"Assessment of thrombotic and non-thrombotic troponin elevation"

A dissertation submitted to

THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

In partial fulfillment of DM-Branch II CARDIOLOGY

Examination to be held in August 2015

C E R T I F I C A T E

This is to certify that the dissertation entitled

"Assessment of thrombotic and non-thrombotic troponin elevation" is a bonafide work done by

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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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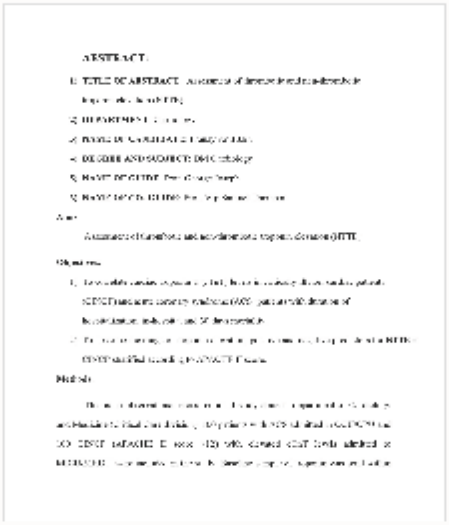

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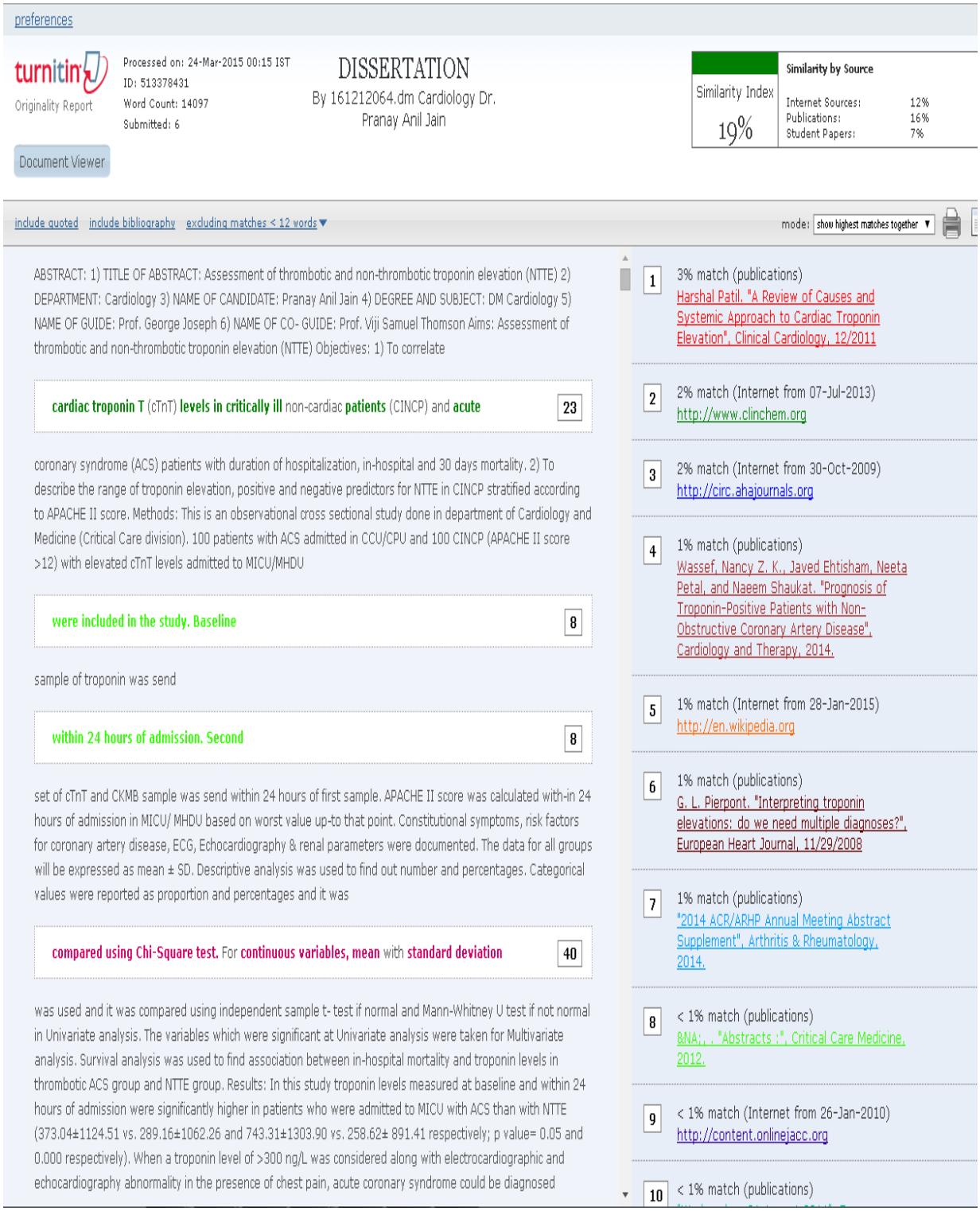

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ABSTRACT: 1) TITLE OF ABSTRACT: Assessment of thrombotic and non-thrombotic troponin elevation (NTTE) 2) DEPARTMENT: Cardiology 3) NAME OF CANDIDATE: Pranay Anil Jain 4) DEGREE AND SUBJECT: DM Cardiology 5) NAME OF GUIDE: Prof. George Joseph 6) NAME OF CO- GUIDE: Prof. Viji Samuel Thomson Aims: Assessment of thrombotic and non-thrombotic troponin elevation (NTTE) Objectives: 1) To correlate

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compared using Chi-Square test. For continuous variables, mean with standard deviation 40

was used and it was compared using independent sample t- test if normal and Mann-Whitney U test if not normal in Univariate analysis. The variables which were significant at Univariate analysis were taken for Multivariate analysis. Survival analysis was used to find association between in-hospital mortality and troponin levels in thrombotic ACS group and NTTE group. Results: In this study troponin levels measured at baseline and within 24 hours of admission were significantly higher in patients who were admitted to MICU with ACS than with NTTE (373.04±1124.51 vs. 289.16±1062.26 and 743.31±1303.90 vs. 258.62± 891.41 respectively; p value= 0.05 and 0.000 respectively). When a troponin level of >300 ng/L was considered along with electrocardiographic and echocardiography abnormality in the presence of chest pain, acute coronary syndrome could be diagnosed

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Dr. Pranay Anil Jain, Registrar, Cardiology, Dr. George Joseph, Cardiology, Dr. Viji Samuel Thompson, Cardiology, Dr. JV Peter, Critical Care.

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Finally I bow before almighty God.

Dated: Dr. Pranay Anil Jain

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ABSTRACT

Title of Abstract: **Assessment of thrombotic and non-thrombotic troponin elevation (NTTE)**

Department: **Cardiology**

Name of Candidate: **Pranay Anil Jain**

Degree and Subject: **DM Cardiology**

Name of Guide: **Prof. George Joseph**

Name of Co-Guide: **Prof. Viji Samuel Thomson**

Aims:

Assessment of thrombotic and non-thrombotic troponin elevation (NTTE)

Objectives:

1) To correlate cardiac troponin T (cTnT) levels in critically ill non-cardiac patients (CINCP) and acute coronary syndrome (ACS) patients with duration of hospitalization, in-hospital and 30 days mortality.
2) To describe the range of troponin elevation, positive and negative predictors for NTTE in CINCP stratified according to APACHE II score.

Methods:

This is an observational cross sectional study done in department of Cardiology and Medicine (Critical Care division). 100 patients with ACS admitted in CCU/CPU and 100 CINCP (APACHE II score >12) with elevated cTnT levels admitted to MICU/MHDU were included in the study. Baseline sample of troponin was send within 24 hours of admission. Second set of cTnT and CKMB sample was

send within 24 hours of first sample. APACHE II score was calculated with-in 24 hours of admission in MICU/ MHDU based on worst value up-to that point. Constitutional symptoms, risk factors for coronary artery disease, ECG, Echocardiography & renal parameters were documented.

The data for all groups will be expressed as mean ± SD. Descriptive analysis was used to find out number and percentages. Categorical values were reported as proportion and percentages and it was compared using Chi-Square test. For continuous variables, mean with standard deviation was used and it was compared using independent sample t-test if normal and Mann-Whitney U test if not normal in Univariate analysis. The variables which were significant at Univariate analysis were taken for Multivariate analysis. Survival analysis was used to find association between in-hospital mortality and troponin levels in thrombotic ACS group and NTTE group.

Results:

In this study troponin levels measured at baseline and within 24 hours of admission were significantly higher in patients who were admitted to MICU with ACS than with NTTE (373.04±1124.51 vs. 289.16±1062.26 and 743.31±1303.90 vs. 258.62± 891.41 respectively; p value= 0.05 and 0.000 respectively). When a troponin level of >300 ng/L was considered along with electrocardiographic and echocardiography abnormality in the presence of chest pain, acute coronary syndrome could be diagnosed with a sensitivity of 100%, specificity of 77.59%, and Positive Predictive value of 76.36%. Critically ill patients with high troponin levels had early

in-hospital mortality. (Pearson co-relation coefficient = -0.068 and -0.072; p value 0.501 and 0.479 respectively). In MICU group there was a positive trend toward increased mortality or re-hospitalization ≤ 30 days after discharge with elevated troponin T levels at baseline and within 24 hours of the first sample but this was not statistically significant (p value = 0.157 and 0.564). We found sepsis (77.59%), ARF (68.97%), anemia (43.10%), shock (31.03%), pneumonia (25.86%), ARDS (20.69%), myocarditis (12.07%), CHF (10.34%), tachyarrhythmia (6.9%), CPR (6.9%) as common causes associated with NTTE.

Conclusion:

The study showed that baseline troponin level was significantly associated with in-hospital mortality. In CINCP patients with troponin value >300 ng/L along with chest pain, ECG and ECHO abnormalities; acute coronary syndrome could be diagnosed with reasonable accuracy. The commonest causes of NTTE were renal failure, anemia, sepsis and septic shock.

Key words: Critically ill non cardiac patients, non thrombotic troponin elevation, ACS.

INTRODUCTION:

In the assessment of patients presenting to emergency department with acute onset chest pain; troponins now play a major role in diagnosing acute coronary syndrome (ACS) and its management. Cardiac troponin denotes myocardial necrosis and does not always signify major coronary artery disease. Troponin level elevation indicates myocardial injury but in critically ill patients it may occur without evidence of myocardial ischemia and can lead to incorrect diagnosis of ACS (**1-3).** In patients admitted to Medical intensive care unit (MICU) about 50% of them have troponin elevation without ACS (**4**).

Damaged cardiac myocytes releases different proteins into the circulation, measurement of which we can recognize myocardial necrosis. Cardiac proteins such as cardiac troponin T (cTnT) and I (cTnT), CKMB, LDH, myoglobulin, myeloperoxide and many others are released into blood with myocardial necrosis (**1)**. CKMB has been replaced as the gold standard by cardiac Troponins. For myocardial cell damage cTn is now the most sensitive and specific laboratory marker (**4-6).**

There is no specific International Classification of Diseases 9 (ICD-9) code for severe extra-cardiac problems such as acute respiratory distress syndrome (ARDS), sepsis or septic shock causing myocardial injury as evidenced by transient cardiac biomarker's elevations in serum. In the new Universal Definition of Myocardial infarction (MI) such conditions can be called as Type 2 MI (**7)**. Type 2 MI in such patients can be a 'working diagnosis', as a coronary artery lesion was not totally excluded (**8)**. Troponin is a sensitive marker to rule out Non ST elevation

myocardial infarction, but it is less useful to rule in this event as it has less specificity (**8)**.

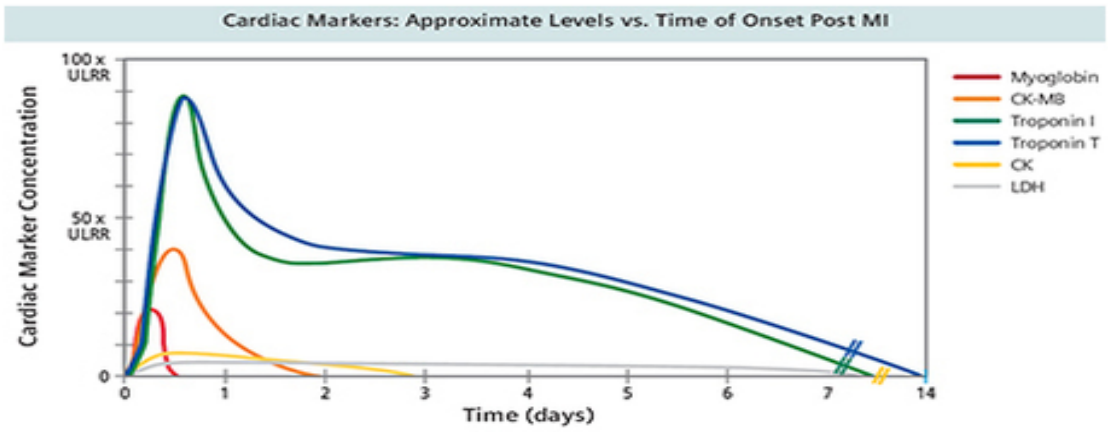


FIGURE 1: CARDIAC BIOMARKERS: APPROXIMATE SERUM LEVELS VS. TIME OF ONSET FROM ACS

In intubated or sedated patients with elevated serum troponin levels; symptoms cannot be used as a major guide for diagnosing ACS. Patients having pre-existing ST–T abnormalities, LBBB or paced ventricular rhythm; transient ECG changes can be missed. Critically ill non-cardiac patients commonly have relative contra-indications to anticoagulants or cardiac catheterization (diagnostic and therapeutic) due to underlying medical conditions. Non-invasive evaluation in such patients is difficult to perform and gives limited information. Even after performing coronary angiogram in such patients, the decision to label patients as having ACS is difficult if there is no clear cut evidence of visible thrombus, slow flow or no flow phenomenon visualized on angiogram. These patients may also have coronary artery lesion which are stable and longstanding and not contributing to the present condition

(**9**). Finally, critically ill patients have multiple stressors and pro-coagulant conditions which can cause acute progression in severity of pre-existing coronary artery lesion (Type 1). There can also be concomitant cell damage from hypo-perfusion or hypoxia of non coronary etiology (Type 2) making simultaneous occurrence of both types of MI a possibility (**8)**. In patients of Type 2 MI, the nature and severity of the illness inducing the cardiac biomarker elevation significantly alters the prognosis (**8).**

Non-thrombotic troponin elevation **(**NTTE) is defined as troponin elevation in the existence of an additional definitive diagnosis which is known to be associated with rise in troponin levels, with lack of sufficient criteria for diagnosing acute coronary syndrome as per universal definition given by ESC/ACC/AHA and serial samples showing <20 % rise or fall from first value i.e. constantly elevated at the same level. NTTE will also be considered when normal epicardial coronaries are demonstrated on coronary angiography in patients with troponin elevation (**10-12)**.

In this study we have attempted to determine the significance of NTTE in relation to in- hospital and 30 day mortality, duration of hospitalization and its association with non cardiac conditions which are commonly associated with the NTTE in critically ill non-cardiac patients (CINCP). The presence of such association would mean that patients with NTTE are at high risk which may benefit from intensive management. For clinical comparison we have also looked at cohort of patients in Coronary Care Unit (CCU)/ Chest Pain Unit (CPU) who presented with proven diagnosis of ST segment elevation myocardial infarction and Non ST elevation myocardial infarction (STEMI/ NSTEMI).

AIM:

Assessment of thrombotic and non-thrombotic troponin elevation

OBJECTIVES:

1) To correlate troponin levels in critically ill non-cardiac patients (CINCP) and ACS patients with duration of hospitalization, in-hospital and 30 days mortality.
2) To describe the range of troponin elevation, positive and negative predictors for non thrombotic troponin elevation (NTTE) in critically ill non-cardiac patients stratified according to APACHE II score.

REVIEW OF LITERATURE:

Troponins are proteins in thin filaments of sarcomere. It regulates the calcium-triggered interaction of actin and myosin. Troponin T and I has cardiac form, which differs in amino acid sequence from muscle form. These proteins can be measured with monoclonal antibodies directed at epitopes present only in cardiac form. Troponin C has a common isoform in cardiac and smooth muscle hence not used clinically. Troponin is a marker of myonecrosis, and does not always imply coronary artery disease (**1)**.

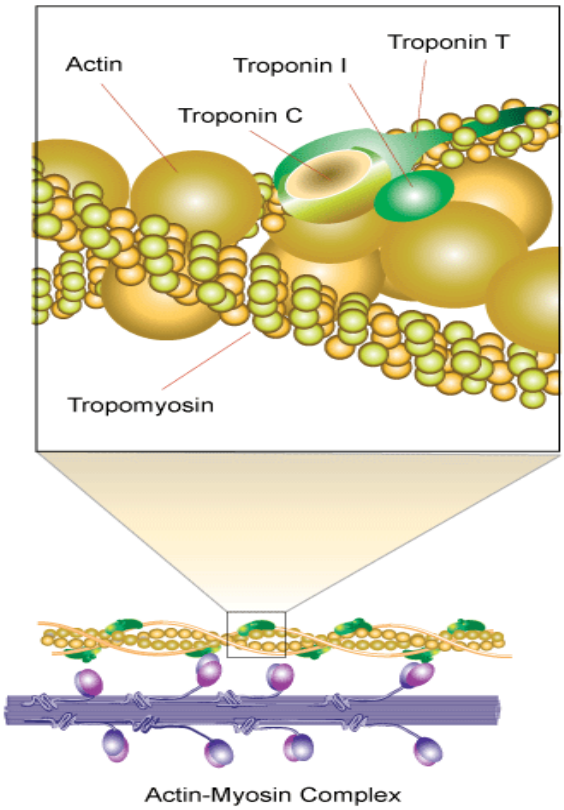


FIGURE 2: TROPONINS LOCATIONS IN RESPECT TO ACTIN AND MYOSIN

It has been documented that low level serum troponin elevation is chronically present in stable patients with diabetes mellitus, chronic renal failure, heart failure and

left ventricular hypertrophy (LVH) (**8)**. In approximately 25% to 33% of athletes after the marathon, transient serum troponin elevations above the normal range can be detected, with no evident functional consequences (**13-15)**. In critically ill non-cardiac patients assessing troponin elevations has became complicated due to growing list of causes of elevated troponin (**6, 10, 16-19).** Elevated serum troponin levels above 99th percentile can identify critically ill non-cardiac patients having a worse prognosis (**20 to 24).** In a study by Ammann et.al 72 % of troponin positive patients at autopsy or stress echocardiography did not showed any flow-limiting coronary artery stenosis (**20)**. As compared to patients with Type I MI prognosis may be worse for critically ill non- cardiac patients with serum troponin elevation without any other evidence of MI (**9, 11)**.

Term 'Secondary Unstable Angina' has been used by the ACC/AHA guidelines for management of unstable angina (UA)/non-ST-elevation myocardial infarction (NSTEMI) to portray enzyme elevation precipitated by a condition which is extrinsic to the coronary arterial bed (**25)**. In critically ill patients with troponin elevation, angina may not be present so terminologies such as 'non-thrombotic troponin elevation' (NTTE), 'non-specific troponin elevation', 'troponin leak', 'concomitant myocardial injury' and 'troponin positive non-ACS' ,have also been used (**11)**. A meta-analysis was performed by Fleming et al. of the published trials of ACS patients without ST elevation and found that cTnI and cTnT provide similar information in for adverse events (death and myocardial infarction) (**26)**.

CARDIAC TROPONIN T (27-39):

Troponin T occurs in three diverse isoforms, each encoded by individual genes. It has been recognized in cardiac muscle, fast-twitch and slow-twitch skeletal muscle (**27, 28)**. Cardiac troponin T has a sequence homology with skeletal muscle troponin T (sTnT), [56.6% homology- fast twitch sTnT and 56.6% homology- slow-twitch sTnT]. There is a differentiation of 125 amino acid residues between adult sTnT from fast-twitch skeletal muscle (fast sTnT) and cTnT and 120-residue differentiation between adult sTnT from slow-twitch skeletal muscle (slow sTnT) and cTnT (**28)**. The majority of cTnT is found within the contractile apparatus (~10 mg/g tissue). 6% to 8% of cTnT occur as a free cytosolic component (**29)**.

Apoptosis, normal myocyte turnover, cellular release of proteolytic degradation products, increased cell wall permeability, and formation and release of membranous blebs are some of the proposed mechanisms of cardiac troponin release. In circulation the half-life of cTnT is considered to be 120 min, with the extended window of detection being due to incessant release of cTnT from the myofibrillar pool as within cell there is degradation of the contractile apparatus during necrosis (**29)**. In patients with renal disease there is no evidence that adult TnT in skeletal muscle has any corresponding isoform which cross reacts with adult cTnT on use of the antibody pair specific to adult cTnT (**31)**.

The high sensitivity-cTnT assay uses fragment antigen binding (FAB) portions of two cTnT-specific mouse monoclonal antibodies (MAbs) directed against epitopes in the central region of human cTnT (**32)**.

CARDIAC TROPONIN I (27 -39):

A single isoform of human Cardiac troponin I (cTnI) occurs in cardiac muscle tissue. It has 209 amino acid residues and molecular weight in the order of 23–24 kDa. Three human cTnI isoforms have been described: one each is produced in cardiac muscle (cTnI), slow-twitch and fast-twitch skeletal muscles (slow sTnI and fast sTnI, respectively). Approximately 40% sequence overlap occurs between cTnI and slow sTnI and fairly less overlap for fast sTnI (**27, 28)**. Antibodies preferred for cTnI assays can have cross-reaction with slow and fast skeletal isoforms of the troponin (**27)**.

Protein Kinase A can phosphorylate the two serine residues in cTnI molecule. Thus cTnI can coexist in the cell in different forms: dephosphorylated, monophosphorylated, and bisphosphorylated form. Interaction of cTnI with anti-TnI antibodies is modified by phosphorylation, as it changes conformation of proteins this modifies its interaction with other troponins. In circulation post-translationally modified TnI isoforms that are oxidized, reduced, or partially digested by proteases and phosphorylated isoforms appears in blood stream (**33)**.

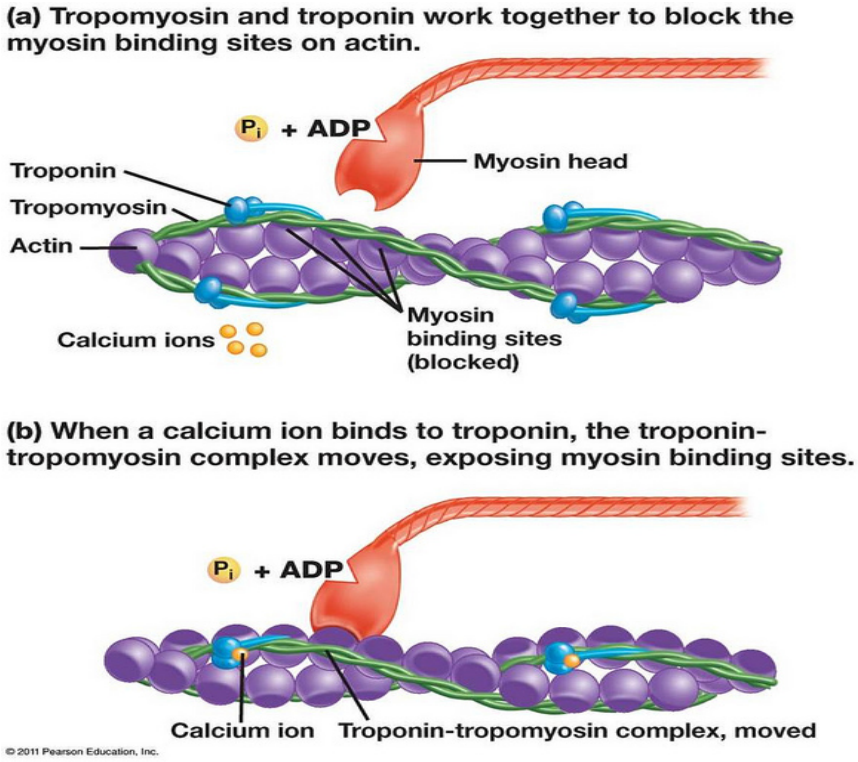


FIGURE 3: ROLE OF CALCIUM IN TROPONIN-TROPOMYOSIN COMPLEX - MECHANISM OF ACTION

The greater part of cTnI is found in the contractile apparatus (~ 4–6 mg/g tissue). 2% to 8% of cTnI occurs as a free cytosolic component. It is released by proteolytic degradation (**34)**. Cummins et al. described the first cTnI immunoassay in 1987 (**35)**. All known cTnI modifications circulating in the blood are recognized by monoclonal antibodies (MAbs) directed to selected epitopes of cTnI. Multiple factors influence cTnI measurement like the posttranslational modifications [phosphorylation, proteolytic degradation] and complexing with other molecules [e.g., human antimouse antibodies or heterophile, TnC, heparin and cTnI-specific autoantibodies circulating in patients' blood] (**36)**. It is recommended that in development of precise immunoassays combinations of >2 MAbs to be used. Antibodies used in the assay should identify

cTnI in complex with TnC as in human blood >95% of cTnI occurs as a binary cTnI–TnC complex. Ideally, all circulating cardiac troponin forms should be recognized by antibodies used in assays on an equimolar basis **(27)**.

