	7.8		42.4	1	4	1	4	2	1	0				
15	45	137	3.7	241	83	49	179	6		36.5	2	4	1	4	2	1	0				
176	4807	134	3.4	188	188	37	121	6.5		41.6	2	3	2	4	2	1	0				
3	87	128	4.7	137	104	26	91	5.8		44.1	2	3	2	4	2	1	0				
10	10000			178	116	40	113	5.7		40.4	2	4	2	10	2	1	0				
21.7	274.6	135	3.2							33.2	2	3	2	4	2	1	0				
76.7	423.5	134	3.7	115	60	35	74	5.8		42.7	2	3	2	4	2	1	0				
		140	3.5	140	93	42	87	8.4		42.6	2	2	2	4	2	3	0				
111	3188	137	3.6	179	221	29	127	7.8		44.2	2	3	2	4	2	3	0				
17	110	135	4	112	152	37	56	7.3		53	2	2	2	4	2	1	0				
420	136	4.2	170	113	35	128	118	6		53.4	2	2	2	4	2	1	0				
23	251	136	4	140	169	28	92	5.9	2	50	2	3	2	4	2	1	0				
78	493	136	4.1	179	88	43	122	6		30	2	9	1	4	2	1	0				
4	41.4	132	3.8	143	128	40	92	8.7		47.9	2	3	2	4	2	1	0				
150	7095	137	4.2	137	58	52	79	6.4	0.33	50.7	2	3	2	4	2	1	0				
20.9	117.7	132	3.8	178	131	32	138	7.9		46.1	2	3	2	4	2	1	0				
41	236	138	3.6	165	91	36	111	10.7		52.2	2	3	2	4	2	1	0				
6.2	150.4	138	3.8	158	275	30	89	7.2		52	2	4	2	4	2	1	0				
7.6	59.6	129	3.8	187	235	31	116	8.5		42.6	2	3	2	4	2	1	0				
145	636	131	3.6	127	118	32	80	5.5	3.8	50.4	2	4	2	4	2	1	0				
163	6690	130	4.2	198	101	53	130	5.4		38.4	2	3	2	4	2	1	0				
5.4	57.2	132	3.5	187	74	40	134			38.9	2	2	2	4	2	1	0				
120	1162	136	3.9	164	64	63	96	5.1		38	2	3	2	4	2	1	0				
		1377	3.4	145	40	37	100	11.9	0.17	31.3	2	4	2	4	2	1	0				
	4781	141	2.7	170	116	29	119	5.1		48	2	3	2	4	2	1	0				
111.2	4116	141	4.4	149	49	50	84	10.5		56	2	3	2	4	2	1	0				
1.75	23.7	132	4.1	201	167	43	130	9.3	3.1	47	2	5	2	4	2	1	0				
28.5	200.5	134	4.5	156	125	42	99	6.7		46.8	2	6	2	4	2	1	0		Primary VT DC verted		
26.5	3667	136	3.8	121	136	31	73	6.5		40	2	3	2	4	2	1	0				
100	9128	135	2.9	124	102	28	77	5.5		38	2	3	2	4	2	1	0				
2.2	72.4	136	3.7	112	101	37	68	14.2		43	2	3	2	4	2	1	0				
7.65	87.2	136	2.8	194	463	38	104	10.1		40	2	3	2	1,5	2	1	0				
128	10000	134	4	171	112	45	108	6.2		46.1	2	3	2	4	2	1	0				
268	10000	136	4.5	184	169	36	122	7.5		29.8	2	4	2	4	2	1	0				
58	2271	138	3.5	163	134	26	106	7.5		52.2	2	3	2	4	2	1	0				
17.8	455	136	4.6	211	87	39	155			34.1	2	2	2	4	2	1	0				
31.6	662	140	4.5	167	143	97	56		0.8	34.2	2	3	2	4	2	1	0				
152	98.6	136	3.5	177	122	32	118			42.5	2	3	2	4	2	1	0				
14.1	150.8	138	3.4	176	176	32	113	4.2	1.58	38.4	2	3	1	1	2	1	0				
6.5	60.15	134	3.7	183	88	58	113	5.2		50.2	2	3	2	4	2	1	0				
430	10000	137	4.5	133	75	36	89			41.9	3	2	2	4	2	1	0				
55	1753	137	3.5	163	88	45	101	6		45	2	3	2	4	2	1	0				
5.7	95	139	4.4	97	105	38	45	4.9		48	2	4	2	4	2	1	0				
500	10000	134	3.8	188	102	35	126	5.9	2	33	2	4	2	4	2	1	0				
19	6399	137	3.8	189	151	37	134	5.9	1.4	42	2	3	2	4	2	1	0				
14.5	183.4	133	3.5	166	97	40	107	6		45.5	2	3	2	8	2	1	0				
14.5	140	132	3.7	128	60	42	75	8		48.6	2	7	2	4	2	1	0				
22.1	310	134	3.8	143	68	37	94	5.4		47.8	2	3	2	4	2	1	0				
12.2	109.5	132	4.1	233	186	50	162	7.5		38	2	3	2	3	2	1	0	DAMA			

Annexure 4

Glossary for data sheet

Strategy

1 Primary
2 Pharmacoinvasive

Mode of transport

1 Ambulance
2 car
3 taxi
4 auto
5 two wheeler
0,6 NA
7 bus

1 Yes
2 No

CVS
1 Normal
2 LVS S#

RS
1 Normal
2 Crepitations

Diagnosis
1 AWMI
2 IWMI
3 LWMI
4 PWMI
5 RVMI+Lateral extension

Access site
1 radial
2 femoral

Angiographic profile
1 SVD
2 DVD
3 TVD
4 LM+TVD
5 LM+DVD
6 LM+SVD
7 recanalised vessel
8 not stented

Infarct related artery
1 LAD
2 LCX
3 RCA

Follw up at 30 days
1 alive
2 death
3 Don’t know

Mechanical complications
1 MR
2 No
3 LV clot

Gender
1 male
2 female

Stents
1 Biomatrix flex
2 supraflex
3 promus element
4 pronova
5 yukon choiceflex
6 xincev
7 vision bms

8 rapstrong
9 integrity BMS
10 could not stent
11 small vessel not stented
12 Supralimus
13 Endavor Resolute
14 Xience
Xpedition
15 AlisCR
16 Tecmic BMS
17 Multilink BMS
18 Resolute Integrity
0 recanalised

Other complications after
procedure

1 heart failure
2 arrhythmia
3 angina
4 nil
5 worsening of renal function
6 acute limb
ischemia
7 Re MI due to stent thrombosis
8 Ischemic stroke
9 Intracranial hameorrhage and other bleeding manifestations
10 Radial or femoral haematoma

In hospital cause of death
0 NA
1 Multiorgan failure
2 cardiac arrest with arrythmias
3 cardiac arrest
4 stent thrombosis