Using commercial cTnT and cTnI assays which was measured on collected fractions, it was instituted that troponin is released into blood as free cTnT, a binary complex of cTnI-C, a ternary complex of cTnT-I-C and with no free cTnI within the confines of the analytical methodologies **(27)**.

TABLE 1: LIMIT OF DETECTIONS OF DIFFERENT TROPONIN GENERATIONS:

TROPONIN	**LIMIT OF DETECTION**
FIRST GENERATION	0.3 µg/L
SECOND GENERATION	0.05 µg/L
THIRD GENERATION	0.01-0.02 µg/L
FOURTH GENERATION	0.03 ng/ml
FIFTH GENERATION	3 ng/L

HIGH SENSITIVITY ASSAY (27, 40, 41):

The term “high sensitivity” reflects the assay's characteristics and does not refer to a difference in the form of cardiac troponin being measured.

- It was proposed that the assay would be labeled to be high sensitive if it met two essential criteria:

1) The total imprecision (CV) at the 99th percentile value should be ≤10%.
2) Measurable concentrations below the 99th percentile should be attainable with an assay at a concentration value above the assay's limit of detection for at least 50% (and ideally >95%) of healthy individuals.

- Concentrations for high sensitivity assays are expressed in nanograms per liter (ng/L) or picograms per milliliter (pg/ml).
- Increase in assay sensitivity is achieved by:

1) The sample volume was increased from 15 to 50 μL.
2) The detection antibody ruthenium concentration was increased.
3) Buffer optimization- lowers the background signal.

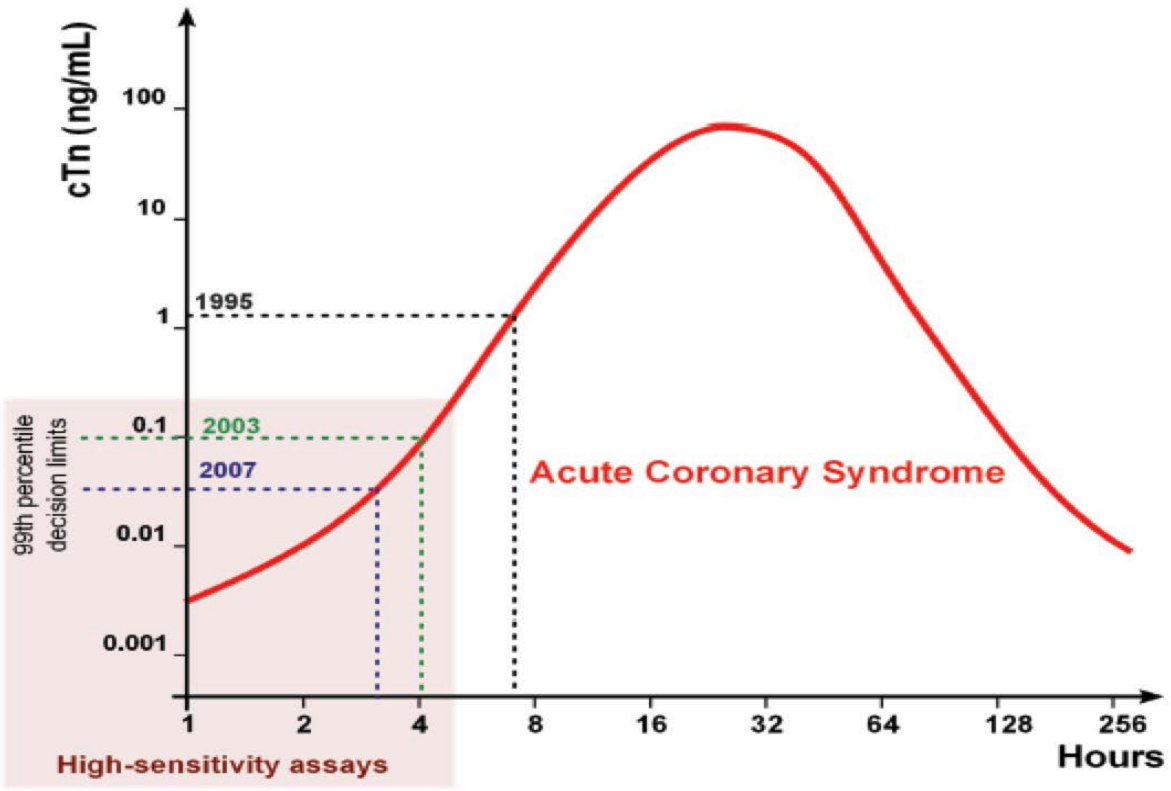
- The calibration of assay is done against Escherichia coli cell culture produced recombinant human cTnT.
- Fourth- generation assay cutoff of 30-ng/L equals to, 50 ng/L of the new high sensitivity assay.

- UNITS:

1) Units are changed now the results are reported as whole numbers (old units x 1000).
2) Current 0.03 limit is in **ng/ml:**
3) 03 **ng/ml** = 0.03 **µg/l** = 30 **ng/l** = 30 **pg/ml**
4) Proposed change use **ng/l** or **pg/ml** when **hsTnT** adopted threshold for 'elevation' (99th percentile) at 14 ng/L (0.014 ng/ml).

TABLE 2: HIGH SENSITIVITY TROPONIN ASSAY **(27)**

ROCHE ELECSYS (COMPANY ASSAY)	CARDIAC TROPONIN CONCENTRATION AT:			AMINO ACID RESIDUES OF EPITOPES RECOGNIZED BY CAPTURE © AND DETECTION (D) MAbs
	LIMIT OF DETECTION (ng/L)	99% PERCENTILE (ng/L)- CV	10% CV Concentration (ng/L)	
	5.0	14 (8%)	13	C:136-147;d125-131



cTn Assay	Diagnostic cutoff	Implementation
TnI	≥ 1.5 ng/mL	1995
cTnI	> 0.10 ng/mL	2003
TnI-Ultra	> 0.04 ng/mL	2007

FIGURE 4: DIFFERENCES IN GENERATIONS OF TROPONIN ASSAY

In meta-analysis of troponin T trials it was found to have PPV of 22.4% and NPV of 94% when sample timing was after 6 hours from onset of pain. Current assays for troponin estimation use population defined upper limit of 99th percentile for normality with coefficient of variation of less than 10% (**25, 26, 42-45)**. Increased sensitivity can be achieved with sampling troponin every 6 to 8 hours (**25)**.

Myocardial necrosis leads to disruption of sarcolemmal membrane, disintegration of myofilament and subsequently macromolecules (serum cardiac markers) disperse into the cardiac interstitium and finally within microvasculature and lymphatic's. Initially the troponin from cytoplasmic pool is released. Cardiac troponin

levels increase to twenty to fifty times the upper reference limit (URL) in ACS patients with elevated CKMB levels. Troponin T is detected within 3 hours of myocardial ischemia, peaks in 12 to 24 hours and return to normal in 5 to 14 days. In patients with large reperfused myocardial infarction cTnT shows a characteristic biphasic time release pattern as compared with the monophasic release pattern that is visualized with cTnI. Idea regarding quality of micro vascular reperfusion may be given by the early appearing pool of troponin, whereas myocardial infarct size is reflected by the concentration of troponin T on 3rd or 4th day (**29)**.

UNIVERSAL DEFINITION OF MYOCARDIAL INFARCTION:

Universal definition of myocardial infarction **(ACS)** as given by ESC/ACC/AHA includes typical rise and/or fall of biochemical markers of myocardial necrosis preferably cardiac troponins with at-least one value above 99th percentile of URL as major factor for diagnosis of ACS (**5)**. The NACB recommends a 20% alteration from baseline value to be suggestive of and MI that is either evolving (delta positive) or resolving (delta negative).

As given by **Thygesan et.al (5)** the term myocardial infarction should be used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischemia. Under these conditions, any 1 of the following criteria meets the diagnosis for MI-

1. Detection of rise and/or fall of cardiac biomarkers (preferably Troponins) with ≥1 value above the 99th percentile of the URL together with evidence of myocardial ischemia with ≥1 of the following:

a. Symptoms of ischemia

b. ECG changes indicative of new ischemia (new ST-T changes or new LBBB)

c. Development of pathological Q waves in the ECG

d. Imaging evidence of new loss of viable myocardium or new regional wall-motion abnormality

e. Pathological findings of an acute MI.

The troponin value below 99th percentile has a very high negative predictive value for ACS. Type 2 MI as per ESC/ACC/AHA classification is due to supply demand mismatch without CAD. Myocardial infarctions can be classified temporally from clinical and other features, as well as according to the pathological appearance, as evolving (<6 h), acute (6 h–7days), healing (7–28 days), and healed (29 days and beyond). Myocardial infarctions are usually classified by location of the infarct and by size: microscopic (focal necrosis), small [10% of the left ventricular (LV) myocardium], moderate (10–30% of the LV myocardium), and large (>30% of the LV myocardium) (**9**, **29)**.

ISCHEMIC SYMPTOMS:

A discomfort or pain (in a variety of combinations) in chest, jaw, left or right shoulder epigastric, wrist or arm occurring with exertion or at rest, persisting for at least 20 minutes, with radiation to the arm, hand, jaw, shoulder or back. It could be associated with diaphoresis, dyspnea, nausea, vomiting, or lightheadedness. The discomfort or pain is not exaggerated or decreased by pressure, movements and

position of body. Patient with ACS might not have typical symptoms. The atypical symptoms may comprise (but are not restricted to) of weakness, extreme fatigue, apprehension, mental confusion, anxiety, giddiness, syncope, nervousness and psychosis. Presentation with atypical symptoms is more common in females, old age, demented and diabetic patients. These symptoms are not precise for ACS and can also be caused by disorders associated with gastrointestinal, neurological, pulmonary, or musculoskeletal systems; thus, further assessment is needed to complement the clinical history.

Types of Myocardial infarction (Joint ESC/ACCF/AHA/WHC Task force) (5):

1) Type 1**:** Spontaneous myocardial infarction
2) Type 2: MI secondary to an ischemic imbalance
3) Type 3: MI resulting in death when biomarker values are unavailable
4) Type 4a: MI related to PCI. (By convention, increases of biomarkers >3 ×99th-percentile URL have been designated as defining PCI-related MI.)
5) Type 4b: MI related to stent thrombosis (as documented by angiography or at autopsy.)
6) Type 5: MI related to CABG (By convention, increases of biomarkers >5 ×99th-percentile URL either new pathological Q waves or new LBBB, or angiographically documented new graft or native coronary artery occlusion, or imaging evidence of new loss of viable myocardium, have been designated as defining CABG-related MI)

ELECTROCARDIOGRAM (46):

I) ECG manifestations of acute Myocardial Ischemia (in Absence of LVH and LBBB):

1) New ST elevation at the J-point in 2 contiguous leads with the cutoff points: ≥0.2 mV in men or ≥0.15 mV in women in leads V_2 through $V_3$and/or ≥0.1 mV in other leads.
2) New horizontal or down sloping ST depression ≥0.05 mV in 2 contiguous leads; and/or T inversion ≥0.1 mV in 2 contiguous leads with prominent R wave or R/S ratio >1.

II) ECG Changes Associated With Prior MI:

1) Any Q wave in leads V_2 through V_3 ≥0.02 s or QS complex in leads V_2 and V_3.
2) Q wave ≥0.03 s and ≥0.1 mV deep or QS complex in leads I, II, aVL, aVF, or V_4 through V_6 in any 2 leads of a contiguous lead grouping (I, aVL,V_6; V_4 through V_6; II, III, and aVF)
3) R wave ≥0.04 s in V_1 through V_2 and R/S ≥1 with a concordant positive T wave in the absence of a conduction defect.

ECHOCARDIOGRAPHY IN ACUTE CORONARY SYNDROMES: (47-57)

(ACC/AHA/ASE Guidelines, 2011 appropriate use criteria for echocardiography)

Regional wall motion abnormalities (RWMAs) –

1) It is defined as a localized decrease in the rate and amplitude of myocardial excursion with a blunted degree of myocardial thickening and associated local remodeling (**47)**.

2) RWMA is produced by ischemia which is visualized within seconds on echocardiography after coronary artery occlusion. In two series after transient coronary artery balloon occlusion within 12±5seconds and 19±8 seconds respectively RWMAs was visualized (**48, 49)**.

3) If RWMA is due to acute ischemia the wall thickness and reflectivity will be preserved, but if RWMA is chronic, the wall will be akinetic and reflective (**50)**.

4) Ischemic RWMAs occurs prior to symptoms onset, chest pain occurring without RWMAs active myocardial ischemia is probably ruled out.

5) RWMAs can occur due to prior infarction, prior surgery, focal or localized myocarditis, accessory pathway causing ventricular preexcitation, left bundle branch block, and cardiomyopathy.

Study by **Sabia et.al** in 180 patients coming to emergency department with chest pain showed that echocardiography has a high sensitivity but a relatively lower specificity (**51).** Study results were as follows: In patients with ACS 27 of 29 had RWMA (93% sensitivity), RWMA suggested ACS in 31% patients and only 2.2% patients without RWMA were diagnosed as having NSTEMI on basis of cardiac enzymes (**51)**.

Myocardial ischemia is diagnosed if there is appearance of reversible RWMAs and reversible ECG changes (**52)**.

NON THROMBOTIC TROPONIN ELEVATION (NTTE) (**10-12)**:

NTTE is defined as

1) Troponin elevation in the existence of an additional definitive diagnosis which is known to be associated with rise in troponin levels, with lack of sufficient criteria for diagnosing acute coronary syndrome as per universal definition given by ESC/ACC/AHA and serial samples showing <20 % rise or fall from first value i.e. constantly elevated at the same level.
2) NTTE will also be considered when normal epicardial coronaries are demonstrated on coronary angiography in patients with troponin elevation.

Non-thrombotic troponin levels elevation has been seen in many more conditions other than ACS. Troponins have compelling clinical values as it has superior potential in predicting the prognostic outcome of patients with elevated levels.

CAUSES OF NONTHROMBOTIC TROPONIN ELEVATION (1, 58):

• Chronic or acute renal dysfunction

• Severe congestive heart failure—acute and chronic

• Hypertensive crisis

• Tachy- or brady-arrhythmias

• Pulmonary embolism, severe pulmonary hypertension

• Inflammatory diseases, e.g., myocarditis

• Acute neurological disease, including stroke or subarachnoid hemorrhage

• Aortic dissection, aortic valve disease or hypertrophic cardiomyopathy

• Cardiac contusion, ablation, pacing, cardioversion, or endomyocardial biopsy

• Hypothyroidism

• Apical ballooning syndrome (Takotsubo cardiomyopathy)

• Infiltrative diseases, e.g., amyloidosis, hemochromatosis, sarcoidosis, scleroderma

• Drug toxicity, e.g., Adriamycin, 5-fluorouracil, Herceptin, snake venoms

• Burns, if affecting [≥30% of body surface area]

• Rhabdomyolysis

• Critically ill patients, especially with respiratory failure, or sepsis.

• Vital exhaustion.

• Transplant vasculopathy

TABLE 3: SERUM TROPONIN CONCENTRATION AND ASSOCIATED CONDITIONS AS PER SERUM LEVEL**:**

Troponin concentration (µg/L)	**Clinical Conditions**
100	Large myocardial infarction
10	Medium sized myocardial infarction, Severe myocarditis
1	Small myocardial infarction, myocarditis, pulmonary embolism, shock.
0.1	Micro myocardial infarction, pulmonary embolism, shock, acute heart failure, renal failure (acute/chronic), subarachnoid hemorrhage.
0.01	Stable angina, chronic heart failure, left ventricular hypertrophy, subclinical heart disease,
0.001	Healthy individual

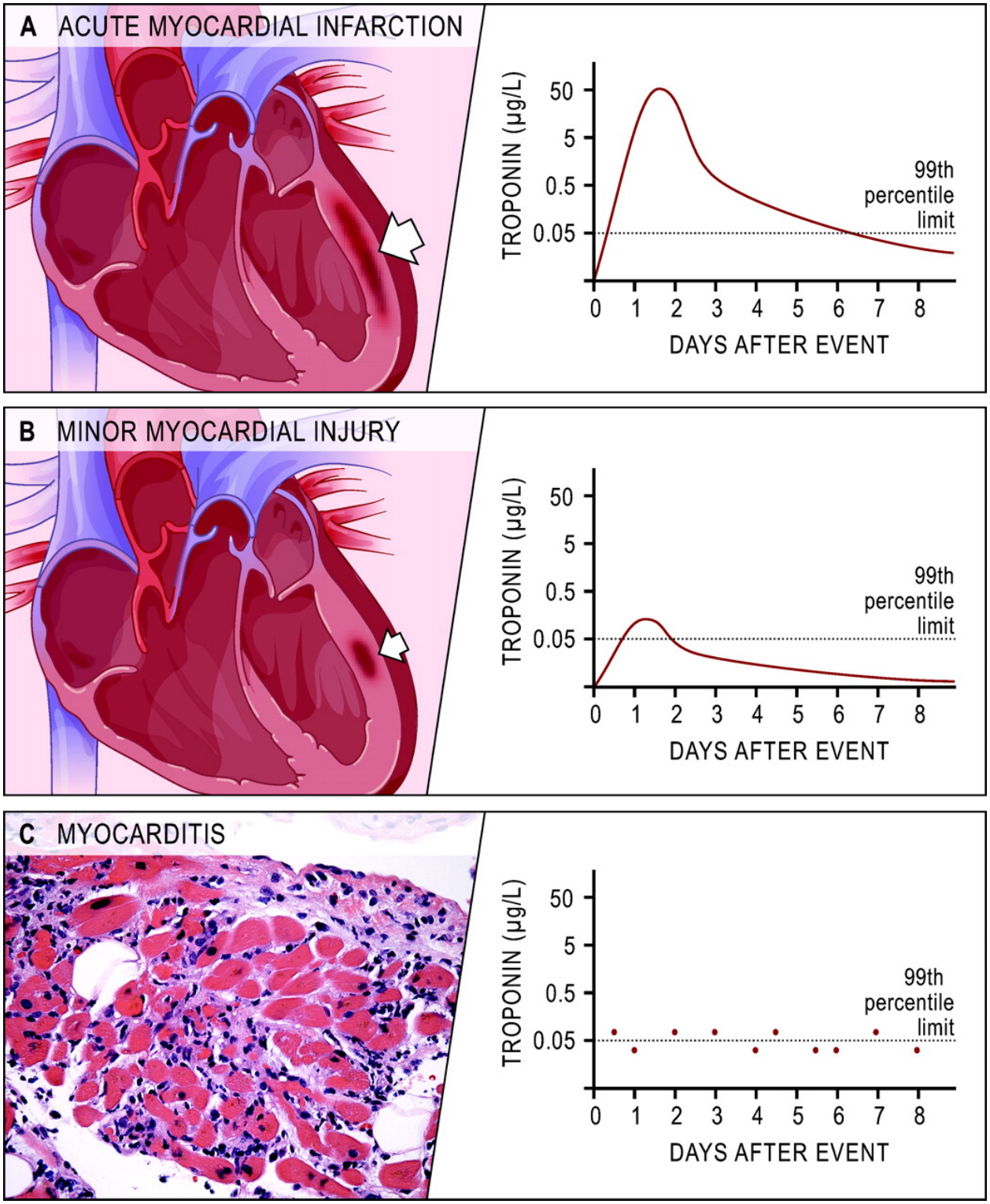


FIGURE 5: TROPONIN LEVEL VARIATION AMONG DIFFERENT EVENTS

In acute pulmonary embolism, serum troponins perhaps rise due to acute right heart strain and overload. The troponin release as compared to unstable angina is of shorter

duration, and the peak level of troponin has prognostic value linked to the outcome (**59, 60).**

The reasons for troponin T and I elevations in patients with severe renal dysfunction are not yet convincingly explained but it cannot be linked to myocardial injury. Different analyses and investigators excluded re-expression of cardiac isoforms in skeletal muscles **(61, 62)**. Due to impaired renal excretion normal low level troponins are amplified, also membrane integrity of myocytes is lost and there is constant outpouring from the free cytosolic troponin pool leading to abnormal troponin values (**19)**. Significantly higher biological variation was seen in end-stage renal disease (ESRD) patients as compared to healthy reference individuals. In ESRD patient's baseline serum cardiac troponin T concentration was more than 99th percentile of the healthy reference population and as the duration of haemodialysis increased the serum troponin level progressively increased. As compared to cTnI, Troponin T has high unbound cytosolic pool and molecular weight. This explains frequent elevation of cTnT as compared to cTnI in renal failure patients (**19)**. When the cut off limit of 99^{th} percentile was used in ESRD patients only 6% patients had increase in cTnI while 82% patients had an increase in cTnT (**63)**. In GUSTO IV trial it was convincingly demonstrated that troponin T has predictive value for cardiac events in patients presenting with chest pain across all creatinine clearance levels (**64)**. A study by Dierkes et al. in 102 ESRD patients found that cardiac troponin T is an important predictors of mortality (**65)**. The prospective study by Apple et al documented 2- to 5-fold increase in one, two and three year all-cause mortality associated with levels of troponins. Independent of other variables, gradual rise in risk

occurs with rise in troponin T levels at various discriminator levels (**63)**. After kidney transplantation the lower troponin levels was also associated with improved outcome (**65)**. Studies suggest that if baseline serum troponin concentration is increased, additional rise in concentration higher than that at baseline occurs in acute ischemic injury. Consequently acute rise in serum troponin levels can be discriminated from more chronic persistent elevations when a rising pattern of results is seen (**60, 66, and 67)**.

Microembolization is a cause of troponin elevation. It induces inflammatory responses and might occur as a single or as multiple, repetitive events. When the fibrous cap of an atheroma ruptures, the lipid pool with or without added thrombus formation diffuses out of atheroma in the microcirculation causing spontaneous microembolization. Such phenomenons with progressive increase in thrombus burden are described as cyclic flow variations. The incidence of plaque rupture is more as compared to previous assumptions, i.e. in patient with normal structural heart having traffic accidents 9% had plaque rupture and similarly in diabetic and hypertensive patients 22% had event. Microembolisation is commonly found in patients with sudden death. In patients with normal epicardial coronaries on angiogram with ACS, ischemic and diabetic cardiomyopathy pathogenesis can be explained better by appreciation of microembolisation. During PTCA rise in cTnI and cTnT and associated ST segment changes in ECG is caused due to microembolization (**68, 69)**.

Increases serum cTn levels are frequently seen amongst critically ill patients, e.g., patients with sepsis and it is co-related with prognosis. It is strongly

associated with the grade of left ventricular dysfunction and the requirement for inotropic support (**70, 71)**.

Troponins are regularly elevated in histologically confirmed myocarditis, but in patients suspected of having myocarditis clinically 50% also shows rise in cTn levels. Abnormal troponins levels are more likely in the diffuse myocarditis than in focal disease. Troponins prognostic value in assessment of progression of left ventricular dysfunction in a patient of myocarditis has not yet been convincingly established (**72, 73).**

In study done by Mehta et al. patients on coronary angiogram having < 50% stenosis of any major coronary artery with elevated serum troponin levels (n = 83) were compared to patients with negative troponin levels. It was found that there was an increased incidence of re-infarction and death over a period of 2.5 years follow-up in the patients with elevated troponin and non-obstructive-epicardial CAD. The risk of death and MACCE at thirty days and one year was equal in the patients with elevated troponin levels and non-obstructive CAD with recognized cause as compared to patients with obstructive CAD (**73)**.

Sanchis et al. did a study on 1372 patients with non-ST-segment elevation acute chest pain indicated that the replacement of conventional cTn by hs-cTn patients increased pharmacological treatments for acute coronary syndrome and the number of coronary angiograms and revascularizations performed at the index episode, while the number of patients initially evaluated with non-invasive stress tests in the chest pain unit decreased. Despite of all clinical outcomes at 6 months were not changed (**74)**.

Javed et al. studied 701 patients with elevated troponin I found that among them only a minority of patients had ACS by the universal definition. The criteria of ACS were fulfilled by 216 patients (30.8% with increased TnI). Type 1 MI was diagnosed in 143 patients (20.4%), Type 2 MI was diagnosed in 64 patients (9.1%), while the criteria for ACS was not fulfilled by 461 patients (65.8%). ACS can be diagnosed with 70% sensitivity and specificity at Troponin I level of 0.28 ng/ml. Type 1 AMI patients had increased chances of undergoing angiography and it was the most common AMI. Higher TnI values were seen with Type I AMI patients as compared to other patients. In the study illicit drug use was most common cause of Type 2 AMI (**12)**.

Study by Ammann et.al showed that ICU patients with rise in serum cT levels above 99th percentile, >70% patients on stress echocardiography or at autopsy did not have flow-limiting epicardial coronary artery stenosis (**20).**

S Hajsadegh et al. studied one hundred thirty five patients admitted to the medical ICU. Patients mean age was 60.9 ± 21.5 years. Four blood samples were taken for troponin levels for evaluation. First sample was send within twenty four hours of ICU admission and then on the fourth, seventh and tenth days samples were send. Among the deceased and discharged patients the alteration in cTnT levels was not significantly different (p = 0.4). There was no significant trend visualized between changes in cTnT levels during ICU stay (power: 0.26). Worst survival was noted in patients whose cTnT levels were increased on the 1st and 7th days of ICU stay. This could be related with cardiac events on admission or at specific times during the stay in ICU (**75).**

Relos RP et.al studied, 58 critically ill non-cardiac patients found that elevated troponin levels on admission were associated with a fourfold increased risk of mortality (**76).**

Vlad et.al studied in 2078 patients with ARDS, and found that high levels of cTnT on admission had an independent association with in-hospital, short and long-term mortality (**77).**

Altmann et al. studied 38 patients with sepsis, septic shock or systemic inflammatory response syndrome (SIRS) without evidence of an ACS. Out of 38, 22 (58%) patients were cTnI-positive. They analyzed coagulation parameters using rotational thrombelastometry and found no differences between cTnI-positive and -negative patients with SIRS, severe sepsis, and septic shock. They concluded that pathophysiological mechanisms other than thrombus-associated myocardial damage might play a major role, including reversible myocardial membrane leakage and/or cytokine mediated apoptosis in these patients (**78).**

Brekke et al. studied 396 COPD patients with acute exacerbation; elevated cTnT was associated significantly with increased all-cause mortality including in-hospital mortality and after discharge (**79).**

In general ICU critically ill non-cardiac patients increased serum troponin levels prevalence ranges from 15% to 70%, and similarly the prevalence is 31% to 80% in critically ill patients with sepsis or septic shock. Estimated prevalence of non-cardiac diagnoses in NTTE ranged from 16% to 55% **(19, 80).**

Mcfalls et.al showed that in 21,668 patients with elevated serum troponin 57.2% (12,400) patients had NTTE. Among NTTE patients CHF, Chronic CAD attributed mainly for NTTE. They found higher mortality in NTTE patients (22.8%) as compared to ACS patients (Odds Ratio=1.39; 95%CI: 1.30–1.49) (**81)**.

APACHE II SCORE ("**Acute Physiology and Chronic Health Evaluation II**"):

It is a severity-of-disease classification system given by **Knaus et al. (82**). It is applied to critically ill patients within 24 hours of admission to intensive care unit (ICU). Based on several measurements an integer score is computed from 0 to 71. Patients with higher APACHE II scores correspond to more severe disease and a higher risk of death. APACHE II score was designed to measure the severity of disease for adult patients admitted to intensive care units. APACHE II score is not validated for patient's age ≤ 16.

The point score is calculated from a patient's age and twelve routine physiological measurements as follows:

1. (A-a)ΔO2 or PaO2. (If FiO2 < 50% PaO2 is used; If FiO2 ≥ 50% A-a gradient is used)
2. Temperature (rectal)
3. Mean arterial pressure
4. Arterial pH
5. Heart rate
6. Respiratory rate
7. Serum Sodium

8. Serum Potassium
9. Serum Creatinine
10. Hematocrit
11. Total white blood cell count
12. Glasgow coma scale

These were measured during the first 24 hours after admission, information about previous health status, and some information obtained at admission (such as age). The score is not recalculated during the stay; it is by definition an admission score. If a patient is discharged from the ICU and readmitted, a new APACHE II score is calculated. Patient's prognosis (specifically, predicted mortality) was computed based on the patient's APACHE II score in combination with the principal diagnosis at admission.

It has been seen that critically ill non-cardiac patients with increased troponins were having increased proinflammatory cytokine, TNF alpha, IL6, CRP and associated with increased mortality. It has been found that there is a negative correlation between troponin levels and LV systolic function (LVEF). Commonest cause of elevated troponin level in critically ill non-cardiac patients was ACS in around 50% to 60% of patients. Elevated cardiac troponin serum levels indicate myocardial injury but underlying mechanism can be multifactorial, signifying that to establish a diagnosis of ACS clinical evidence of coronary artery thrombosis is required (**83)**. Troponins in ACS patients suggests that abnormal concentration of troponins almost always represents some irreversible myocardial injury, but in

critically ill non-cardiac patients the cardiac troponins might be released due to reversible myocardial ischemia without irreversible myocardial damage.

Cardiac troponin levels correlate with higher risk of death and recurrent ischemic event in patients with ACS. In a medical ICU, critically ill non-cardiac patients with elevated serum levels of troponins exhibited a fourfold higher mortality rate (**77).** Similarly in critically ill non-cardiac patients in a surgical ICU, moderate elevations in troponin I were associated with increased duration of ICU and hospital stay and higher mortality rates (**84, 85)**. Mortality in patient with non-thrombotic troponin elevation is many folds higher as compared to patient with ACS (**86)**. Various studies has show that patients with a diagnosis of myocardial infarction benefit from thrombolytic therapy, coronary revascularization, and use of anticoagulants, antiplatelet agents, β-blockers, statins, and angiotensin-converting enzyme inhibitors. However patients with non-thrombotic troponin elevations may not respond favorably to antithrombotic agents and the impact of these therapies on outcomes in ICU patients with myocardial infarction is unknown. Understanding the etiology of troponin elevation in critically ill non-cardiac patients will help to better target appropriate therapies for ACS in these patients. This study will show significance of thrombotic and non-thrombotic troponin elevation, its clinical importance in early risk stratification and prognostication.

MATERIAL AND METHODS:

The study was conducted in department of Cardiology and Medicine-division of Critical Care, Christian Medical College Vellore. The study subjects were selected from 100 critically ill non-cardiac patients (APACHE II score ≥12) with troponin elevation more than 99th percentile of the Upper Reference Limit (URL) i.e. ≥14 ng/L or pg/ml and 100 patients with ACS admitted in MICU/HDU and CCU/CPU respectively in our hospital. The study was conducted for 18 months from August 2013 to December 2014 in the department of Cardiology and Medicine-division of Critical Care.

TYPE OF STUDY:

Observational cross sectional study.

INCLUSION CRITERIA:

1) Age ≥ 18 years
2) Patients of ACS admitted in CCU/CPU
3) Critically ill non-cardiac patients (APACHE II score ≥ 12) with elevated troponin levels (≥ 14 pg/ml or ng/L) admitted in MICU/MHDU**.**

EXCLUSION CRITERIA:

1) Consent not given by patient/relatives

2) Out of hospital cardiac arrest who died within 48 hours of hospitalization

3) Age < 18 years

METHODS:

1) Serum troponin measurement within 24 hours of admission (Baseline sample).

2) Second set of troponin and CKMB sample was send within 24 hours (preferably within 6 to 8 hours) of baseline sample (Second sample)

3) APACHE II score was calculated with-in 24 hours of admission in MICU/ MHDU based on worst value up-to that point.

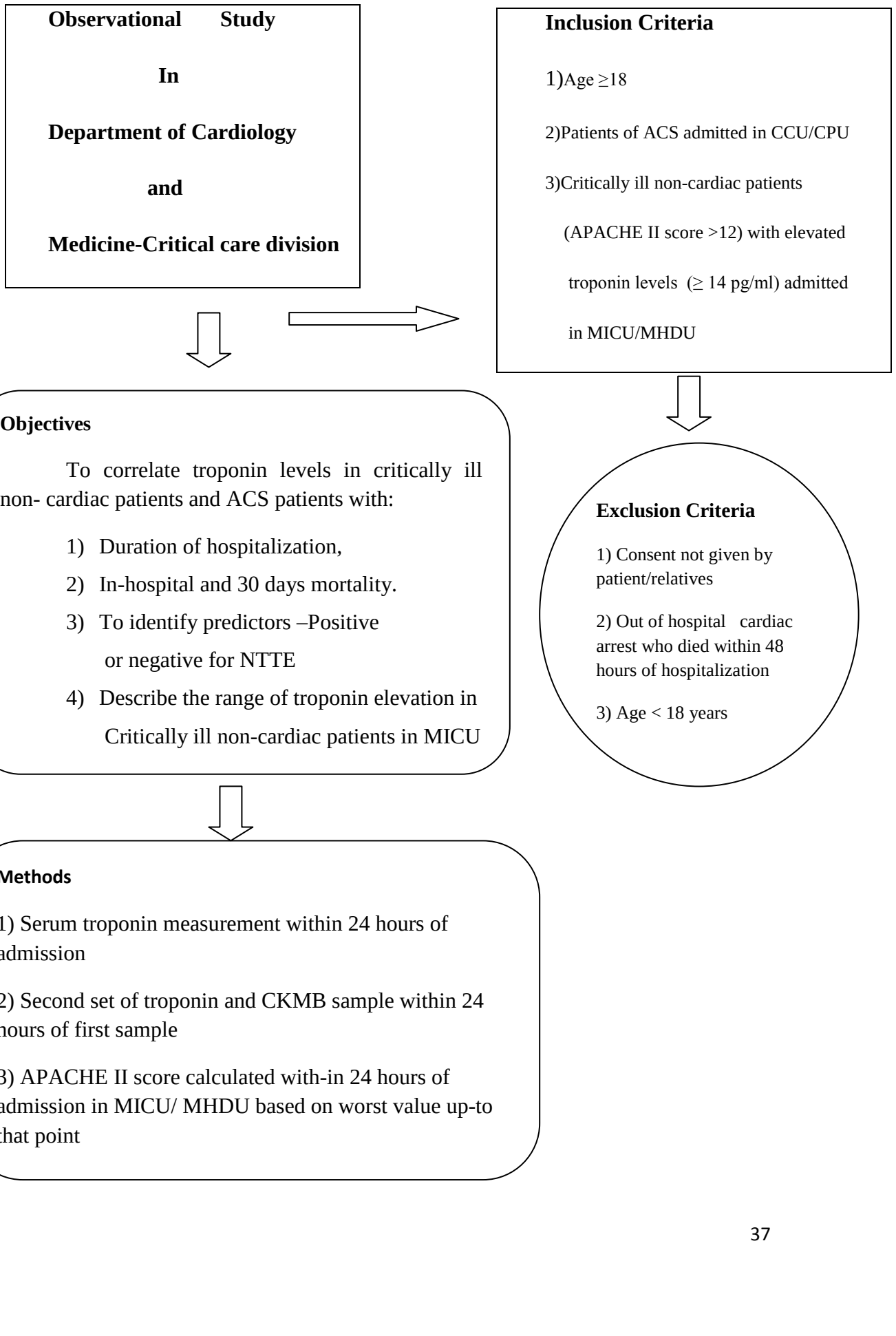
We collected details of the patients based on their clinical presentation, in which we looked for constitutional symptoms, risk factors for coronary artery disease, ECG, Echocardiography -LV systolic function, RWMA & renal parameters on the basis of thorough history taking and clinical examination. Details of patients were entered in a detailed pro forma. If patient was not able to give history it was assessed from relatives of the patient.

Troponin baseline sample was send by the respective Unit looking after the patient on clinical suspicion of ACS and patients with elevated troponin level above 99^{th} percentile of URL for normal population ($\geq$14 pg/ml) within 24 hours of admission were included in the study. Second set of troponin and CKMB samples were send within 24 hours (preferably within 6 to 8 hours) of baseline sample.

All patients were treated as per standard protocols and guidelines. Statistical analysis was performed to correlate troponin levels in critically ill non-cardiac patients and ACS patients with duration of hospitalization, in-hospital and 30 days mortality.

We studied the demographic profile of these patients. We looked for predictors (age, sex, diabetes mellitus, hypertension, past history of coronary artery disease, renal function, troponin levels) which are positively and negatively associated with NTTE in critically ill non-cardiac patient with elevated troponin levels.

DETAILED DIAGRAMMATIC ALGORITHM OF THE STUDY:



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b. ECG changes indicative of new ischemia (new ST-T changes or new LBBB)

c. Development of pathological Q waves in the ECG

d. Imaging evidence of new loss of viable myocardium or new regional wall-motion abnormality

e. Pathological findings of an acute MI.

PREDICTORS OF ACS:

Following were used as predictors of ACS in the study

1) Chest pain
2) ECG changes suggestive of ACS
3) Echocardiogram changes suggestive of ACS
4) ≥ 20% troponin level delta change from first to second troponin serum sample

- When ≥ 3 predictors are present the patient was diagnosed as having ACS.

ISHCHEMIC SYMPTOMS (5, 25, 87):

1) A discomfort or pain (in a variety of combinations) in chest, jaw, left or right shoulder epigastric, wrist or arm occurring with exertion or at rest, persisting for at least 20 minutes, with radiation to the arm, hand, jaw, shoulder or back. It could be associated with diaphoresis, dyspnea, nausea, vomiting, or lightheadedness.
2) The discomfort or pain is not exaggerated or decreased by pressure, movements and position of body.
3) Patient with ACS might not have typical symptoms. The atypical symptoms may comprise (but are not restricted to) of weakness, extreme fatigue, apprehension, mental confusion, anxiety, giddiness, syncope, nervousness and psychosis.

TYPICAL CHEST PAIN (87):

In 1978 **Heberden et.al** has given initial definition of typical chest pain that can be attributed to ischemia:

1) A painful sensation or discomfort in the chest accompanied by a strangling sensation, anxiety and occasional radiation of pain to the left arm.
2) Chest pain increasing with exertion and/ or emotional stress.
3) Chest pain relieving with rest and/or nitroglycerine.

   When all three criteria's were fulfilled chest pain was labeled as typical chest pain. Symptoms out of these definitions were labeled as atypical.

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I) ECG manifestations of acute Myocardial Ischemia (in Absence of LVH and LBBB):

1) New ST elevation at the J-point in 2 contiguous leads with the cutoff points: ≥0.2 mV in men or ≥0.15 mV in women in leads V_2 through $V_3$and/or ≥0.1 mV in other leads.
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Regional wall motion abnormality (RWMA):

1) It is defined as a localized decrease in the rate and amplitude of myocardial excursion with a blunted degree of myocardial thickening and associated local remodeling.
2) Detection of new RWMAs with preserved wall thickening and reflectivity will support for diagnosing ACS in association with other ACC/AHA criteria.

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NTTE is defined as

1) Troponin elevation in the existence of an additional definitive diagnosis which is known to be associated with rise in troponin levels, with lack of sufficient criteria for diagnosing acute coronary syndrome as per universal definition

given by ESC/ACC/AHA and serial samples showing <20 % rise or fall from first value i.e. constantly elevated at the same level.

2) NTTE will also be considered when normal epicardial coronaries are demonstrated on coronary angiography in patients with troponin elevation.

ANAEMIA (88, 89):

In critically ill patients significant anemia was defined as a hemoglobin concentration <9 gm/dl.

ACUTE KIDNEY INJURY (KDIGO):

It was defined by any of the following (**90**)**:**

1) Within 48 hours the serum creatinine increased by ≥0.3 mg/dl (≥26.5 µmol/L); or
2) Increase in serum creatinine by ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or
3) Urine output < 0.5 ml/ kg per hour for 6 hours

TROPONIN T ASSAY (27, 40, 41):

Roche Elecsys high sensitivity troponin T assay was used for the study. The limit of detection of the assay was 5 ng/L, 10% co-efficient of variation was 13 ng/L and 99% percentile value was 14 ng/lt.

1) It works on Sandwich principle.

2) Duration required for completion of assay is 18 minutes.
3) Sample is incubated twice.
4) During 1st incubation 50 µL of sample is taken which forms a sandwich complex with a ruthenium labeled and biotinylated monoclonal cTnT specific antibody respectively
5) During 2nd incubation streptavidin-coated micro particles are added to sandwich complex. With interaction of biotin and streptavidin it is then bound to the solid phase.
6) This reaction mixture is aspirated into the measuring cell where the micro particles are magnetically captured onto the surface of the electrode.
7) Chemiluminescent emission is induced after application of a voltage to the electrode which is measured by a photomultiplier.
8) Calibration curve was used to determine the result. Curve was instrument-specifically generated by 2-point calibration and a master curve (5-point calibration) provided via the reagent barcode.

STATISTICAL ANALYSIS:

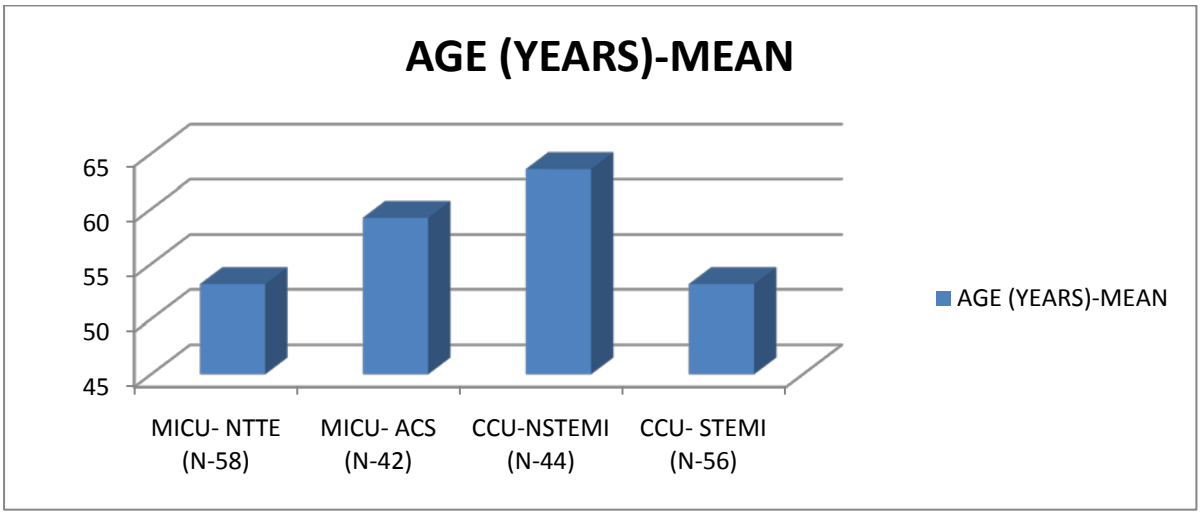
The data for all groups will be expressed as mean ± SD. Descriptive analysis was used to find out number and percentages. Categorical values were reported as proportion and percentages and it was compared using Chi-Square test. For continuous variables, mean with standard deviation was used and it was compared using independent sample t-test if normal and Mann-Whitney U test if not normal in Univariate analysis. The variables which were significant at Univariate analysis were taken for Multivariate analysis. Survival analysis was used to find association between in-hospital mortality and troponin levels in thrombotic ACS group and NTTE group. To find the cut-off levels of Apache score, ROC curve was used. The analysis was performed by using SPSS 16 version.

RESULTS:

TABLE 1: AGE OF PATIENTS AMOUNG MICU AND CCU GROUPS AND SUBGROUPS

GROUPS	SUBGROUPS	SUBGROUPS AGE (YEARS) MEAN ± SD	CCU AND MICU GROUPS AGE (YEARS) MEAN ± SD
MICU (N=100)	**NTTE (58)**	53.16 ± 18.321	55.7 ± 16.864
	MICU-ACS (42)	59.21 ± 14.079	
CCU (N=100)	**CCU- NSTEMI (N=44)**	63.59 ± 10.957	57.75 ± 11.673
	CCU- STEMI (N=56)	53.16 ± 10.133	

FIGURE 1: AGE OF PATIENTS AMOUNG MICU AND CCU SUBGROUPS



- **Table and bar diagram showing that age of patients (Mean and SD) in CCU and MICU groups included in study were comparable.**
- **CCU- NSTEMI cases were significantly older than STEMI cases (63.59 ± 10.957 vs. 53.16 ± 10.133, p value 0.000).**

TABLE 2: SEX DISTRIBUTION IN CCU GROUP- STEMI VS. NSTEMI

SEX DISTRIBUTION IN CCU GROUP- STEMI VS. NSTEMI				
CCU			TOTAL	P VALUE
SEX	STEMI	NSTEMI		
FEMALE	5(26.3%)	14(73.7%)	19	0.05
MALE	51 (63%)	30 (37%)	81	
TOTAL	56	44	100	

FIGURE 2: BAR DIAGRAM SHOWING SEX DISTRIBUTION IN CCU GROUP- STEMI VS. NSTEMI

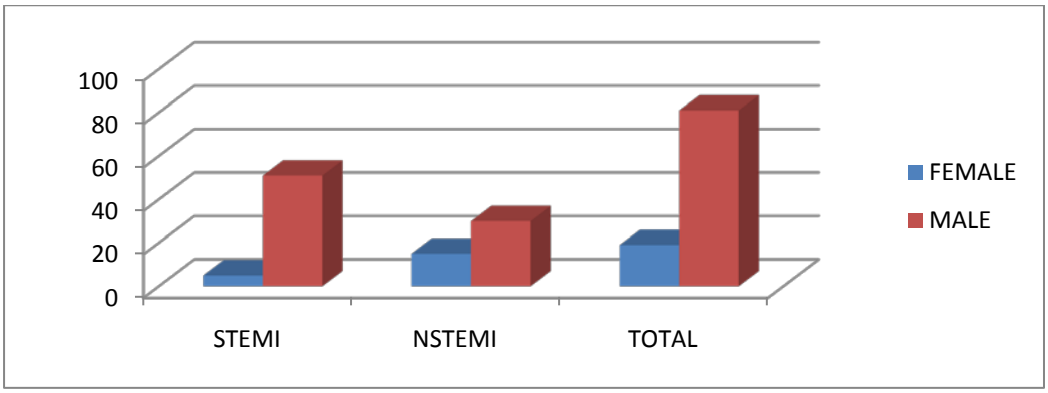


Table and bar diagram revealing that male sex was statistically significantly associated with ACS in CCU group (P value 0.05).

TABLE 3: MICU GROUP (THROMBOTIC AND NTTE) DEMOGRAPHICS

TABLE 3A: TABLE SHOWING SEX DISTRIBUTION IN NTTE AND THROMBOTIC ACS PATIENTS

SEX	THROMBOTIC - ACS	NTTE	TOTAL	P VALUE
MALE	23	28	51	0.522
FEMALE	19	30	49	
TOTAL	42	58	100	

FIGURE 3A: BAR DIAGRAM COMPARING SEX DISTRIBUTION IN NTTE AND THROMBOTIC ACS PATIENTS

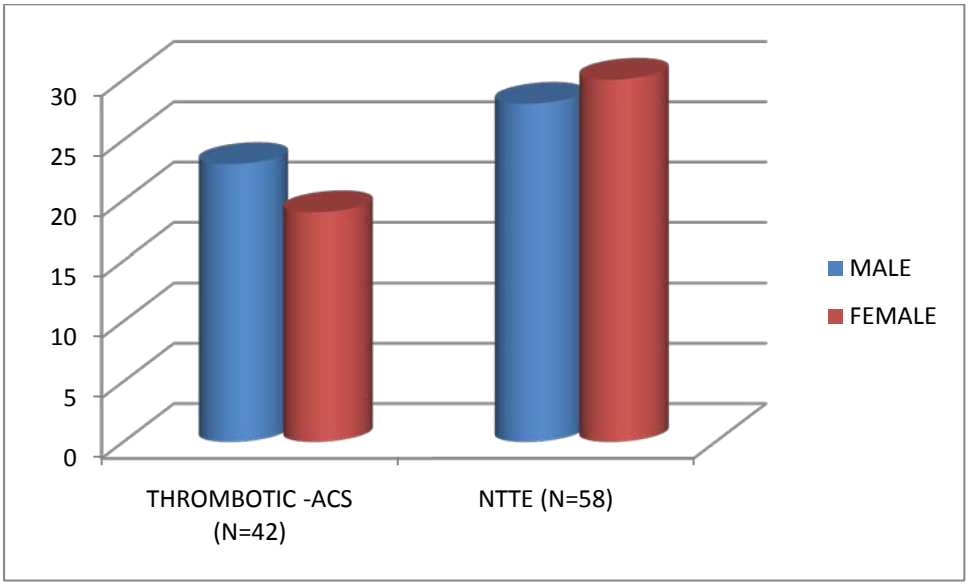


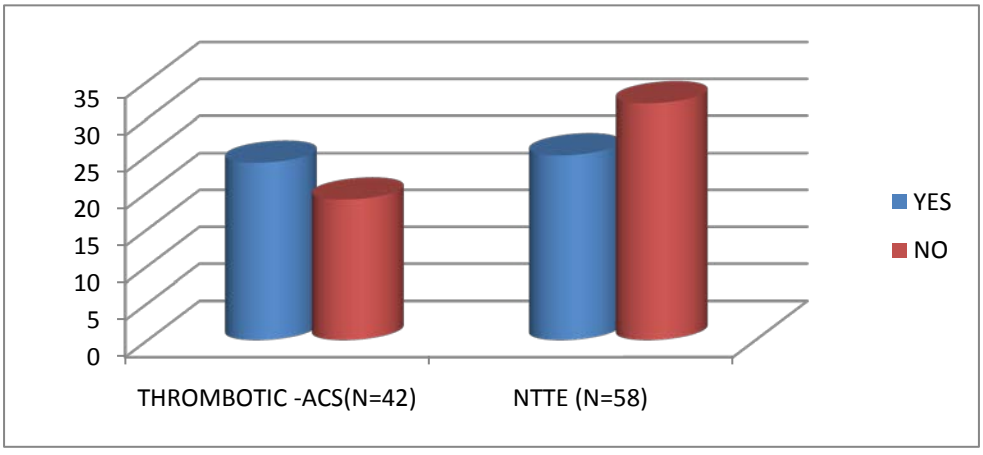
Table and bar diagram showing that sex was not significantly associated with troponin elevation in NTTE patients (LR-0.411, p value 0.522).

TABLE 4: MICU GROUP PATIENTS COMORBIDITIES:

TABLE 4A: TABLE SHOWING INCIDENCE OF HYPERTENSION IN NTTE AND THROMBOTIC ACS PATIENTS

HYPERTENSION	THROMBOTIC - ACS	NTTE	TOTAL	P Value	ODDS RATIO	95% CONFIDENCE INTERVAL	
						LOWER	UPPER
YES	24	25	49	0.191	0.586	0.262	1.310
NO	19	32	51				
TOTAL	43	57	100				

FIGURE 4A: BAR DIAGRAM COMPARING INCIDENCE OF HYPERTENSION IN NTTE AND THROMBOTIC ACS PATIENTS

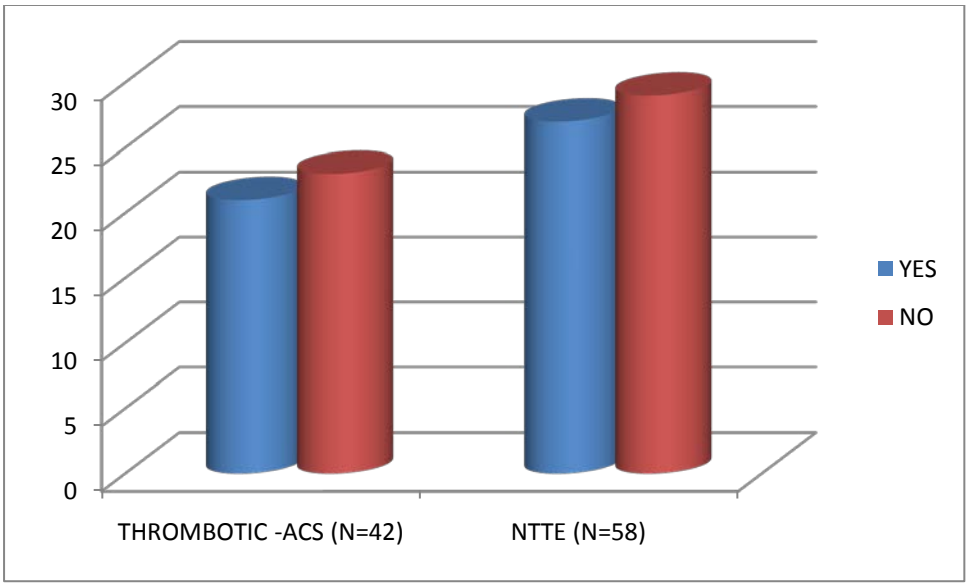


- **Table and bar diagram showing hypertension incidence among thrombotic ACS and NTTE patients.**
- **Hypertension was non-significantly associated with troponin elevation in NTTE patients (LR- 1.712; p value 0.191; Odds ratio-0.586; 95% CI- 0.262 -1.310%)**

TABLE 4B: TABLE COMPARING INCIDENCE OF DIABETES MELLITUS IN NTTE AND THROMBOTIC ACS PATIENTS AND ITS SIGNIFICANCE

DM II	THROMBOTIC -ACS	NTTE	TOTAL	P VALUE	ODDS RATIO	95% CONFIDENCE INTERVAL	
YES	21	27	48	0.416	0.812	LOWER	UPPER
NO	23	29	54			0.367	1.801
TOTAL	44	56	100				

FIGURE 4B: BAR DIAGRAM COMPARING INCIDENCE OF DIABETES MELLITUS IN NTTE AND THROMBOTIC ACS PATIENTS

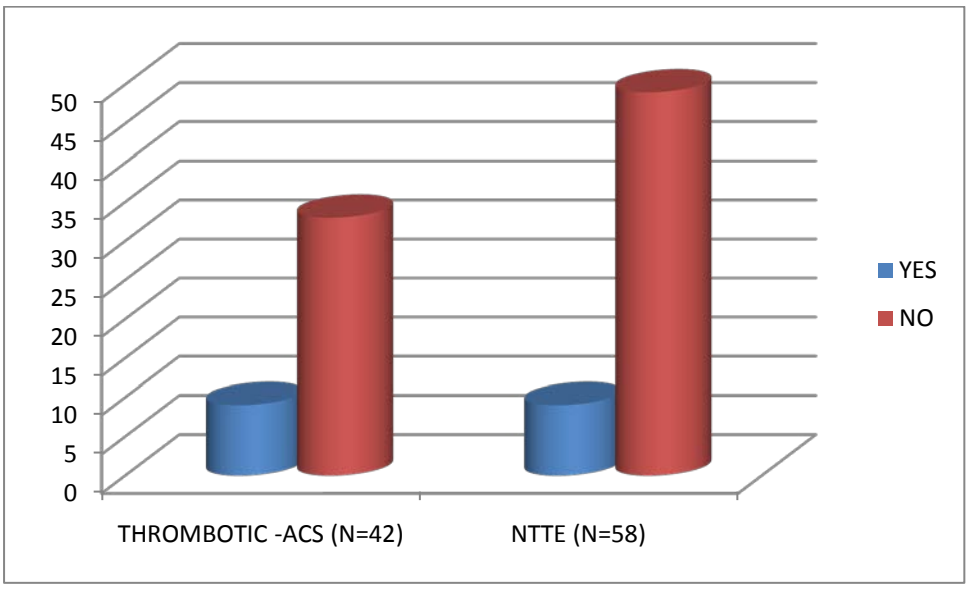


- **Table and bar diagram comparing incidence of diabetes mellitus type II among thrombotic ACS and NTTE patients.**
- **DM II was non-significantly associated with troponin elevation in NTTE patients (Likelihood Ratio-2.485; p value 0.416; Odds ratio-0.812; 95% confidence interval- 0.367 – 1.801%)**

TABLE 4C: TABLE SHOWING INCIDENCE OF PAST HISTORY OF CAD IN NTTE AND THROMBOTIC ACS PATIENTS

PAST H/O CAD/CVA	THROMBOTIC -ACS	NTTE	TOTAL	P VALUE	ODDS RATIO	95% CONFIDENCE INTERVAL	
YES	9	9	18	0.451	0.673	LOWER	UPPER
NO	33	49	82			0.242	1.875
TOTAL	42	58	100				

FIGURE 4C: BAR DIAGRAM COMPARING INCIDENCE OF PAST HISTORY OF CAD IN NTTE AND THROMBOTIC ACS PATIENTS

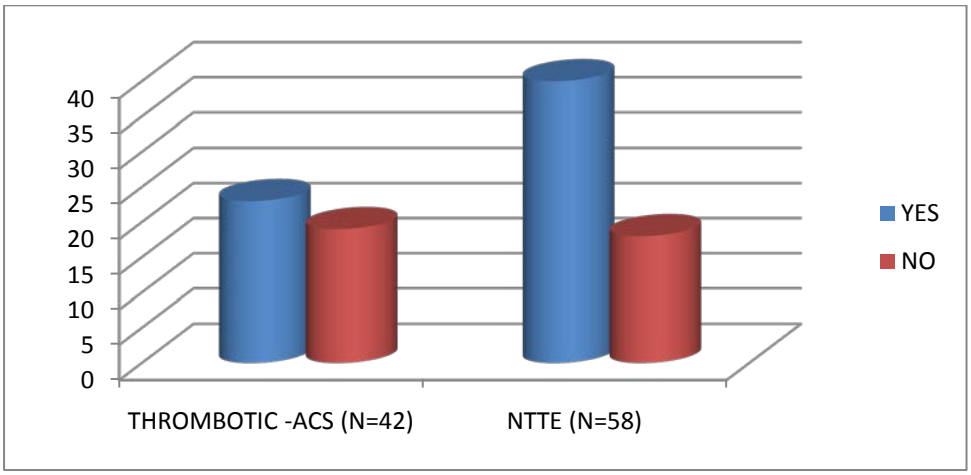


- **Table and bar diagram comparing incidence of past history of CAD among thrombotic ACS and NTTE patient.**
- **It was non-significantly associated with troponin elevation in NTTE patients (LR-4.382, p value 0.451; Odds ratio-0.673; 95% CI - 0.242 -1.875%)**

TABLE 4D: TABLE SHOWING INCIDENCE OF RENAL FAILURE IN NTTE AND THROMBOTIC ACS PATIENTS

RENAL FAILURE	THROMBOTIC -ACS	NTTE	TOTAL	P VALUE	ODDS RATIO	95% CONFIDENCE INTERVAL	
YES	23	40	63	0.147	1.836	LOWER	UPPER
NO	19	18	37			0.805	4.184
TOTAL	42	58	100				

FIGURE 4D: BAR DIAGRAM COMPARING INCIDENCE OF RENAL FAILURE IN NTTE AND THROMBOTIC ACS PATIENTS



- **Table and bar diagram comparing incidence of renal failure among thrombotic ACS and NTTE patients.**
- **It was significantly associated with troponin elevation in NTTE patients in comparison to ACS patients but the association was not statistically significant (LR-0.037; p value 0.147; Odds ratio-1.836; 95%CI - 0.805 -4.184%).**

TABLE 5: TABLE COMPARING INCIDENCE AND PERCENTAGES OF DIFFERENT COMORBIDITIES AMONG PATIENT OF CCU AND MICU GROUPS

COMORBIDITIES	**CCU (N=100)**	**MICU (N=100)**
CHEST PAIN	**88 (88%)**	**11* (11%)**
TYPICAL CHEST PAIN	**60 (68%)**	**5 (45%)**
DIABETES MELLITUS	**46 (46%)**	**48 (48%)**
HYPERTENSION	**50 (50%)**	**49 (49%)**
SMOKER	**43 (43%)**	**23 (23%)**
HYPOTHYROIDISM	**5 (5%)**	**3 (3%)**
DYSLIPIDEMIA	**81 (81%)**	**35 (35%)**
PAST HISTORY OF CAD/CVA	**21 (21%)**	**18 (18%)**
RENAL FAILURE	**15 (15%)**	**63 (63%)**
INTUBATED	**2 (2%)**	**73 (73%)**

- **73 Patients in MICU were intubated and symptoms cannot be assessed.**

TABLE 6: COMORBIDITIES ASSOCIATED WITH MICU GROUP (THROMBOTIC ACS AND NTTE) PATIENTS AND THEIR SIGNIFICANCE

COMORBIDITIES	**THROMBOTIC - ACS**	**NTTE**	**P VALUE**
HYPERTENSION	24	25	0.191
DIABETES MELLITUS	21	25	0.416
SMOKER	10	13	0.870
HYPOTHYROIDISM	2	1	0.379
DYSLIPIDEMIA	19	16	0.068
PAST HISTORY OF CAD	9	9	0.451
RENAL FAILURE	26	37	0.147
INTUBATED	30	43	0.450

FIGURE 5: BAR DIAGRAM COMPARING INCIDENCE OF DIFFERENT COMORBIDITIES IN NTTE AND THROMBOTIC ACS PATIENTS

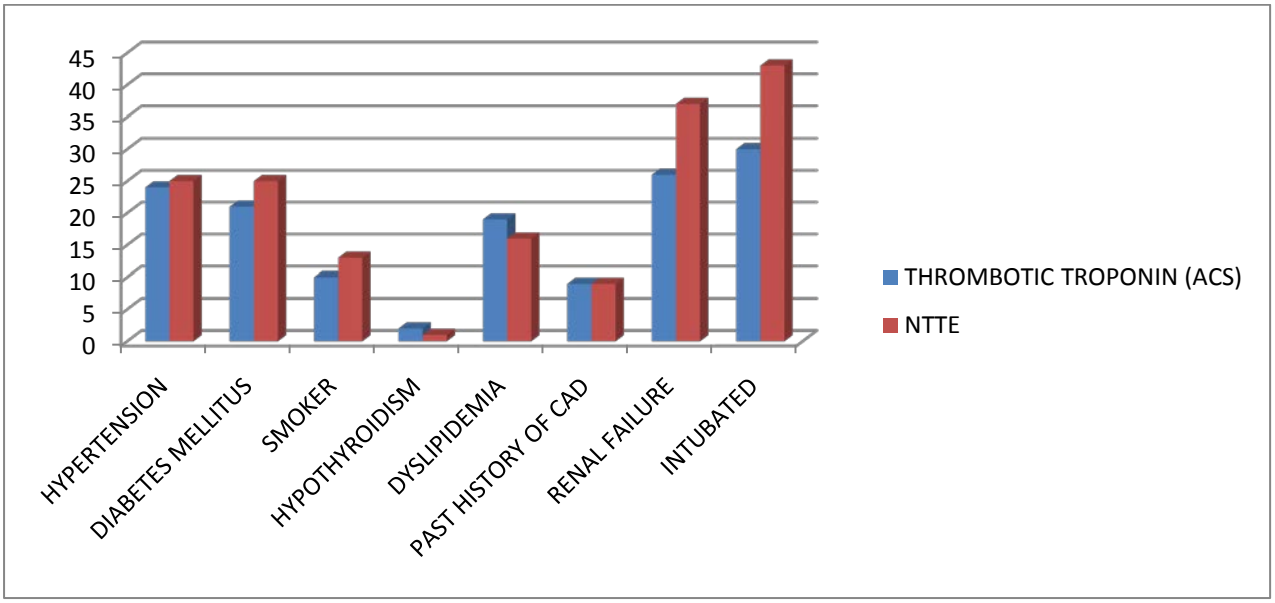


TABLE 7: DIAGNOSIS ASSOCIATED WITH ELEVATED TROPONIN IN CRITICALLY ILL MICU PATIENT SUBSET**:**

ETIOLOGICAL DIAGNOSIS	**INCIDENCE (N=100)**	**NTTE (N=58)**	**THROMBOTIC ACS (N=42)**
SEPSIS	70	45 (77.59%)	25(59.52%)
ARF/ACUTE ON CKD	63	40 (68.97%)	23 (54.76%)
ANAEMIA	43	25(43.10%)	18 (42.87%)
SHOCK	18	18(31.03%)	0
PNEUMONIA	15	15(25.86%)	0
ARDS	12	12(20.69%)	0
MYOCARDITIS	7	7(12.07%)	0
CHF	6	6(10.34%)	0
TACHYARRHYTHMIA	4	4(6.9%)	0
CPR	4	4(6.9%)	0
COPD	2	2(3.49%)	0
BRADYARRHYTHMIA	1	1(1.72%)	0
RHD	1	1(1.72%)	0
DIABETIC KETOACIDOSIS	1	1(1.72%)	0
AIDP	1	1(1.72%)	0
CVA	1	1(1.72%)	0
GTCS	1	1(1.72%)	0
SNAKE BITE	1	1(1.72%)	0

FIGURE 6A: BAR DIAGRAM SHOWING DIAGNOSIS ASSOCIATED WITH ELEVATED TROPONIN IN CRITICALLY ILL MICU PATIENT SUBSET

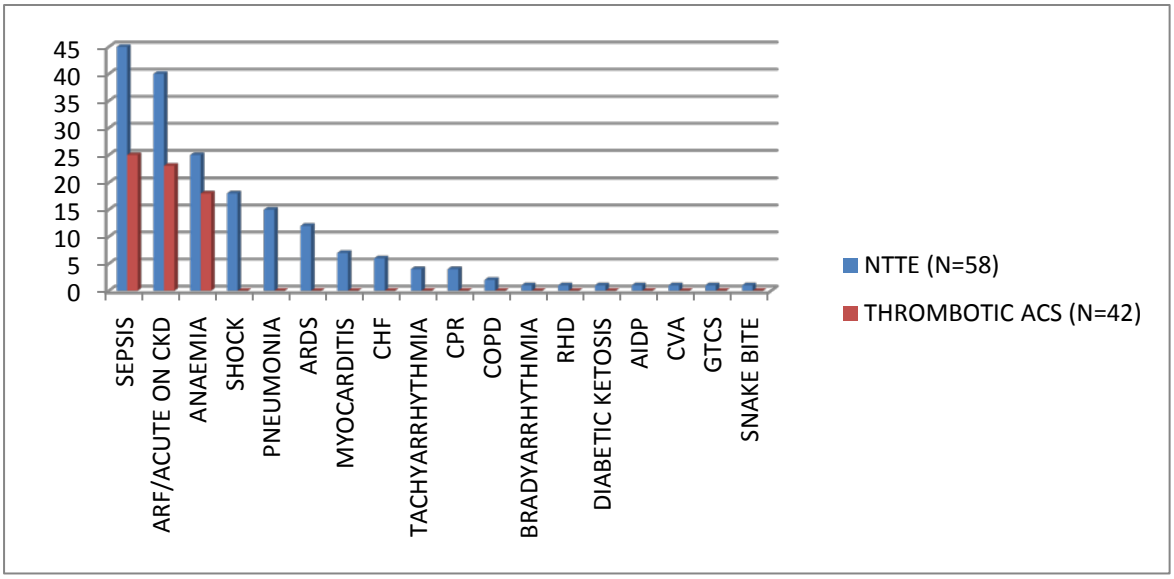


FIGURE 6B: PIE DIAGRAM SHOWING DIAGNOSIS ASSOCIATED WITH ELEVATED TROPONIN IN CRITICALLY ILL MICU PATIENT SUBSET

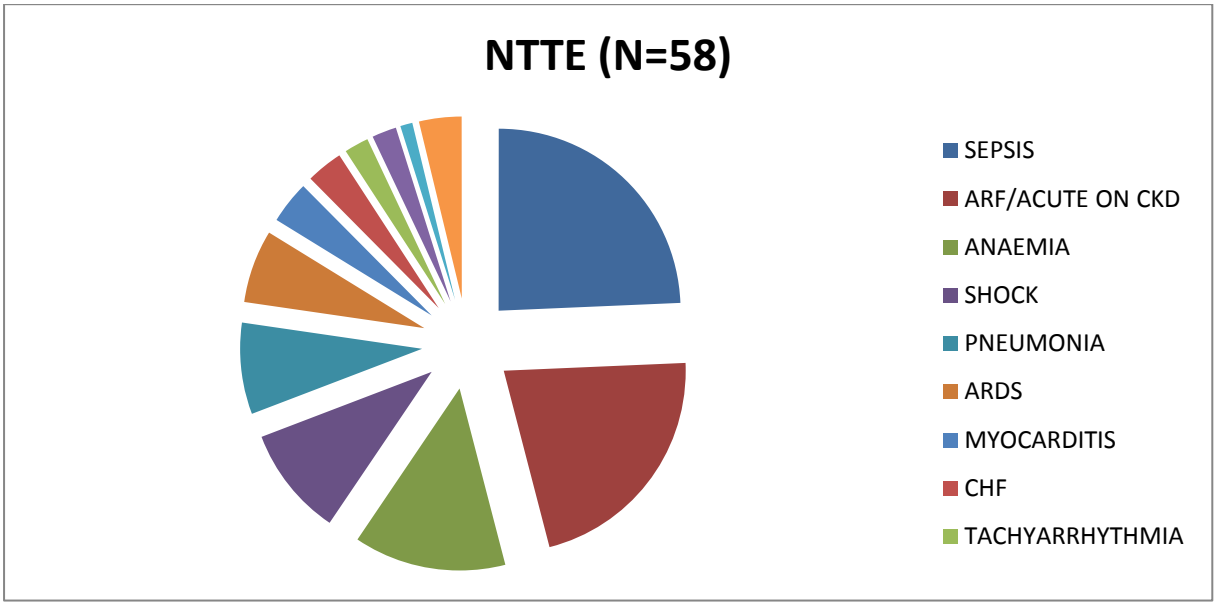


TABLE 8: COMPARISION OF CARDIAC BIOMARKERS AMONG FOUR SUBGROUPS

COMPARISION OF CARDIAC BIOMARKERS AMONG SUBGROUPS				
SUBGROUPS	CKMB1	CKMB2	TROP T1	TROP T2
CCU-STEMI (N=56)	77.16 ± 136.59	162.80 ± 157.87	1294.41 ± 2418.77	5162.20 ± 3635.84
CCU-NSTEMI (N= 44)	21.02 ± 45.88	34.57 ± 43.23	347.17 ± 566.90	983.47 ± 1922.34
MICU-ACS (N= 42)	9.88 ± 11.42	30.36 ± 64.63	373.04 ± 1124.51	743.30 ± 1303.90
NTTE (N=58)	8.19 ± 9.16	7.5102 ± 6.39	289.16 ± 1062.26	258.62 ± 891.41

FIGURE 7A: BAR DIAGRAM SHOWING COMPARISION OF TROPONIN T LEVELS AMONG ALL FOUR SUBGROUPS OF STUDY

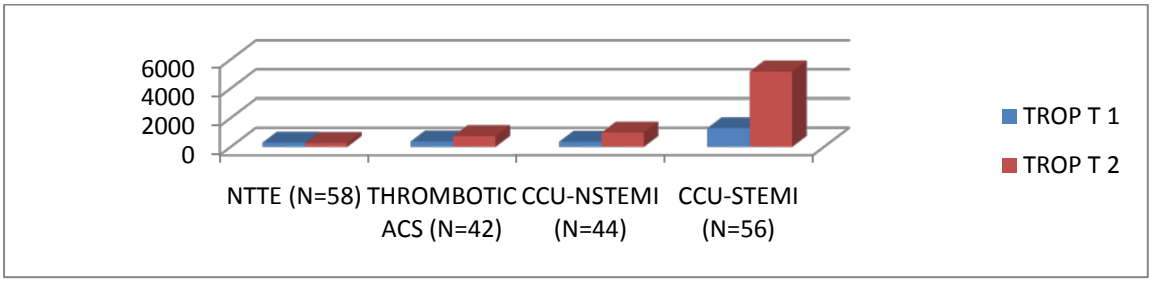
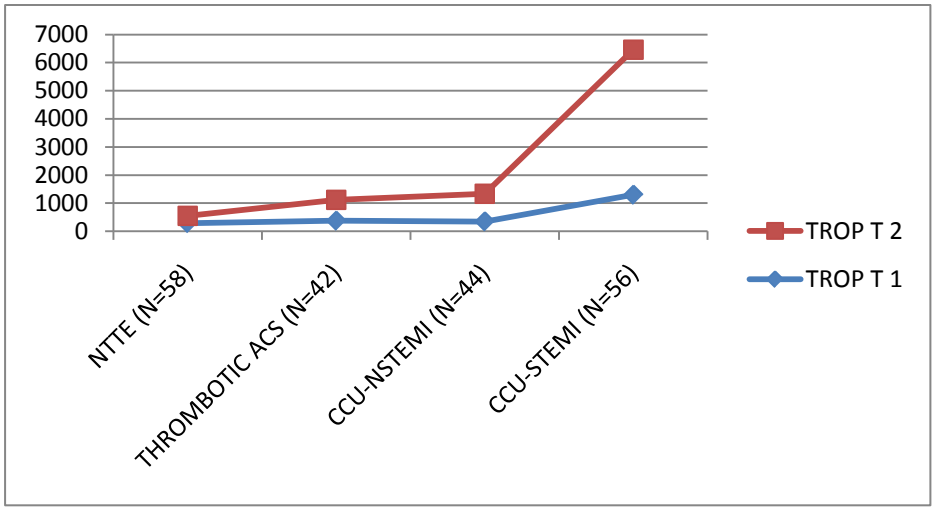


FIGURE 7B: LINE DIAGRAM SHOWING COMPARISION OF TROPONIN T LEVELS AMONG ALL FOUR SUBGROUPS OF STUDY



- **Table and bar diagram showing variation of cardiac biomarkers levels among all four subgroups included in the study.**
- **A range of troponin elevation is seen --- CCU-STEMI > CCU-NSTEMI > MICU-ACS > MICU-NTTE.**
- **First Sample- Baseline sample send within first 24 hours of patient's admission to CCU/ CPU/ MICU/ MHDU.**
- **Second sample- Blood sample send within 24 hours of first sample (6 to 8 hours on an average).**
- **Troponin level was measured in ng/L or pg/ml and CKMB level was measured in ng/ml.**

TABLE 9: COMPARISION OF CARDIAC BIOMARKERS LEVELS IN CCU-NSTEMI PATIENTS WITH MICU-ACS PATIENTS

COMPARISION OF CARDIAC BIOMARKERS LEVELS IN CCU-NSTEMI PATIENTS WITH MICU-ACS PATIENTS					**P value**		95% Confidence Interval of the Difference	
	group	N	Mean ± Std. Deviation	Std. Error Mean	Equal variances assumed	Equal variances not assumed	Lower	Upper
CKMB1	CCU-NSTEMI	44	21.02 ± 45.88	6.92	0.130	0.125	-3.31	25.64
	MICU-ACS	42	9.88 ± 11.42	1.76			-3.21	25.49
CKMB2	CCU-NSTEMI	44	34.57 ± 43.23	6.52	0.722	0.724	-19.26	27.69
	MICU-ACS	42	30.36 ± 64.63	9.97			-19.54	27.97
TROPT1	CCU-NSTEMI	44	347.17 ± 566.90	85.46	0.892	0.894	-405.17	353.42
	MICU-ACS	42	373.04 ± 1124.51	173.52			-412.78	361.04
TROPT2	CCU-NSTEMI	44	983.47 ± 1922.34	289.81	0.502	0.498	-467.56	947.87
	MICU-ACS	42	743.31 ± 1303.90	201.20			-462.51	942.83

FIGURE 8A: LINE DIAGRAM COMPARING CARDIAC BIOMARKERS LEVELS IN CCU-NSTEMI PATIENTS WITH MICU-ACS PATIENTS

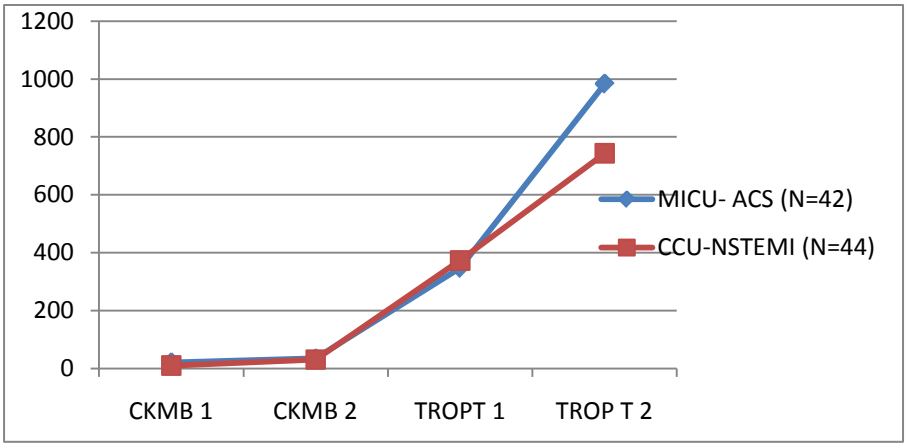
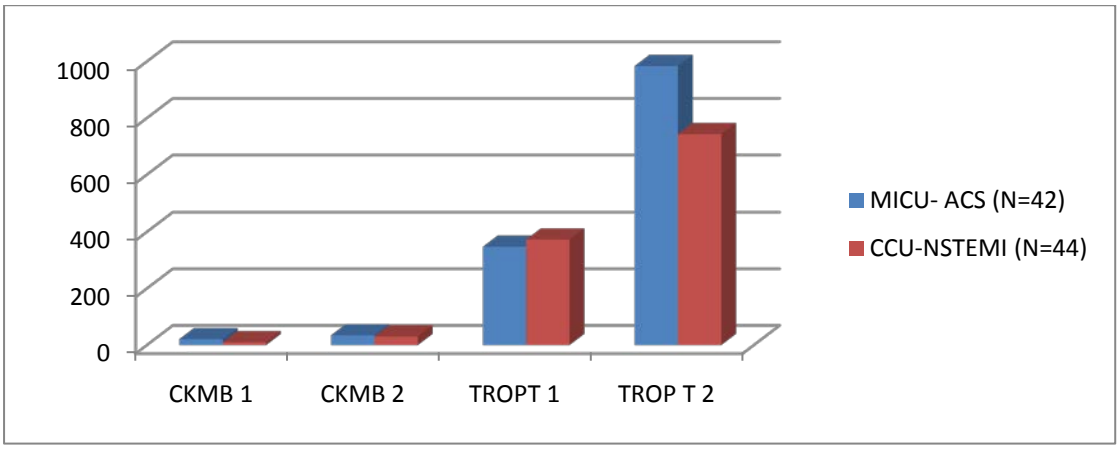


FIGURE 8B: BAR DIAGRAM COMPARING CARDIAC BIOMARKERS LEVELS IN CCU-NSTEMI PATIENTS WITH MICU-ACS PATIENTS



- **Serum troponin levels in NSTEMI patient in CCU (Trop T 1st and 2nd sample was 347.17 ± 566.90 and 983.47 ± 1922.34 respectively) and ACS patients in MICU (Trop T 1st and 2nd sample was 373.04 ± 1124.51 and 743.31 ± 1303.90 respectively) were comparable.**
- **'p value' for Troponin T 1st and 2nd sample is 0.892 and 0.502 respectively**.

TABLE 10: COMPARISION OF CARDIAC BIOMARKERS LEVELS IN MICU-NTTE PATIENTS WITH MICU-ACS PATIENTS

COMPARISION OF MICU-NTTE PATIENTS WITH MICU-ACS PATIENTS					
GROUP		CKMB1	CKMB2	TROPT1	TROPT2
MICU-ACS (N=42)	Mean ±Std. Deviation	9.88 ± 11.42	30.36 ± 64.63	373.04 ± 1124.51	743.31 ± 1303.90
NTTE (N=58)	Mean ± Std. Deviation	8.19 ± 9.16	7.51 ± 6.39	289.16 ± 1062.26	258.62 ± 891.41
P VALUE		0.675	0.000	0.058	0.000

FIGURE 9A: BAR DIAGRAM COMPARING CARDIAC BIOMARKERS LEVELS IN MICU-NTTE PATIENTS WITH MICU-ACS PATIENTS

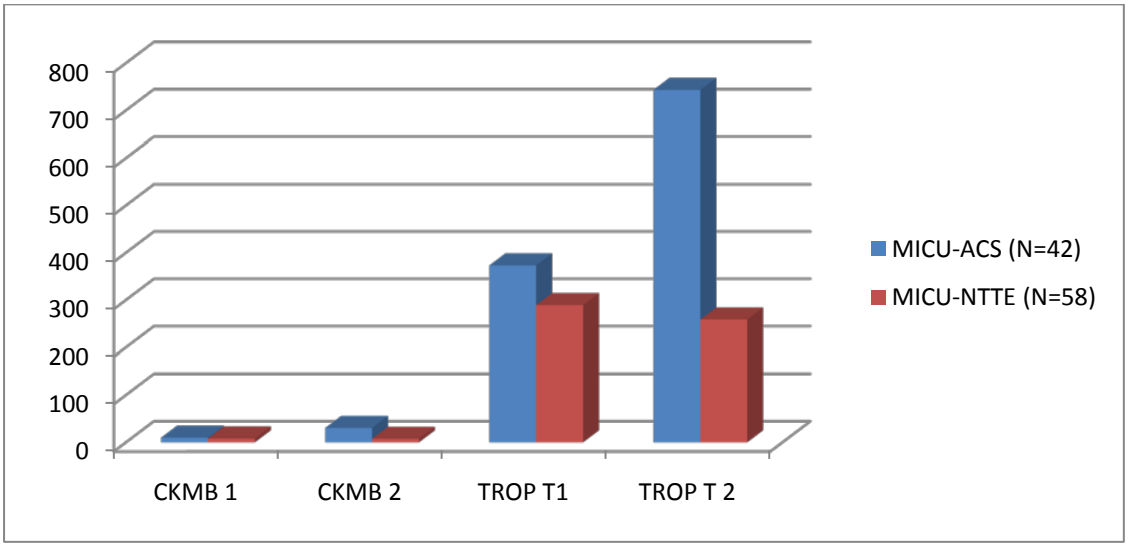
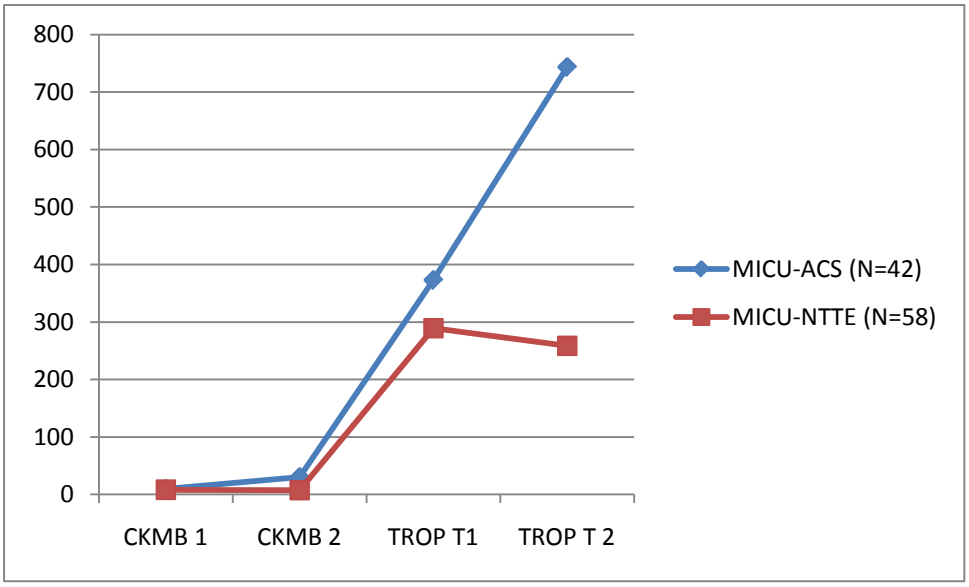


FIGURE 9B: LINE DIAGRAM COMPARING CARDIAC BIOMARKERS LEVELS IN MICU-NTTE PATIENTS WITH MICU-ACS PATIENTS



- **Troponin T level in ACS patients (Trop T 1st and 2nd sample was 373.04 ± 1124.51 and 743.31 ± 1303.90 respectively) was significantly higher than troponin T level in NTTE patients (Trop T 1st and 2nd sample was 289.16 ± 1062.26 and 258.62 ± 891.41 respectively)**
- **'p value' for Troponin T 1st and 2nd sample is 0.05 and 0.000 respectively for the association.**

TABLE 11: CO-RELATION OF TROPONIN T 1^{ST} AND 2^{ND} SAMPLE LEVEL WITH DURATION OF HOSPITALISATION IN MICU PATIENTS

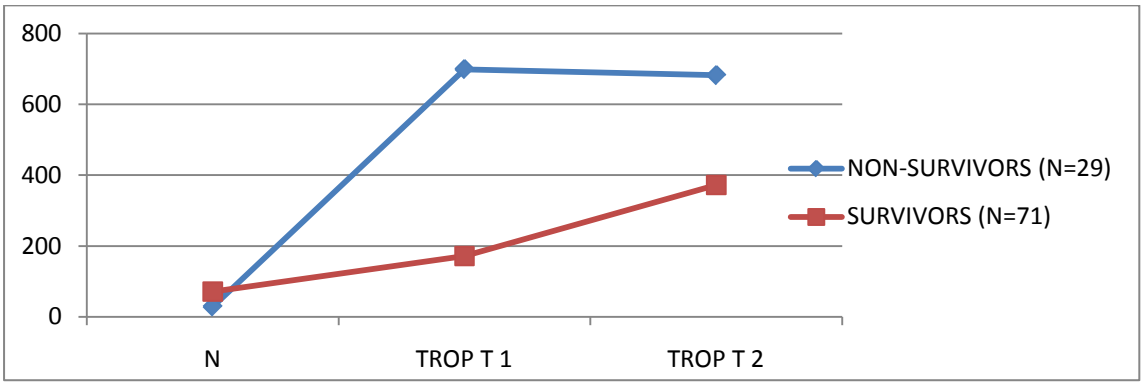
DURATION OF HOSPITALISATION	**N**	**PEARSON CORRELATION**	**SIGNIFICANCE**
TROP T 1	100	-0.068	0.501
TROP T 2	100	-0.072	0.479

- **Table presenting co-relation and significance of Troponin T 1^{st} and 2^{nd} sample levels with duration of hospitalization.**
- **Troponin T 1^{st} and 2^{nd} sample level were negatively co-related with duration of hospitalization, Pearson co-relation coefficient for Trop T 1^{st} and 2^{nd} sample was -0.068 and -0.072 respectively.**
- **It was not a significant co-relation (p value for Trop T 1^{st} and 2^{nd} sample 0.501 and 0.479 respectively).**

TABLE 12: ASSOCIATION OF TROPONIN T BASELINE AND SECOND SAMPLE LEVELS WITH IN-HOSPITAL MORTALITY IN MICU PATIENTS

	In-hospital mortality	**N**	**Mean ± Std. Deviation**	**Std. Error Mean**	**P value**
TROPT1	Mortality	29	699.03 ± 1953.03	362.67	0.026
	Alive	71	171.36 ± 233.05	27.67	
TROPT2	Mortality	29	681.96 ± 1782.04	330.92	0.205
	Alive	71	372.42 ± 652.81	77.47	

FIGURE 10: LINE DIAGRAM SHOWING ASSOCIATION OF TROPONIN T 1st AND 2nd SAMPLE LEVEL WITH IN-HOSPITAL MORTALITY IN MICU PATIENTS



- **Baseline troponin levels were significantly associated with in-hospital mortality (p value 0.026) while 2nd sample troponin levels were not significantly associated with in-hospital mortality (p value 0.205).**

TABLE 13: CO-RELATION OF TROPONIN BASELINE AND SECOND SAMPLE LEVELS WITH OUTCOME (MORTALITY, RE-HOSPITALISATION) ≤ 30 DAYS AFTER DISCHARGE IN MICU PATIENTS

		N	Mean ± Std. Deviation	Std. Error	95% Confidence Interval for Mean		P VALUE
					Lower Bound	Upper Bound	
TROPT 1^{ST} SAMPLE	MORTALITY	9	148.82 ± 246.54	82.18	40.69	338.32	0.157
	REHOSPITALISATION	5	161.60 ± 290.18	129.77	198.71	521.90	
	NONE	57	169.51 ± 228.38	30.25	108.92	230.11	
TROP T 2^{ND} SAMPLE	MORTALITY	9	438.40 ± 964.39	321.46	302.90	1179.69	0.564
	REHOSPITALISATION	5	175.50 ± 324.68	145.20	227.65	578.65	
	NONE	57	370.43 ± 624.05	82.66	204.85	536.02	

Table showing troponin T baseline and second sample levels were not significantly associated with mortality or re-hospitalization ≤ 30 days after discharge (p value = 0.157 and 0.564).

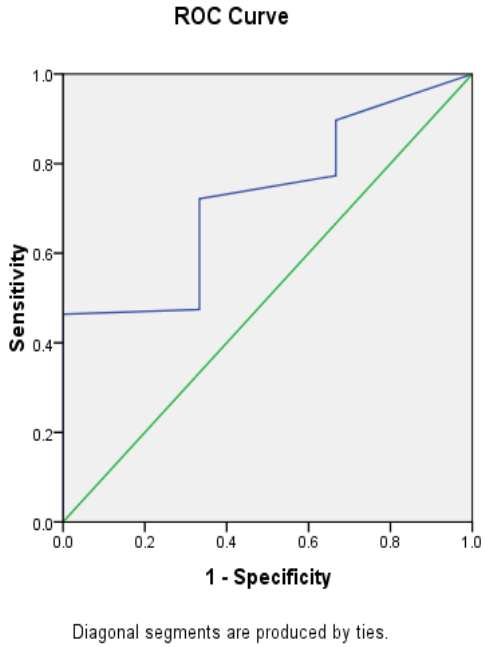
TABLE 14: CO-RELATION OF SERUM TROPONIN LEVELS WITH OUTCOME (IN-HOSPITAL MORTALITY, MORTALITY ≤ 30 DAYS AFTER DISCHARGE, RE-HOSPITALISATION) IN CCU PATIENTS

SAMPLE	**OUTCOME**	**N**	**MEAN ± STD. DEVIATION**
TROP T 1ST (BASELINE)SAMPLE	NO EVENTS	94	868.34 ± 1943.938
	INHOSPITAL MORTALITY	1	2580.00
	MORTALITY ≤ 30 DAYS AFTER DISCHARGE	3	1130.50 ± 975.337
	REHOSPITALISATION	2	83.34 ± 57.368
	TOTAL	100	877.62 ± 1900.831
TROP T 2ND SAMPLE	NO EVENTS	94	3464.42 ± 3713.211
	INHOSPITAL MORTALITY	1	1776.00
	MORTALITY ≤ 30 DAYS AFTER DISCHARGE	3	1137.00 ± 887.610
	REHOSPITALISATION	2	756.55 ± 767.140
	TOTAL	100	3323.56 ± 3646.258

TABLE 15: CO-RELATION OF APACHE II SCORE WITH NTTE

CO-ORDINATES OF THE CURVE		
APACHE II SCORE	**SENSITIVITY**	**SPECIFICITY**
13.00	89.7%	33.3%
14.50	88.7%	33.3%
15.50	85.6%	33.3%
16.50	81.4%	33.3%
17.50	77.3%	33.3%
18.50	**72.2%**	**67.33%**
19.50	70.1%	67.7%
20.50	67.0%	67.7%
21.50	61.9%	67.7%
22.50	59.8%	67.7%
23.50	50.5%	67.7%
24.50	47.4%	67.7%
25.50	46.4%	100%

FIGURE 11: ROC CURVE



Area Under the Curve				
Test Result Variable: **APACHE II SCORE**				
Area	Std. Error	P value	95% Confidence Interval	
			Lower Bound	Upper Bound
0.722	0.119	0.192	0.488	0.955

- **APACHE II score ≥18 in critically ill MICU patients was associated with NTTE with 72% sensitivity and 67.33% specificity. While APACHE II score ≥ 25 was associated with NTTE with 100% specificity and sensitivity decreased to 46.4%.**
- **Co-relation between APACHE II score and NTTE was not statistically significant (Area under curve- 0.722; p value-0.192; 95% CI- 0.488-0.955%).**

TABLE 16A: COMPARISON OF FEMALE SEX WITH IN-HOSPITAL MORTALITY AMONG NTTE AND THROMBOTIC ACS PATIENTS

FEMALE	THROMBOTIC ACS	NTTE	TOTAL	P VALUE
MORTALITY	5 (45.5%)	6 (54.5%)	11	0.751
ALIVE	14 (36.8%)	24(63.2%)	38	
TOTAL	19	30	49	

TABLE 16B: COMPARISON OF FEMALE SEX WITH IN-HOSPITAL MORTALITY AMONG NTTE AND THROMBOTIC ACS PATIENTS

MALE	THROMBOTIC ACS	NTTE	TOTAL	P VALUE
MORTALITY	6 (33.3%)	12 (67.3%)	18	0.251
ALIVE	17 (51.5%)	16(48.5%)	33	
TOTAL	23	28	51	

- **I t is revealed in table that the co-relation between sex and in-hospital mortality in NTTE and thrombotic ACS patients in MICU group was not statistically significant.**

TABLE 17: CO-RELATION OF IN-HOSPITAL MORTALITY IN THROMBOTIC ACS AND NTTE SUBGROUPS.

GROUPS	THROMBOTIC ACS	NTTE	TOTAL	P VALUE
IN- HOSPITAL MORTALITY	11 (37.9%)	18 (62.1%)	29	0.669
ALIVE	31 (43.7%)	40 (56.3%)	71	
TOTAL	42	58	100	

FIGURE 12:

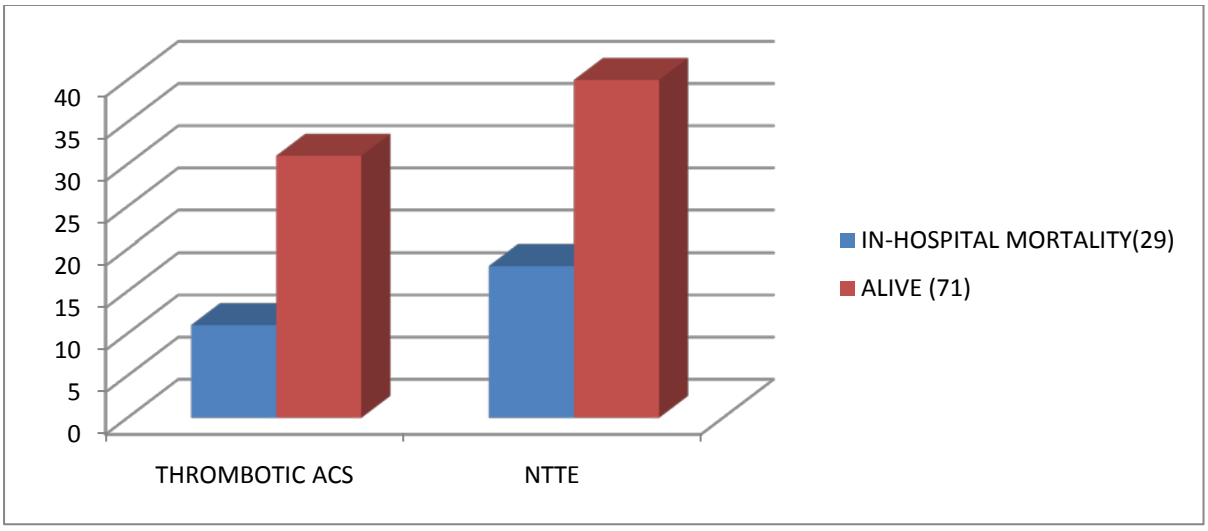
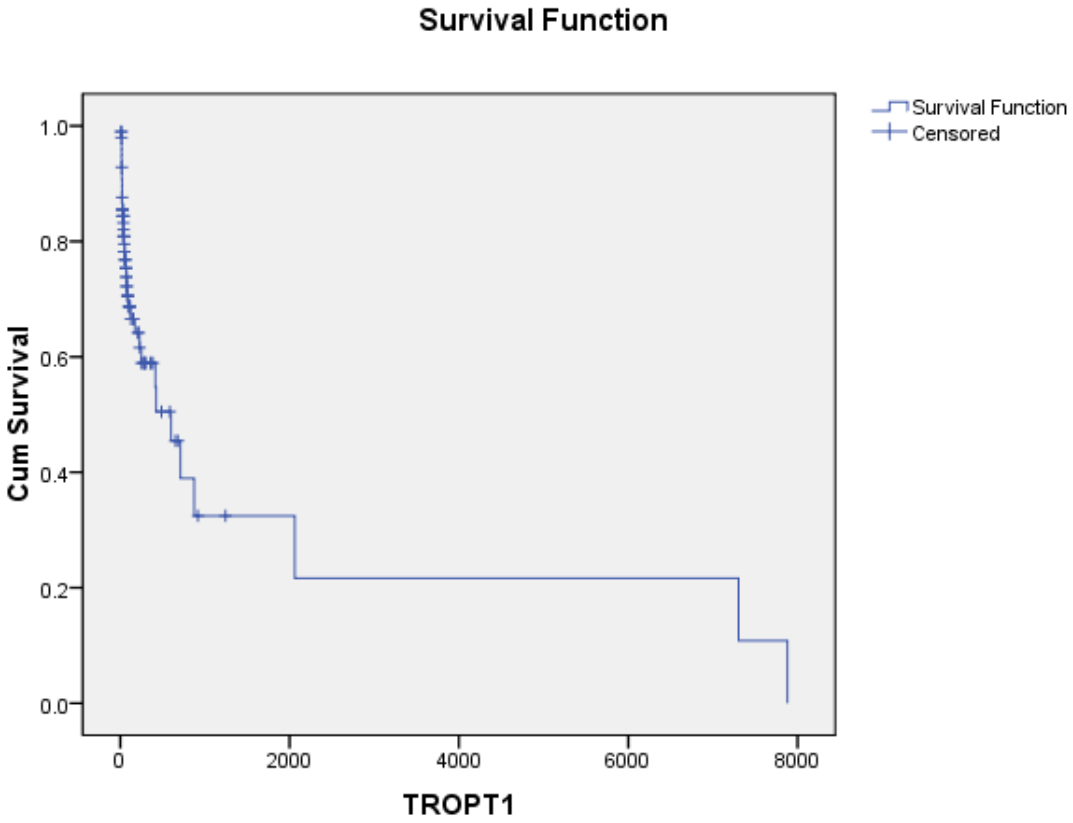


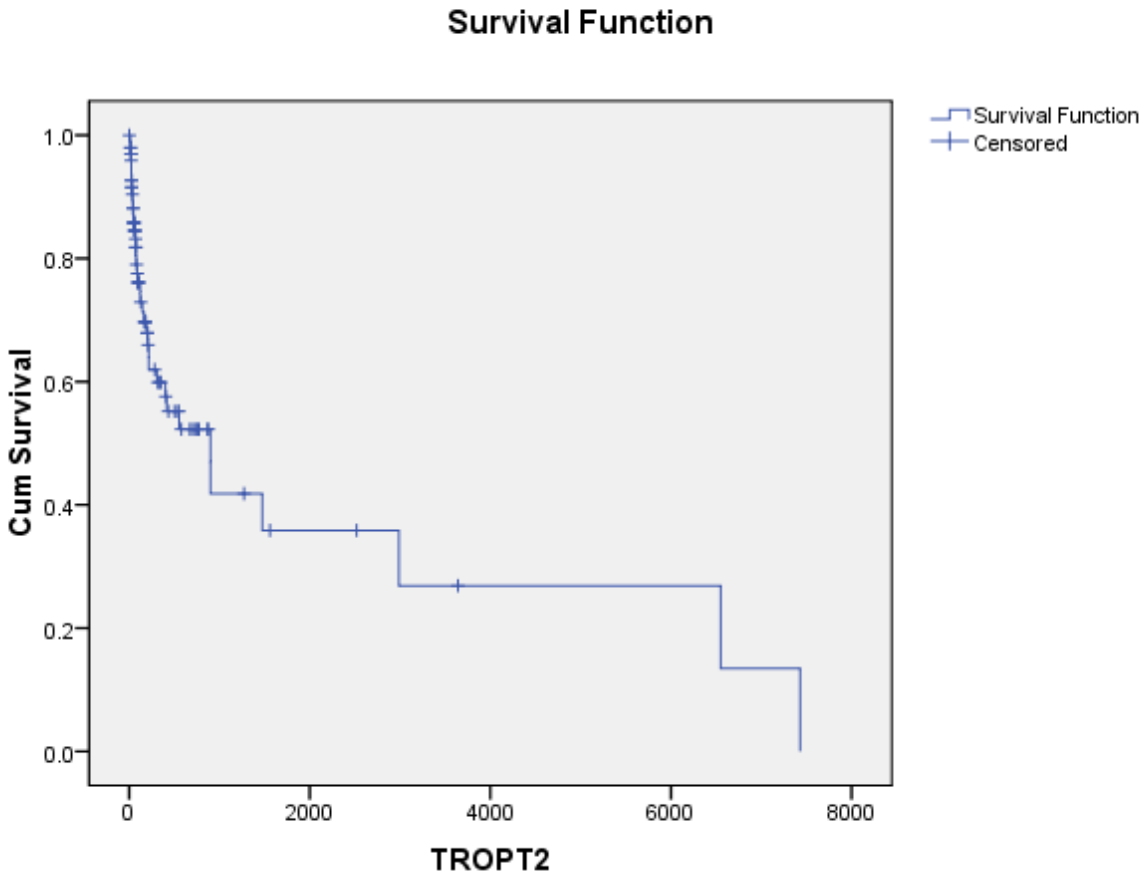
Table and bar diagram showing no statistically significant co-relation between in-hospital mortality in thrombotic ACS patients and NTTE patients

FIGURE 13A: KAPLAN - MEIER ANALYSIS CURVE FOR TROPONIN T BASELINE SAMPLE:



- **Kaplan-Meier analysis curves for troponin T 1st sample: suggesting that higher the serum troponin level higher is the mortality.**

FIGURE 13B: KAPLAN- MEIER ANALYSIS CURVE FOR TROPONIN T 2ND SAMPLE:



- **Kaplan-Meier analysis curves for troponin T 2nd sample: suggesting that higher the serum troponin level higher is the mortality.**

TABLE 18A: TABLE SHOWING INCIDENCE OF ≥ 3 PREDICTORS OF ACS IN MICU GROUP **(chest pain, abnormal ECG, abnormal ECHO suggestive of ACS and troponin level change ≥20% from baseline sample)**

	Thrombotic ACS	**NTTE**	**Total**
≥ 3 Predictors of ACS are present	42	30	72
< 3 Predictors of ACS are present	0	28	28
Total	42	58	100

TABLE 18B: PROBABILITY OF ACS IF ≥ 3 PREDICTORS OF ACS ARE PRESENT.

	Percentages and ratios	**95% Confidence interval**	
		Lower limit	**Upper limit**
Sensitivity	100%	91.51%	100%
Specificity	48.28%	34.95%	61.78%
Positive Likelihood Ratio	1.93	1.51	2.48
Disease Prevalence	42%	32.20%	52.29%
Positive predictive value	58.33%	46.11%	69.85%
Negative predictive value	100	87.54%	100%

- **When ≥ 3 predictors are present the patient was diagnosed as having ACS.**
- **For diagnosing ACS this gives us a sensitivity of 100%, specificity of 48.25%, positive predictive value of 58.33%, negative predictive value of 100% and positive likelihood ratio of 1.93.**

TABLE 19A: TABLE SHOWING INCIDENCE OF ≥ 3 PREDICTORS OF ACS IN MICU GROUP (chest pain, abnormal ECG, ECHO suggestive of ACS and at least one troponin value > 300 pg/ml or ng/lt.)

	Thrombotic ACS	**NTTE**	**Total**
≥ 3 Predictors of ACS are present	42	13	55
< 3 Predictors of ACS are present	0	45	45
Total	42	58	100

TABLE 19B: PROBABILITY OF ACS IF ≥ 3 PREDICTORS OF ACS ARE PRESENT.

	Percentages and ratios	**95% Confidence interval**	
		Lower limit	**Upper limit**
Sensitivity	100%	91.51%	100%
Specificity	77.59%	64.72%	87.48%
Positive Likelihood Ratio	4.46	2.76	7.2
Disease Prevalence	42%	32.20%	52.29%
Positive predictive value	76.36%	62.98%	86.76%
Negative predictive value	100	92.05%	100%

- **At least one serum troponin level > 300 pg/ml or ng/L was used instead of ≥ 20% troponin change from baseline sample as predictors of ACS in the study.**
- **For diagnosing ACS there was similar sensitivity (100%) but increase in specificity, positive predictive value and positive likelihood ratio (77.59% vs. 48.25, 76.36% vs. 58.33 and 4.46 vs. 1.93 respectively).**

TABLE 20A: DIVISION OF NTTE AND THROMBOTIC ACS PATIENTS INTO GROUPS AS PER APACHE II SCORE

APACHE II SCORE	THROMBOTIC ACS	NTTE	TOTAL	P VALUE
≥ 12 TO 17	5 (21.7%)	18 (78.3%)	23	.074
≥ 18 TO 24	14 (45.2%)	17 (54.8%)	31	
≥ 25	23 (50%)	23 (50%)	46	

FIGURE 14: PIE DAIAGRAM SHOWING DISTRIBUTION OF PATIENTS INTO GROUPS AS PER APACHE II SCORE.

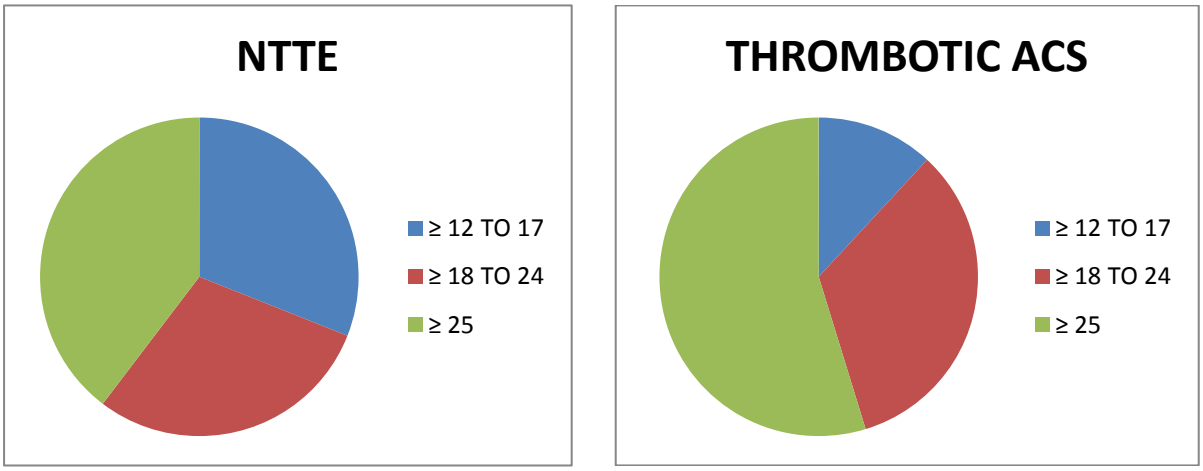


TABLE 20B: COMPARISON OF THROMBOTIC ACS AND NTTE GROUP AS PER APACHE II SCORE

APACHE II SCORE	NTTE	THROMBOTIC ACS	TOTAL	P VALUE
≥ 25	23 (56.1%)	23(82.14%)	46	0.022
≥ 12 TO 17	18 (43.9%)	5 (17.86%)	23	
TOTAL	43	28	69	

TABLE 20C: PROBABILITY OF NTTE WHEN APACHE II SCORE IS ≥ 25

	Percentages and ratios	**95% Confidence Interval**	
		Lower	**Upper**
Sensitivity	56.10%	39.75%	71.52%
Specificity	17.86%	6.13%	36.91%
Positive Likelihood ratio	0.68	0.50	0.94
Negative Likelihood ratio	2.46	1.03	5.85
Disease Prevalence	59.52%	46.92%	71.08%
Positive Predictive Value	50%	34.91%	65.09%
Negative Predictive Value	21.74%	7.54%	43.71%

- **Critically ill patients with elevated troponin levels having APACHE II score ≥ 25 were compared to (≥ 12 to 17).**
- **Our result showed that critically ill patients with elevated troponin level, having APACHE II score with in 24 hour of admission to ICU of ≥ 25 there is 50% probability that patient is having NTTE.**

TABLE 20D: PROBABILITY OF NTTE WHEN APACHE II SCORE IS ≥ 12 TO 17

	Percentages and ratios	**95% Confidence Interval**	
		Lower	**Upper**
Sensitivity	43.90%	28.48%	60.25%
Specificity	82.14%	63.09%	93.87%
Positive Likelihood ratio	2.46	1.03	5.85
Negative Likelihood ratio	0.68	0.5	0.94
Positive Predictive Value	78.26%	56.29%	92.46%
Negative Predictive Value	50%	34.91%	65.09%

- **Our result showed that critically ill patients with elevated troponin level, having APACHE II score with in 24 hour of admission to ICU of (≥ 12 to 17) there is 80% probability that patient is having NTTE.**
- **In critically ill ICU patients with elevated troponin levels as severity of illness increases {as documented by increase in APACHE II score from (≥ 12 to 17) to ≥ 25)} the probability of NTTE decreases (80% vs. 50%).**

DISCUSSION:

Elevated serum troponin levels are common in critically ill non-cardiac patients (CINCP) admitted in MICU. Non-invasive evaluation in such patients is difficult to perform and gives limited information. Even after performing coronary angiogram in such patients, the decision to label patients as having ACS is difficult if there is no clear cut evidence of visible thrombus, slow flow or no flow phenomenon visualized on angiogram. These patients may also have coronary artery lesion which are stable and longstanding and not contributing to the present condition. It has been documented that NTTE is associated with CINCP [**S.Agewall et.al, R. Alcali et. al, Fleming et. al, Jeremias A et.al**, **Juan Sanchis et.al, Wallace TW et. al** – (**10, 20, 25, 45, 74, and 91** respectively**)].**

The present study was aimed to determine the significance of NTTE in relation to in- hospital and 30 days mortality, duration of hospitalization and its association with non cardiac conditions which are commonly associated with the NTTE in CINCP. 200 cases were included in the study, 100 patients of ACS from CCU/CPU (56-STEMI and 44-NSTEMI) and 100 critically ill patients from MICU/MHDU (42-ACS and 58-NTTE).

One patient (2%) had in-hospital mortality in the CCU-NSTEMI group. In MICU group 29 (29%) patients had in-hospital mortality, 11(26.2%) in Thrombotic ACS group and 18 (31%) in NTTE group. Both thrombotic ACS and NTTE group had higher in-hospital mortality than CCU-NSTEMI patients and this was statistically significant (p value = 0.0038 and < 0.001 respectively. NTTE and ACS in critically ill

patients had mortality that is to a greater extent higher than that associated with STEMI/ NSTEMI patients admitted to CCU/ CPU with no acute medical co-morbidities (**20 to 24).** Both NTTE and thrombotic ACS group had increased and similar in-hospital mortality despite higher troponin level in thrombotic ACS group (p value=0.669). In contrast a similar study done by R Alcalai et.al showed that NTTE patients had lower serum troponin level but higher mortality than thrombotic ACS patients (**24**).

In patients who died during index hospitalization baseline troponin T level was significantly higher than patients who were discharged alive (699.03 ± 1953.03 vs. 171.36 ± 233.05 respectively). Baseline troponin level was significantly associated with in-hospital mortality (p value 0.026). In patients who died during index hospitalization second sample of troponin T was not statistically significantly higher than patients who were alive and discharged (681.96 ± 1782.04 vs. 372.42 ± 652.811 respectively; p value 0.205).

In accordance with our study **Ammann et.al** showed that elevated troponin level is associated with increased in-hospital mortality (**19)**. **King DA et.al** also showed similar prognostic role of elevated troponin in critically ill patients (**21)**. Similarly in a study by **Wu TT et.al** elevated serum troponin levels was a risk factor for multiple organ dysfunction (MODS) and mortality in non-cardiac critically ill patients (**23)**. Similar results were found by **Wendy Lim et.al, Guest TM et.al**, **Christian W.Hamm et.al, Mehta S et.al, Di AE et.al** (**19, 20, 46, 73** respectively). A sub-study of TACTIC-TIMI 18 had shown that 4% of patients who were diagnosed to have ACS, on angiogram had normal or no significant coronary artery disease

suggesting false positive troponin test. These patients had significantly higher major adverse cardiovascular events than patients without troponin elevation (**92**). **Keller et.al** stated that higher in-hospital mortality in CINCP is due to fact that these patients are sicker and troponin is marker or indicator of a critical state of non-cardiac disease condition (**93**).

Kaplan-Meier analysis curves for troponin 1st and 2nd sample suggested that mortality showed positive correlation with increasing troponin levels. In accordance with the result of our study and previous literature serum troponin level have prognostic role in CINCP and is associated with MODS and increased in-hospital mortality.

In MICU group 11(22.4%) females had in-hospital mortality (p value= 0.751). Similarly 18(26.1%) males had in-hospital mortality (p value= 0.251). Sex was not a determinant of in-hospital mortality in CINCP with troponin elevation in MICU group.

In MICU group, patients with the highest troponin levels had higher APACHE II score i.e. more severe medical illness; and early in-hospital death. This explains paradoxical result of negative co-relation of baseline elevation of troponin T with duration of hospitalization in our study; but the co-relation was not statistically significant (Pearson co-relation coefficient = -0.068 and -0.072 respectively; p value = 0.501 and 0.479 respectively)

In accordance with our study **Landesberg G et.al** showed that patient with higher APACHE II score had higher troponin levels and increased mortality (**22)**.

When patients with in-hospital mortality (29%) were removed from analysis there was positive co-relation between troponin levels and duration of hospitalization but this was not statistically significant (Pearson co-relation coefficient = 0.120 and 0.137; p value = 0.338 and 0.237 respectively).

In CCU-NSTEMI group within thirty day of discharge only one (2%) patient died, no patient required re-hospitalization and 42 (98%) patients were alive without any complications. In MICU group within thirty day of discharge 9 (13%) patients died, 5 (7%) patients had re-hospitalization and 57 (80%) patients were alive without any complications. There was increased trend of mortality ≤ 30 days in thrombotic ACS and NTTE (MICU group) as compared to CCU group but this was not statistically significant (p value = 0.543 and 0.268 respectively).

In MICU group there was a positive trend towards increased mortality or re-hospitalization ≤ 30 days after discharge with elevated troponin T but it was not statistically significant (p value for baseline and second sample = 0.157 and 0.564 respectively). Troponin T level in all these patients were as follows; (Mortality ≤ 30 days **vs**. Re-hospitalization **vs**. Alive without complications: **Baseline sample** = 148.82 ± 246.54 **vs.** 161.60 ± 290.18 **vs.** 169.51 ± 228.38 respectively; **Second sample** = 438.40 ± 964.385 **vs**. 175.50 ± 324.683 **vs**. 370.43 ± 624.049 respectively).

In harmony with our study **Landesberg G et.al** showed that elevated troponin levels predicted increased 1-month, 6-month, and 2-yr mortality (odds ratio = 6.0, 3.2, and 2.99, respectively; p < .001) (**22)**. In a study by **Maynard C et.al** showed that critically ill patients with ACS had higher 30-day mortality (odds ratio-2;

95%confidece interval, 1.7-2.4; P<.001) (**94)**. Similarly **S Hajsadeghi et.al, Relos RP et.al, Mcfalls et.al, Antman et.al** found that in patients with ACS, cTn provides prognostic information and identifies patients at increased risk of mortality (**75, 76, 81, and 95** respectively). In comparison to other studies our study showed a trend towards increased ≤ 30 days mortality but this was not statistically significant due to the limited sample size in our study.

In our study troponin T levels (at baseline and within 24 hours of baseline sample) in thrombotic ACS (MICU group) patients were significantly higher than troponin T levels in NTTE patients (373.04 ± 1124.51 vs. 289.16 ± 1062.26 and 743.31 ± 1303.90 vs. 258.62 ± 891.41 respectively; p value for Troponin T 1^{st} and 2^{nd} sample is 0.05 and 0.000 respectively). Similarly in accordance with our result studies by **Relos RP et.al, Vasile VC et.al, Altmann et.al, Booker KJ et.al, Wendy Lim et.al, Jensen JK et.a**l, **Baillard C et.al** also showed an entity of non-thrombotic troponin elevation in critically ill patients (**76-82, 96** respectively).

In our study troponin T baseline and second sample levels in NSTEMI patients in CCU group were not statistically significantly higher than troponin T level in thrombotic ACS patients in MICU group (347.17 ± 566.90 vs. 373.04 ± 1124.51 and 983.47 ± 1922.34 vs. 743.31 ± 1303.90 respectively; p value = 0.892 and 0.502 respectively). Suggesting that serum troponin levels in NSTEMI patient in CCU and ACS patients in MICU were in similar range.

Chest pain, ECG and ECHO changes suggestive of ACS along with ≥ 20% troponin change from first to second sample were used as predictors of ACS in

the study. When ≥ 3 predictors are present the patient was diagnosed as having ACS. For diagnosing ACS we found a sensitivity of 100% (95% CI: 91.51% to 100%), specificity of 48.25% (95% CI: 34.95% to 61.78%), positive predictive value of 58.33% (95% CI: 46.11% to 69.85%), negative predictive value of 100% (95% CI: 87.54% to 100%) and positive likelihood ratio of 1.93 (95% CI: 1.51 to 2.48). In CINCP with low pre test probability of CAD the diagnostic accuracy of troponin for ACS is only 58%.

When at least one serum troponin level > 300 pg/ml or ng/L was used instead of ≥ 20% troponin change from first to second troponin as predictors of ACS in the study; for diagnosing ACS we found a sensitivity of 100% (95% CI: 91.1% to 100%), specificity of 77.59% (95% CI: 64.72% to 87.48%), positive predictive value of 76.36% (95% CI: 62.98% to 86.75%) and positive likelihood ratio of 4.46 (95% CI: 2.76 to 7.20). With the troponin cut off > 300 pg/ml there was similar sensitivity and NPV (100%) for diagnosing ACS but there was increase in specificity, positive predictive value and positive likelihood ratio (77.59% vs. 48.25, 76.36% vs. 58.33 and 4.46 vs. 1.93 respectively).

Thus in critically ill patients with abnormal ECG and ECHO suggestive of ACS and at least one serum troponin level ≥300 pg/ml or ng/L within 48 hours of admission to intensive care unit; ACS can be diagnosed with 100% sensitivity and 77% specificity. Our study shows that in critically ill patients, elevated troponin values (more than twenty times above the 99^{th} percentile of URL) can occur without acute coronary syndrome. The results of our study substantiate the evidence that increase in cTnT is common in CINCP as they are suffering from large range of

underlying medical illness. As per our study in CINCP with low pre test probability of CAD, even if troponin level cut off for diagnosing thrombotic ACS is taken as 300 ng/L (> 20 times above the 99th percentile of URL); 22% patients can still have NTTE and not acute coronary syndrome.

Patients were classified according to APACHE II score into three- groups. Critically ill patients with elevated troponin levels having APACHE II score ≥ 25 were compared to (≥ 12 to 17). Our study shows that in critically ill patients with APACHE II score ≥ 25 with elevated troponin level, the probability that the patient has NTTE is only 50%. For diagnosing NTTE in patients with APACHE II score ≥ 25; the sensitivity was only 56.10% (95% CI: 39.75% to 71.52%), specificity was 17.86% (95% CI: 6.13% to 36.91%), positive predictive value was 50% (95% CI: 34.91% to 65.09%), disease prevalence was 59.52% (95% CI: 46.92% to 71.08%) and Negative predictive value was 21.74% (95% CI: 7.54% to 43.71%).

Similarly for diagnosing NTTE in patients with APACHE II score (≥ 12 to 17) there was sensitivity of 43.90% (95% CI: 28.48% to 60.25%), specificity of 82.14% (95% CI: 63.29% to 92.87%), Positive Predictive value of 78.26% (95% CI: 56.29% to 92.46%), Positive Likelihood ratio of 2.46 (95% CI: 1.03 to 5.85), disease prevalence of 59.52% (95% CI: 46.92% to 71.08%) and Negative predictive value of 50 %. (95% CI: 34.91% to 65.09%). This was statistically significant (p value = 0.022). Our result showed that critically ill patients with elevated troponin level having APACHE II score ≥ 12 to 17 within 24 hour of admission to ICU; the probability that the patient is having NTTE is 80%.

In critically ill ICU patients with elevated troponin levels as severity of illness increases {as documented by increase in APACHE II score from (≥ 12 to 17) to ≥ 25)} the probability of NTTE decreases (80% to 50%). This can be explained as critically ill patients have multiple stressors and pro-coagulant conditions which increases as patient becomes more severely ill {as documented by increase in APACHE II score from (≥ 12 to 17) to ≥ 25)}can cause acute progression in severity of pre-existing coronary artery lesion (Type 1 MI). There can also be concomitant cell damage from hypo-perfusion or hypoxia of non coronary etiology (Type 2 MI) making simultaneous occurrence of both types of MI a possibility (**8)**.

Renal failure was present in 15 patients (15%) in CCU group and 63 patients (63%) in MICU group. In MICU group renal failure was present in 23 cases (54.76%) in ACS and 40 cases (68.97%) in NTTE group. There was a positive trend between renal failure and troponin elevation in NTTE patients as compared to ACS patients but the association was not statistically significant (LR- 1.86; p value 0.147; Odds ratio-1.836; 95%CI - 0.805 -4.184%). In accordance with our finding a study by **R. Alcalai et.al** found that renal failure was significantly associated with troponin elevation, favoring NTTE (p value=0.03) (**24). Landesberg G et.al** found renal failure associated with troponin elevation (**22)**. In presence of renal failure in CINCP the probability of NTTE is high as compared to thrombotic ACS (**24, 45**).

Past history of coronary artery disease was present in 21 patients (21%) in CCU group and 18 patients (18%) in MICU group. In MICU group past history of coronary artery disease was present in 9 cases (50.00%) in ACS and 9 cases (50.00%) in NTTE group. It showed a positive trend between past history of CAD and

thrombotic ACS but it was not statistically significant (LR-4.382, p value 0.451; Odds ratio-0.673; 95% CI - 0.242 -1.875%). In harmony with our result study by **R. Alcalai et.al** showed that past history of coronary artery disease was significantly associated with troponin elevation, favoring thrombotic ACS (p value=0.05) (**24)**

Diabetes mellitus was present in 46 patients (46%) in CCU group and 48 patients (48%) in MICU group. In MICU group DM II was present in 21 cases (45.65%) in ACS and 25 cases (54.35%) in NTTE group. It was non-significantly associated with troponin elevation in NTTE patients (Likelihood Ratio (LR) -2.485; p value 0.416; Odds ratio-0.812; 95% confidence interval (CI) - 0.367 – 1.801%). **R. Alcalai et.al** had shown that DM II was not statistically significantly associated with troponin elevation, favoring thrombotic ACS (p value=0.42) (**24).**

Hypertension was present in 50 patients (50%) in CCU group and 49 patients (49%) in MICU group. In MICU group hypertension was present in 24 cases (48.98%) in ACS and 25 cases (51.02%) in NTTE group. It was non-significantly associated with troponin elevation in NTTE patients (LR- 1.712; p value 0.191; Odds ratio-0.586; 95% CI- 0.262 -1.310%). **R. Alcalai et.al** had shown that hypertension was significantly associated with troponin elevation, favoring NTTE (p value=0.03) (**24)**

In the present study, the medical diagnosis which were most commonly associated with NTTE includes Sepsis (77.59%), ARF (68.97%), Anemia (43.10%), Shock (Septic or hypo-volumic- 31.03%), Pneumonia (25.86%), ARDS (20.69%), Myocarditis (12.07%), CHF (10.34%), Tachyarrhythmia (6.9%), CPR

(6.9%), COPD (3.49%), Brady-arrhythmia, RHD, CVA, AIDP, Diabetic ketoacidosis, GTCS, Snake bite (Each 1.72%).

Out of 100 cases in CCU/CPU group there were 81 males (81%) and 19 females (19%). Male sex was statistically significantly associated with ACS in CCU group (P value 0.05). In STEMI patients there were 51 males (9.1%) and 5 females (8.9%). In NSTEMI patients there were 30 males (68.2%) and 14 females (31.8%). Mean age of cases in CCU/CPU group was 55.7 ± 16.86 years. NSTEMI cases were significantly older than STEMI cases (63.59 ± 10.957 vs. 53.16 ± 10.133, p value 0.000).

Out of 100 cases in MICU/MHDU group there were 51 male (51%) and 49 females (49%). In NTTE group 28 patients were males (48%) and 30 patients were females (52%). Sex was not significantly associated with troponin elevation in NTTE patients (LR-0.411, p value 0.522). In harmony to our result a study done by **R. Alcalai et.al** showed that sex was not significantly associated with troponin elevation, favoring thrombotic ACS (p value=0.94) (**24)**.

Mean age of cases in MICU/MHDU group was 57.75 ± 11. 67 years. NTTE cases were non-significantly younger than ACS cases (53.16 ± 18.32 vs. 59.21 ± 14.08; p value 0.076). In disagreement to our study **R. Alcalai et.al** showed that age between 40 to 80 years was significantly associated with troponin elevation, favoring thrombotic ACS (p value=0.03 to 0.001) (**24)**

In conclusion elevated serum troponin level is a common presentation in critically ill ICU patients even without any evidence of ACS or with normal

epicardial coronaries or without flow limited coronary artery disease (**1, 8, and 20)**. Our study showed that 50% of critically ill patients with multiple co-morbid conditions with elevated troponin values had non-thrombotic troponin elevation. Increased in-hospital and thirty day mortality in NTTE patients in our study substantiates that NTTE patients have worse prognosis as compared to critically ill patients without troponin elevation (**20 to 24)**. Elevated troponin levels needs to be evaluated in clinical scenario and pre test probability before diagnosing them as having ACS and starting them on treatment for ACS (**7)**. There is no evidence that treatment modalities for thrombotic ACS will be beneficial for NTTE in CINCP having low pre test probability of coronary artery disease (**24, 45**).

LIMITATIONS:

1) The major limitation of this study was the fact that diagnosis of patients having NTTE or thrombotic ACS is subjected to misclassification bias. Coronary angiogram was done in only limited number of critically ill patients due to relative contraindications from their medical diseases. As coronary anatomy is not delineated the diagnosis of ACS could not be confirmed and this can lead to misclassification bias. To minimize this classification of patients into NTTE and thrombotic ACS was done after careful clinical evaluation of patient and with maximal adherence to ACC/AHA and ESC guidelines. The diagnosis was supported by objective evidence such as ECG and Echocardiogram.
2) Though the sample size was calculated using appropriate statistical methods, but it was not powered enough to assess the association of traditional risk factors as independent predictors of NTTE. A larger sample size and study will give better and more consolidated information regarding this unique entity: NTTE and its association with traditional risk factors.

SUMMARY AND CONCLUSIONS:

The study was undertaken in Department of Cardiology and Medicine-Division of Critical Care, Christian Medical College Vellore. The present study is done to assess relationship of thrombotic and non-thrombotic troponin elevation. It was aimed to measure serum troponin levels in critically ill patients in MICU/MHDU and ACS patients in CCU/CPU and to correlate it with duration of hospitalization, in-hospital and 30 days mortality. 200 cases were selected in the study, 100 patients of ACS from CCU/CPU (56-STEMI and 44-NSTEMI) and 100 critically ill patients from MICU/MHDU (42-ACS and 58-NTTE) respectively in our hospital. The study was conducted over a period of 18 months from August 2013 to December 2014 in the department of Cardiology and Medicine- Critical Care division.

Following conclusions were drawn from the study performed:

1) Baseline Troponin T level was significantly associated with in-hospital mortality in critically ill medical patients (p value 0.026).
2) In critically ill patients with abnormal electrocardiogram and echocardiogram suggestive of ACS, with serum Troponin levels ≥300 pg/ml or ng/L within 48 hours of admission to intensive care unit; ACS can be diagnosed with 100% sensitivity and 77% specificity.
3) Our study shows that in critically ill patients with APACHE II score ≥ 25 with elevated troponin level, the probability that the patient has NTTE is only 50%. In critically ill ICU patients with elevated troponin levels, as the severity of

illness increases {as documented by increase in APACHE II score from (≥ 12 to 17) to ≥ 25)} the probability of NTTE decreases (80% to 50%).

4) In CINCP with low pre test probability of CAD, the diagnostic accuracy of serum troponin for ACS when used in isolation is only 58%.

5) Troponin T level in ACS patients was significantly higher than troponin T level in NTTE patients (p value for Troponin T 1^{st} and 2^{nd} sample is 0.05 and 0.000 respectively). However the in-hospital mortality in the two subgroups were comparable (26% vs. 31%; p value = 0.667).

6) Troponin T level in NSTEMI patients in CCU group was not significantly higher than troponin T level in ACS patients in MICU group (p value= 0.892 and 0.502 respectively). Suggesting serum troponin levels in NSTEMI patient in CCU and ACS patients in MICU were in similar range.

7) In the present study, the medical diagnoses which were most commonly associated with NTTE includes Sepsis (77.59%), ARF (68.97%), Anemia (43.10%), Shock (Septic or hypo-volumic- 31.03%), Pneumonia (25.86%), ARDS (20.69%), Myocarditis (12.07%), CHF (10.34%), Tachyarrhythmia (6.9%), CPR (6.9%), COPD (3.49%).

Our findings resonate with the findings and conclusion of numerous studies done all over the world in the last two decades. This is the first study of its sort done on Indian population. Further research should be done in future and criteria should be defined by ACC/ AHA and ESC to diagnose NTTE in critically ill ICU patients and guidelines should be given for its management.

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APPENDIX- I

ABBREVIATION

MICU: Medical Intensive Care Unit.

MHDU: Medical High Dependency Unit.

CCU: Coronary Care Unit.

CPU: Chest Pain Unit.

ACS: Acute Coronary Syndrome.

STEMI: ST Elevation Myocardial Infarction.

NSTEMI: Non ST Elevation Myocardial Infarction.

UA: Unstable angina

NTTE: Non Thrombotic Troponin Elevation.

CINCP: Critically ill non cardiac patients.

CKMB: Creatinine Kinase Muscle Brain

LDH: Lactate dehydrogenase

CAD: Coronary artery disease

cTnT: Cardiac Troponin T

cTnI: Cardiac Troponin I

RWMA: Regional wall motion abnormality.

ARDS: Acute respiratory distress syndrome.

ICD: International Classification of Diseases

APPENDIX II-A

PATIENT CONSENT FORM

Study Title: **Assessment of thrombotic and non-thrombotic troponin elevation**

Study Number:

Participant's Name:

Date of Birth / Age (years):

Please tick boxes

(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 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. [ ]

Signature of the Subject/

Legally Acceptable Representative: _____

(**Or** Thumb impression)

Date: _____/_____/_____

Signatory's Name: _____

Signature of the Investigator: _____

Date: _____/_____/_____

Study Investigator's Name: _____

Signature of the Witness: _____

Date: _____/_____/_____

Name of the Witness: _____

APPENDIX II-B

TOPIC: Assessment of thrombotic and non thrombotic troponin elevation

CASE STUDY PROFORMA

CASE STUDY No:

NAME:

FATHER'S/ HUSBAND'S NAME:

CMC HOSPITAL NO.:

AGE AND SEX:

MARITAL STATUS:

ADDRESS:

PHONE NO.: MOBILE NO: LANDLINE NO:

DATE OF ADMISSION:

DATE OF ENROLLMENT:

PLACE OF ADMISSION: CCU / CPU / MICU / MHDU

CHEST PAIN: YES / NO

A) TYPICAL-ALL THREE PRESENT

B) ATYPICAL-LESS THAN THREE PRESENT

a) SUBSTERNAL CHEST PAIN

b) DISCOMFORT PROVOKED BY EXERTION AND /OR EMOTIONAL STRESS

c) WAS RELIEVED WITH REST AND/OR NITROGLYCERINE

CLINICAL DIAGNOSIS AT ADMISSION:

RISK ASSESSMENT

HYPERTENTION : YES / NO

DIABETES MELLITUS : YES / NO

HISTORY OF SMOKING : YES / NO IF YES: ACTIVE /REFORMED

HISTORY OF CARDIOVASCULAR

DISEASE (ACS/CVA) IN PAST : YES / NO

HYPOTHYROIDISM : YES / NO

DYSLIPIDEMIA : YES / NO

RENAL FAILURE : YES / NO eGFR: ml/min/1.73m2

PATIENT INTUBATED AND VENTILATED : YES / NO IF YES: PEEP-

EXAMINATION FINDINGS AT ADMISSION

JVP - RAISED/NOT RAISED

TEMPERATURE -

PULSE- /MIN REGULAR/IRREGULAR

ALL PERIPHERAL PULSES PALPABLE - YES / NO

BP- / MMHG

RESPIRATORY RATE - /MIN

RS- CLEAR / RHONCHI / CREPTS

CVS- S1 - S2-

S3- YES / NO S4- YES / NO

MURMUR---- YES / NO

ETIOLOGY

A) THROMBOTIC CORONARY ARTERY DISEASE (ACS): YES / NO

B) NON THROMBOTIC CONDITIONS WITH TROPONIN ELEVATION: YES/NO

1) COPD
2) PULMONARY EMBOLISM
3) ARDS

4) PULMONARY OEDEMA

5) INTRACRANIAL HEMMORRHAGE/SUBDURAL HAEMATOM/SAH

6) CVA

7) HYPOTENTION/SHOCK

8) ACUTE/CHRONIC RENAL FAILURE

9) HYPOVOLUEMIA

10) SEPSIS

11) ANAEMIA

12) CARDIAC CONTUSION

13) ACUTE GASTROINTESTINAL BLEED

14) ACUTE PANCREATITIS

15) HYPERCARBIA

16) HYPOXIA

17) DIABETIC KETOACIDOSIS

18) ACUTE AORTIC DISSECTION

19) ACUTE HEART FAILURE

20) PERIMYOCARDITIS

21) ENDOCARDITIS

22) TAKO-TSUBO CARDIOMYOPATHY

23) TACHYARRHYTHMIAS / BRADYARRHYTHMIA

24) RADIOFREQUENCY CATHETER ABLATION

25) TRAUMA

26)CATECHOLAMINES /SYMPATHOMIMETICS

27)CHEMOTHERAPY INDUCED

28)HIGH PEAK END EXPIRATORY PRESSURE

29)OTHER CONDITION THAN LISTED

INVESTIGATIONS:

1) HEMOGLOBIN-
2) HEMATOCRIT-
3) TLC –
4) DLC--

N	E	L	B	M

5) PLATELET COUNT-
6) SERUM SODIUM-
7) SERUM POTASSIUM-
8) SERUM CREATININE-
9) BLOOD UREA

10)LIPIDS :

TOTAL CHOLESTEROL	TRIGLYCERIDES	HDL	LDL

NON HDL CHOLESTEROL-

11)RANDOM BLOOD GLUCOSE LEVEL ATADMISSION-

12)HBA1C-

13)TSH-

14)CKMB

HOURS	WITHIN 24 HOURS OF ADMISSION	WITHIN 24 HOURS OF FIRST SAMPLE
LEVELS		

15)TROPONIN -

HOURS	WITHIN 24 HOURS OF ADMISSION	WITHIN 24 HOURS OF FIRST SAMPLE
LEVELS		

16) **ECG** : a) NEW ONSET LBBB - YES / NO

b) T WAVE INVERSION - YES / NO LEADS-

c) ST SEGMENT DEPRESSION - YES / NO LEADS-

d) ST SEGMENT ELEVATION - YES / NO LEADS-

e) Q WAVES- YES / NO NEW ONSET / OLD LEADS –

17) **ECHO :** LVEF- , MR- ,TR- ,PE-

RWMA- LVIDs- LVIDd- LA-

LVD - YES / NO, IF YES---MILD /MODERATE / SEVERE

18) CORONARY ANGIOGRAPH (WHEN APPLICABLE)-

a) CULPRIT LESION→ a) ≥80% STENOSIS

b) THROMBUS CONTAINING LESION

c) ≤ TIMI II FLOW

b) LEFT MAIN

c) LEFT ANTERIOR DESCENDING

d) LEFT CIRCUMFLEX

e) RIGHT CORNARY

f) INTERVENTION DONE-

19) APACHE II SCORE FOR MICU PATIENTS -

a) ACUTE PHYSIOLOGY SCORE

b) GLASGOW COMA SCORE

c) POINTS FOR AGE

d) CHRONIC HEALTH CONDITIONS

DISCHARGE DIAGNOSIS -

INHOSPITAL OUTCOME -

a) IN HOSPITAL MORTALITY : YES/ NO

b) MYOCARDIAL INFARCTION : YES / NO

IF YES STEMI / NSTEMI

c) CAG AND URGENT REVASCULARISATION - YES / NO

OUTCOME ≤30 DAYS AFTER DISCHARGE -

a) MORTALITY : YES / NO

b) REHOSPITALISATION : YES / NO

IF YES- WITHIN HOW MANY DAYS OF DISCHARGE-

c) RE INFARCTION : YES / NO

SN	HOSP NO	AGE	SEX	DT OF ADMISSION	DATE OF ENROLLMENT	PLACE	CHEST PAIN	TYPE OF CHEST PAIN	DIAGNOSIS AT ADMISSION	KILLIP	HTN	DM II	SMOKER	HYPOTHYROIDISM	DYSLIPIDEMIA	PAST H/O CAD	RENAL FAILURE	INTUBATED
1	713702F	50	M	13/11/2013	13/11/2013	CCU	1	1	IWMI	I	2	2	2	2	1	2	2	2
2	713701F	65	M	13/11/2013	13/11/2013	CCU	1	1	AWMI	IV	1	1	2	2	1	2	1	2
3	713709F	54	M	13/11/2013	13/11/2013	CPU	1	1	IWMI, PWMI, RVMI	II	2	1	2	2	1	2	2	2
4	708552F	70	F	13/11/2013	13/11/2013	CPU	2	3	NSTEMI		1	1	2	2	1	1, CVA	1	2
5	713710F	65	M	13/11/2013	13/11/2013	CPU	1	1	IWMI,RVMI	II	1	2	2	2	1	2	2	2
6	159355F	53	M	15/11/2013	15/11/2013	CPU	1	1	IWMI,RVMI,PWMI	I	2	2	1	2	1	2	2	2
7	713756F	65	F	14/11/2013	15/11/2013	CPU	1	2	NSTEMI		1	1	2	2	1	2	2	2
8	649442C	50	M	15/11/2013	15/11/2013	CPU	1	1	ASMI	II	1	1	2	2	1	1, ACS	1	2
9	569119B	56	M	15/11/2013	15/11/2013	CCU	1	1	NSTEMI		2	2	1	2	1	1, ACS	2	2
10	720081F	44	M	17/11/2013	17/11/2013	CPU	1	2	AWMI	II	2	2	1	2	2	2	2	2
11	275103D	31	M	17/11/2013	17/11/2013	CPU	1	1	AWMI	I	2	2	1	2	1	2	2	2
12	720101F	51	M	17/11/2013	17/11/2013	CPU	1	1	IWMI	I	2	2	1	2	1	2	2	2
13	121465B	78	F	17/11/2013	17/11/2013	CCU	1	2	NSTEMI		1	1	2	2	1	2	2	2
14	720097F	66	F	17/11/2013	18/11/2013	CCU	1	2	NSTEMI		2	2	1	2	1	2	2	2
15	720143F	34	M	18/11/2013	19/11/2013	CCU	1	1	AWMI	I	2	1	2	2	1	2	2	2
16	720130F	72	M	18/11/2013	19/11/2013	CCU	1	1	AWMI	II	1	2	2	2	2	2	2	2
17	903164C	52	M	18/11/2013	19/11/2013	CCU	1	2	NSTEMI		2	2	2	2	1	2	2	2
18	720179F	52	M	19/11/2013	19/11/2013	CPU	1	1	AWMI	I	2	2	2	2	2	2	2	2
19	510679B	38	M	23/11/2013	24/11/2013	CCU	1	1	AWMI	II	2	2	1	2	1	2	2	2
20	623867F	70	M	24/11/2013	25/11/2013	CCU	1	1	NSTEMI		1	1	2	2	1	1, ACS	2	2
21	720649F	50	M	24/11/2013	25/11/2013	CCU	1	1	NSTEMI		1	2	1	2	1	1, ACS	2	2
22	655540F	71	M	24/11/2013	25/11/2013	CCU	2	3	NSTEMI		2	2	2	1	1	1, ACS	1	1
23	720623F	57	M	24/11/2013	25/11/2013	CPU	1	1	AWMI	II	1	2	1	2	2	2	2	2
24	720624F	71	M	24/11/2013	25/11/2013	CPU	1	1	IWMI,RVMI	I	2	2	2	2	1	2	2	2
25	294719B	44	M	22/11/2013	23/11/2013	CCU	2	3	NSTEMI		1	2	2	2	2	2	2	2
26	720735F	59	M	25/11/2013	26/11/2013	CCU	1	1	AWMI	I	2	2	2	2	1	2	2	2
27	720769F	50	F	26/11/2013	26/11/2013	CPU	1	2	IWMI, RVMI, PWMI	I	2	1	2	2	1	2	2	2
28	720731F	80	F	25/11/2013	26/11/2013	CPU	2	3	IWMI, PWMI	I	1	2	2	2	1	2	1	2
29	408888D	51	M	27/11/2013	28/11/2013	CCU	1	1	AWMI	I	2	1	2	1	1	2	2	2
30	151157D	76	F	27/11/2013	27/11/2013	CCU	1	1	IWMI,RVMI, PWMI	I	1	1	2	2	2	2	2	2
31	622344F	65	F	28/11/2013	29/11/2013	CCU	1	1	IWMI	I	1	2	2	1	1	2	2	2
32	791763B	79	M	29/11/2013	29/11/2013	CCU	2	3	NSTEMI		1	1	1	2	1	1, ACS	1	2
33	384654F	59	M	28/11/2013	29/11/2013	CCU	2	3	NSTEMI		2	2	1	2	1	1, ACS	2	2
34	720801F	62	M	29/11/2013	29/11/2013	CCU	1	1	STEMI	I	2	1	1	2	1	1, ACS	1	2
35	740095F	51	M	10/12/2013	11/12/2013	CPU	1	1	AWMI	II	2	2	2	2	2	2	2	2
36	740214F	48	M	11/12/2013	12/12/2013	CPU	1	1	AWMI	I	2	2	1	2	1	2	2	2
37	726959F	54	M	11/12/2013	11/12/2013	CPU	1	1	AWMI	I	2	2	2	2	2	2	2	2
38	726987F	65	M	11/12/2013	12/12/2013	CPU	1	1	AWMI	I	2	2	2	2	2	2	2	2
39	740235F	50	M	12/12/2013	13/12/2013	CPU	1	1	NSTEMI		2	2	1	2	1	2	2	2
40	740247F	70	M	12/12/2013	13/12/2013	CPU	2	3	NSTEMI		1	1	1	2	1	1, ACS	2	2
41	824266C	52	M	13/12/2013	14/12/2013	CPU	1	2	AWMI	I	1	1	1	2	1	2	2	2
42	740625F	50	M	15/12/2013	16/12/2013	CPU	1	1	IWMI, PWMI	I	1	2	1	2	1	2	2	2
43	893481C	52	M	15/12/2013	16/12/2013	CCU	1	1	IWMI	II	2	2	1	2	1	1, ACS	2	2
44	740651F	37	M	16/12/2013	16/12/2013	CPU	1	1	AWMI	I	1	2	1	2	1	2	2	2
45	743754F	52	M	16/12/2013	17/12/2013	CCU	1	2	NSTEMI		1	1	1	2	1	2	2	2
46	740757F	48	M	17/12/2013	18/12/2013	CPU	1	2	IWMI	I	1	1	2	2	1	2	2	2
47	740761F	67	F	17/12/2013	18/12/2013	CPU	1	1	NSTEMI		1	1	2	2	1	2	2	2
48	740336F	52	M	18/12/2013	18/12/2013	CPU	1	2	IWMI, RVMI, PWMI	I	2	1	1	2	1	2	2	2
49	750456F	40	M	22/12/2013	23/12/2013	CCU	1	1	AWMI	I	2	2	1	2	2	2	2	2

50	750448F	43	M	22/12/2013	23/12/2013	CPU	1	2	AWMI	I	2	1	2	2	1	2	2	2
51	750500F	50	M	23/11/2013	24/12/2013	CCU	1	1	NSTEMI	I	1	2	2	2	1	2	2	2
52	744347D	75	F	25/12/2013	26/12/2013	CPU	2	3	NSTEMI		1	1	2	2	1	2	2	2
53	750411D	53	M	23/12/2013	24/12/2014	CPU	1	1	AWMI	I	1	1	1	2	1	2	2	2
54	720775F	28	M	26//12/2013	27/12/2013	CPU	1	1	AWMI	I	2	2	1	2	2	2	2	2
55	180276F	62	M	26/12/2013	27/12/2013	CPU	1	2	NSTEMI		1	1	1	2	1	2	1	2
56	591988C	44	M	27/12/2013	27/12/2013	CPU	1	2	NSTEMI		2	2	2	2	1	2	2	2
57	750175F	53	F	27/12/2013	27/12/2013	CPU	1	1	NSTEMI		2	2	2	2	1	2	2	2
58	448582F	60	M	27/12/2013	28/12/2013	CPU	1	2	LATERAL WALL STEMI		2	1	2	2	1	1, ACS	2	2
59	039957D	71	F	28/12/2013	28/12/2013	CPU	1	2	NSTEMI		1	1	2	2	1	1, ACS	2	2
60	750726F	60	M	30/12/2013	31/12/2013	CCU	2	3	NSTEMI		1	1	1	2	1	1, ACS	2	2
61	332918B	80	M	31/12/2013	01/01/2014	CCU	1	1	NSTEMI		1	2	2	2	2	1, ACS	2	2
62	750255F	63	F	28/12/2013	29/12/2013	CPU	1	2	IWMI,PWMI	III	1	1	2	2	1	2	2	1
63	751213F	59	F	02/01/2014	03/01/2014	CPU	1	1	IWMI, RVMI	I	2	2	2	2	1	2	2	2
64	062059F	57	M	01/01/2014	02/01/2014	CPU	1	1	AWMI	I	2	2	1	2	1	2	2	2
65	751272F	59	M	06/01/2014	06/01/2014	CCU	1	1	NSTEMI		2	2	2	2	1	2	2	2
66	570803D	52	M	06/01/2014	07/01/2014	CCU	1	2	IWMI	I	2	1	1	2	1	2	2	2
67	984562D	61	M	06/01/2014	07/01/2014	CPU	1	2	IWMI	I	1	1	2	2	1	2	2	2
68	750818F	59	M	04/01/2014	05/01/2014	CPU	1	2	IWMI	I	2	2	2	2	1	2	2	2
69	751298F	65	M	06/01/2014	07/01/2014	CPU	1	1	IWMI, RVMI	I	1	1	2	2	1	2	2	2
70	166379F	59	M	08/01/2014	08/01/2014	CCU	1	2	NSTEMI		1	1	2	1	2	2	2	2
71	243871C	63	M	07/01/2014	08/01/2014	CCU	1	1	AWMI	II	1	2	1	2	1	2	2	2
72	751467F	74	F	09/01/2014	10/01/2014	CPU	1	1	NSTEMI		1	2	2	2	1	2	2	2
73	423954C	66	M	09/01/2014	10/01/2014	CPU	1	1	NSTEMI		2	1	1	2	1	2	2	2
74	209951D	45	M	10/01/2014	10/01/2014	CPU	1	2	NSTEMI		1	1		2	1	1, ACS	2	2
75	779922F	51	M	10/01/2014	11/01/2014	CCU	1	2	AWMI	I	1	1	2	2	1	2	2	2
76	751534F	61	M	11/01/2014	12/01/2014	CPU	1	1	NSTEMI		2	2	2	2	1	2	2	2
77	751786F	34	M	13/01/2014	14/01/2014	CCU	1	1	AWMI	I	2	2	2	2	1	2	2	2
78	751885F	60	M	15/01/2014	16/01/2014	CCU	2	3	NSTEMI		1	2	1	2	1	2	1	2
79	753122F	53	M	18/01/2014	19/01/2014	CCU	1	1	ASMI	IV	1	1	1	2	1	2	1	2
80	713237F	59	M	19/01/2014	20/01/2014	CCU	2	3	NSTEMI		2	1	2	2	2	2	2	2
81	753181F	42	M	19/01/2014	20/01/2014	CPU	1	1	AWMI	I	2	2	1	2	2	2	2	2
82	709015F	54	M	20/01/2014	20/01/2014	CPU	1	1	AWMI	I	2	2	1	2	1	2	2	2
83	753193F	44	M	19/01/2014	20/01/2014	CPU	1	1	AWMI	II	2	2	1	2	2	2	2	2
84	753455F	57	M	22/01/2014	23/01/2014	CCU	1	2	IWMI	I	1	1	2	2	1	2	2	2
85	753174F	81	M	23/01/2014	24/01/2014	CCU	1	2	NSTEMI		1	1	2	2	1	2	1	2
86	520229B	75	M	22/09/2014	23/09/2013	CCU	1	1	NSTEMI		1	1	1	2	1	1, ACS	1	2
87	922350F	55	M	18/09/2014	19/09/2014	CCU	1	1	NSTEMI		1	2	1	2	1	2	2	2
88	922802F	64	M	25/09/2014	25/09/2014	CPU	1	1	NSTEMI		2	2	1	2	1	2	2	2
89	923163F	65	M	01/10/2014	01/10/2014	CPU	1	1	NSTEMI		1	1	1	2	1	2	2	2
90	199091D	71	F	29/09/2014	30/09/2014	CPU	1	1	NSTEMI		1	1	2	1	1	1, CABG	1	2
91	505223F	42	F	07/10/2014	07/10/2014	CPU	2	3	NSTEMI		2	1	2	2	1	2	1	2
92	412556C	79	F	09/10/2014	10/10/2014	CPU	1	1	NSTEMI		1	2	2	2	1	2	2	2
93	310290C	49	M	16/10/2014	16/10/2014	CPU	1	1	AWMI		1	1	1	2	1	2	2	2
94	926377F	60	M	17/10/2014	17/10/2014	CPU	1	2	NSTEMI		2	2	2	2	2	2	2	2
95	926356F	62	M	17/10/2014	17/10/2014	CPU	1	2	IWMI	I	2	2	1	2	1	2	2	2
96	116703F	59	M	17/10/2014	17/10/2014	CPU	1	1	AWMI	I	2	1	1	2	1	1	2	2
97	928556F	55	F	30/10/2014	30/10/2014	CPU	1	2	NSTEMI		1	2	2	2	1	2	2	2
98	928322F	83	M	27/10/2014	28/10/2014	CCU	1	1	NSTEMI		1	2	2	2	1	1	1	2
99	928434F	70	F	29/10/2014	29/10/2014	CPU	1	2	NSTEMI		1	1	2	2	1	1	2	2
100	928609F	57	M	31/10/2014	31/10/2014	CPU	1	1	NSTEMI		2	2	2	2	2	2	2	2
101	929351F	74	M	09/11/2014	09/11/2014	CCU	2	3	NSTEMI		1	1	1	2	2	2	1	2

SN	HOSP NO	IF INTUBATED-PEEP	Hb	HCT	TLC	Na	K	S.CREATININE	B.UREA	LIPID PROFILE	TG	HDL	LDL	NON HDL C.	GRBS AT ADMISSION	CKMB1	CKMB2	TROP T1	TROP T2	ECG AT ADM	ECHO
1	713702F		9.4	30.4	12800	135	4.6	1.07	21	193	148	33	151	160	126	18.24	216.4	190.8	8845	2	2
2	713701F		13.7	35.9	11900	139	3.6	1.33	36	156	83	34	113	122	186	4.1	376.5	50.77	>10,000	2	2
3	713709F		14.4	42.7	12300	124	3.8	0.71	25	117	95	31	80	86	147	6.31	29.76	68.19	1516	2	2
4	708552F		9	27	15500	126	4.7	1.67	37	94	113	34	42	52	190	1.96	3.02	23.03	68.03	2	2
5	713710F		12.4	39.1	12100	139	3	1	26						110	4.27	14.91	20.5	1213	2	2
6	159355F		14.5	43.8	8600	141	3.5	1.3	15	221	156	32	165	189	90	4.78	324.4	36.04	>10,000	2	2
7	713756F		10.7	33	12100	128	4.9	1.16	48	163	144	36	112	127	221	11.39	10.54	414.1	815.1	2	2
8	649442C		14.9	46.3	26900	131	5.1	1.51	40	166	198	27	109	139	108	10.31	100.2	217.1	7408	2	2
9	569119B		14.5	42.7	8900	133	3.7	1.07	19	224	72	39	172	185	140	13.82	102.8	52.51	139.8	2	2
10	720081F		14.3	42.4	14500	134		1.17	13						98	427.2	171.3	3853	>10000	2	2
11	275103D		15	44.2	17000	138	3.4	1.14	17	200	115	38	150	162	68	10.28	500	69.78	6330	2	2
12	720101F		12.2	36	21100	133	3.3	1.31	19						90	23.06	357.3	31.65	>10000	2	2
13	121465B		12.7	38	14300	134	4.5	1.03	21	211	176	46	148	165	126	27.99	49.23	324.5	937	2	2
14	720097F		15.3	46	13100	129	4.6	1.32	34	208	156	33	169	175	201	8.5	29.82	77.19	468.9	2	2
15	720143F		14.5	42.3	17800	133	4.1	1.15	17	132	62	33	102	99	210	7.76	59.55	61.34	4262	2	2
16	720130F		15.4	46	7000	136	3.4	1.25	44	174	61	45	128	129	128	4.7	>500	8.28	>10000	2	2
17	903164C		14.2	42.6	8100	134	4.3	1.15	17	138	77	26	98	112	110	18.69	19.26	283.4	315.4	2	2
18	720179F		13.4	40	11000	136	3.9	1.14		160	104	40	107	120	78		89.06	119.3	1029	2	2
19	510679B		17.7	53	14300	135	3.6	1.07	21						150	4.41	114.2	21.87	280	2	2
20	623867F		11.6	34.8		135	4.6	1.08	30	214	98	40	160	174	126	12.78	17.06	355.9	393.2	2	2
21	720649F		18.1	54	9200	137	4.3	0.77	18	241	223	39	164	202	126	6.92	88.45	43.84	396.9	2	2
22	655540F	10	13	41.5	9800	117	5.1	1.87	68						130	27.62	33	110.5	119	2	2
23	720623F		13.5	41.6	12000	135	3.8	1.26	23	142	99	33	86	109	138	115	53.45	1254	7374	2	2
24	720624F		14.8	44.2	9200	136	3.6	1.19	34	126	64	37	81	89	160	43.8	124.9	281	>10000	2	2
25	294719B		14.9	43.9	10500	135	3.6	1.24	19	154	171	43	98	111	100	5.92	5.84	310.5	205.7	2	2
26	720735F		12.8	37.6	15800	133	3.5	0.83	28	152	78	36	110	116	120	26	27.86	2583	2859	2	2
27	720769F		10.3	30.7	12800	132	4.4	0.78	18	169	61	45	112	124	198	61.89		1001		2	2
28	720731F		11.2	37.3	15300	138	4.3	1.52	34	174	149	40	119	134	140	>500	>500	>10000	>10000	2	2
29	408888D		11.6	33.6	11500	135	3.7	1.08		160	116	34	124	126	206	2.99	219.8	3.01	5276	2	2
30	151157D		12.9	39.4	10300	137	3.8	0.73	24	146	90	41	99	105	246	2.94	10.07	31.26	127.7	2	2
31	622344F		13.1	39.3	13900	133	3.5	0.8	29	216	148	33	162	183	119	90.58	128	749.5	2015	2	2
32	791763B		13	39	21200	130	3.7	1.47	29	152	137	46	80	70	160	3.52	5	33.17	183.9	2	2
33	384654F		12.6	36.5	20200	132	4.1	1.22	24	140	122	37	94	103	505	6.62	10.47	21.67	119.5	2	2
34	720801F		13	40.2	17800	130	4.4	1.47	37	162	136	42	107	120	252	5.59	206.2	47.53	>10000	2	2
35	740095F		15.2	41.8	16800	138	3.9	0.62	30	183	47	73	106	110	138	76.43	>500	753	>10000	2	2
36	740214F		12.8	39	16800	135	3.9	1.08	31	127	94	28	79	99	126	45.6	17.77	3258	4251	2	2
37	726959F		13	39.2	14600	134	4.4	1.25	23	115	60	35	74	80	110		8.13	423.5	3917	2	2
38	726987F		13.7	41	15700	134	5.2	0.99	35	150	40	46	108	104	118	>500	22.11	3898	6956	2	2
39	740235F		17	51	7900		3.7	0.7		228	94	57	150	171	116	14.14	89.51	113.8	634.8	2	2
40	740247F		15.2	46	25000	138	4.3	1.29	47	197	137	48	123	149	122	59.37	77.45	1602	2547	2	2
41	824266C		18.4	53	8200		4.1	0.77	14	183	102	46	123	137	132	5.7	4.32	317	480	2	2
42	740625F		15.4	45.3	14300	136	3.3	0.87	26	185	143	25	137	160	162	213	108.5	5092	1436	2	2
43	893481C		14.8	43.6	15500	138	5.4	1.05	20	189	173	37	130	162	150	32.06	25.04	652.6	534.6	2	2
44	740651F		16.5	49.4	21700	137	4.1	1.02	23	153	84	34	104	119	126	31.15	231.1	529.9	9673	2	2
45	743754F		14.4	43.2	9400	136	3.4	1.2	32	137	111	31	92	106	145	4.75	16.69	17.55	107.4	2	2
46	740757F		9	25.4	12100	133	4.3	1.1	29	115	147	23	70	92	210	29.83	25.71	1533	1872	2	2
47	740761F		12.2	36.3	9700	135	3.6	0.74	21	184	155	47	116	137	333	8.63	13.29	238.1	492.2	2	2
48	740336F		18.6	53.6	14500	125	3.9	0.77	13	147	168	31	98	116	368	2.8	60.96	11.05	1467	2	2
49	750456F		15.2	45.7	17200	139	3.5	0.97	21	184	97	42	128	142	122	18.2	>500	285.4	>10000	2	2

50	750448F		15.1	45	9600	130	4.1	0.73	25	142	142	46	81	98	337	428		>10000	6233	2	2
51	750500F		11.9	34	12200	137	3.4	1.01	20	206	112	30	156	176	132	9.83	28.78	403.9	1211	2	2
52	744347D		12.3	46	9300	135	3.9	0.75	23	133	205	39	82	94	302	4.8	6.29	51.28	105.4	2	2
53	750411D		14.9	42.3	13000	131	3.6	0.85	19	179	120	39	121	140	256	8.52	93.61	94.75	6967	2	2
54	720775F		16.2	47	13900	141	3.8	0.76	14	166	152	40	113	126	111	6.47	173.7	85.29	7290	2	2
55	180276F		10.6	32	13100	137	4.7	1.44	64	183	86	28	130	155	108	10.2	109	97.5	400.7	2	2
56	591988C		15.3	42.1	7100	140	4.6	0.85	32	162	190	33	106	129	102	5.29	6.56	133.1	174.7	2	2
57	750175F		11.3	34.5	14100	135	4.1	0.7	18	179	71	54	124	127	210	18	22.96	157.7	310.2	2	2
58	448582F		15.4	45		131	4.9	0.95		164	124	44	108	120	233	145.2	173.3	988.1	>10000	2	2
59	039957D		10.4	31.2	12400	139	4	1.09	25	172	135	65	100	107	110	3.84	23.83	24.44	559.6	2	2
60	750726F		13	39	15900	137	3.8	1.34	27	55	54	33	17	38	188	0.24	15.48	35.55	239.9	2	2
61	332918B		11.6	32.6	8400	130	4.2	1.02	28	125	47	45	74	80	108	66.53	76.98	398.8	640.8	2	2
62	750255F	8	8.5	26.4	16100	136	3.5	0.73	38	110	134	25	61	90	330	4.66	13.87	36.5	1882	2	2
63	751213F		12	36.9	11800	140	3.9	0.92	33	248	190	36	182	212	210	2.35	148.4	32.9	6742	2	2
64	062059F		13.1	38.6	7700	139	3.5	0.87	24	186	62	48	123	138	98	2.78	205.6	12.95	4254	2	2
65	751272F		8.6	19.8	13500	134	4.1	1.47	28	105	65	25	67	80	110	22.06	215.1	274.4	9049	2	2
66	570803D		14.6	42.9	18000	132	3.7	0.87	23	176	153	44	108	132	214	3.39	182.1	24.85	4368	2	2
67	984562D		12.2	46	17500	136	4.5	1.37	27	184	169	36	122	148	289	18.81	268.2	191	>10000	2	2
68	750818F		12.5	37	10000	135	3.6	0.9	20	117	65	33	79	84	103	64.69	46.07	2098	2131	2	2
69	751298F		15.6	45	18500	135	3.7	1.16	47	186	137	37	122	149	254	190.3	175.2	863.7	4370	2	2
70	166379F		10.7	33.2	11300	137	4.1	0.71	19	133	115	55	74	89	301	4.89	8.36	21.1	63.32	2	2
71	243871C		13	38.2	22900	138	4.7	0.92	47	202	52	41	153	161	128	3.9	114.5	64.66	1956	2	2
72	751467F		11.7	34.4	10500	140	3.1	0.9	33	202	132	38	148	164	201	2.53	5.36	21.82	110.8	2	2
73	423954C		13	39.8	8800	137	3.7	0.6	18	134	138	26	90	108	186	24.07	53.68	379.2	1917	2	2
74	209951D		14	42	9300	138	4.4	0.91	19	195	144	34	133	161	201	8.21	18.54	156.7	206.5	2	2
75	779922F		12.4	36.4	11600	136	3.5	0.96	24	149	128	24	92	15	188	4.22	7.81	2227	2262	2	2
76	751534F		14.3	42.6	13700	139	4.3	0.95	30	154	135	28	102	136	98	55.28	114	629.9	1656	2	2
77	751786F		15.3	46	9800	135	3.5	1.06	26	177	60	39	125	138	93	48.76	109	443.5	7856	2	2
78	751885F		15.5	44.8	23200	128	5	1.61	40	133	58	32	93	101	112	4.74	29.02	151.7	629.7	2	2
79	753122F		13.5	39.9	15100	127	4	1.51	51	125	117	29	80	96	239	16.12	17.67	2054	1543	2	2
80	713237F		11.9	36.9	18400	133	4.3	0.91	25	108	92	43	46	65	93	6.86	7.96	123.9	214.1	2	2
81	753181F		13.3	39.6	7300	133	4.5	0.66	25	151	86	43	103	108	88	419.9	113.9	>10000	8562	2	2
82	709015F		14.3	42	9300	137	3.9	0.93	19	185	96	31	134	154	102	3.72	51.88	42.77	1299	2	2
83	753193F		16.2	48	26700	109	4.3	0.88	25	144	83	46	93	98	301	>500	>500	>10000	9502	2	2
84	753455F		15.6	46	16000	129	3.5	0.66	22	131	125	31	87	100	89	277	230	802.8	4392	2	2
85	753174F		11.3	31.4	17300	128	4.5	1.69	65	125	93	39	69	86	111	47.53	13.24	1227	1749	2	2
86	520229B		14.2	43	12500	133	4.3	1.37	26	127	106	34	77	93	137	8.58	7.44	170.2	203.2	2	2
87	922350F		14.9	43	11680	135	4.4	1.03	32	149	103	25	108	124	120	4.33	4.13	42.39	49.25	2	2
88	922802F		14.3	43.6	14400	134	3.9	0.94	36	126	45	36	78	90	112	>300	87.13	2050	5423	2	2
89	923163F		12.2	36.5	13700	133	4.2	0.97	27	150	195	27	94	123	134	3.59	8.86	120	317.6	2	2
90	199091D		9.7	29	17500	132	4.3	1.26	38	184	82	36	144	148	244	37.44	59.33	1521	8119	2	2
91	505223F		11.4	33.8	16000	133	4.4	1.57	64	68	129	11	47	19	223	6.61	8.02	2580	1776	2	2
92	412556C		12.6	38.7	7600	135	4.5	0.67	19	253	248	43	192	210	190		3.76	41.38	151.6	2	2
93	310290C		13.7	41.5	15100	133	3.7	0.77	17						210	5.35	3	190.4	461.3	2	2
94	926377F		12.6	36.8	9400	133	3.9	0.87	22	131	91	29	81	102	110	2.81	89.53	8.53	60.89	2	2
95	926356F		13	38.8	10900	139	3.9	0.69	34	152	96	32	97	120	108	>300	214.5	4315	5908	2	2
96	116703F		9.3	28.4	3500		4.5	1.1	28	177	97	32	120	145	128	2.41	28.86	54.03	711.6	2	2
97	928556F		13.7	39.8	8300	134	4	0.83	35	245	140	48	175	197	140		5.8	40.4	143.7	2	2
98	928322F		8.5	26.1	14300	132	4.6	1.49	90	102	84	30	55	72	201	10.78	12.31	434.6	452.5	2	2
99	928434F		13	40.4	9880	137	4.2	0.75	30	154	124	40	90	114	202	18.59	15.77	247.3	234.2	2	2
100	928609F		12.6	39.4	7550	141	4.3	1.18	22	152	66	47	94	105	110	3.82	5	31.1	60	2	2
101	929351F		10.5	31.6	13000	128	6.4	2.11	59	157	140	37	94	120	212	4.11	5.81	91.14	371.9	2	2

SN	HOSP NO	CAG	SVD (1), DVD (2), TVD (3), LM (4)	FINAL DIAGNOSIS	STEMI (1)/ NSTEMI(2)	URGENT REVASCULARISATION	INHOSPITAL MORTALITY	OUTCOME ≤30 DAYS AFTER DISCHARGE-(1)-MOR, (2)REHOSPITAL, (3)- Rein (4)- NONE, (5) IN Hosp Pth	≤30 DAY MORTALITY	THROM(1) / NONTHROM (2)
1	713702F	1	2	IWMI	1	1	2	4	2	1
2	713701F	1	4,3	AWMI	1	1	2	4	2	1
3	713709F	1	1	IWMI,PWMI,RVMI	1	1	2	4	2	1
4	708552F	1	4,3	CAD-TVD, NSTEMI	2	2	2	4	2	1
5	713710F	2		IWMI,RVMI	1	2	2	4	2	1
6	159355F	1	1	IWMI,RVMI,PWMI	1	1	2	4	2	1
7	713756F	2		NSTEMI	2	2	2	4	2	1
8	649442C	2		ASMI	1	2	2	4	2	1
9	569119B	1	3	NSTEMI	2	2	2	4	2	1
10	720081F	2		AWMI	1	2	2	4	2	1
11	275103D	1	1	AWMI	1	1	2	4	22	1
12	720101F	2		IWMI	1	2	2	4	2	1
13	121465B	1	1	NSTEMI	2	1	2	4	2	1
14	720097F	1	3	NSTEMI	2	1	2	4	2	1
15	720143F	1	1	AWMI	1	1	2	4	2	1
16	720130F	1	1	AWMI	1	1	2	4	2	1
17	903164C	1	1	NSTEMI	2	1	2	4	2	1
18	720179F	1	2	AWMI	1	1	1	4	2	1
19	510679B	1	1	AWMI	1	1	2	4	2	1
20	623867F	2		NSTEMI	2	2	2	4	2	1
21	720649F	2		NSTEMI	2	2	2	4	2	1
22	655540F	1 (OUTSIDE)	3	NSTEMI	2	2	2	1	1	1
23	720623F	1	2	AWMI	1	1	2	4	2	1
24	720624F	1	1	IWMI, RVMI	1	1	2	4	2	1
25	294719B	2		NSTEMI	2	2	2	4	2	1
26	720735F	1	2, 4	AWMI	1	1	2	4	2	1
27	720769F	2		IWMI, RVMI, PWMI	1	2	2	4	2	1
28	720731F	1	1	IWMI, PWMI	1	1	2	4	2	1
29	408888D	1	1	AWMI	1	1	2	4	2	1
30	151157D	1	3	IWMI, RVMI, PWMI	1	1	2	4	2	1
31	622344F	1	3	IWMI	1	1	2	4	2	1
32	791763B	2		NSTEMI	2	2	2	4	2	1
33	384654F	1 (OUTSIDE)	1	NSTEMI	2	2	2	4	2	1
34	720801F	1	1	AWMI	1	2	2	4	2	1
35	740095F	2		AWMI	1	2	2	4	2	1
36	740214F	1	1	AWMI	1	1	2	4	2	1
37	726959F	1	1	AWMI	1	1	2	4	2	1
38	726987F	1	1	AWMI	1	1	2	4	2	1
39	740235F	1	2	NSTEMI	2	2	2	4	2	1
40	740247F	1	3	NSTEMI	2	2	2	4	2	1
41	824266C	1	1	LATERAL MI	1	1	2	4	2	1
42	740625F	1	1	IWMI, PWMI	1	1	2	4	2	1
43	893481C	1	1	IWMI	1	1	2	4	2	1
44	740651F	1	2	AWMI	1	1	2	4	2	1
45	743754F	1	1	NSTEMI	2	2	2	4	2	1
46	740757F	1	2	IWMI	1	1	2	4	2	1
47	740761F	1	MINOR CAD	NSTEMI	2	2	2	4	2	1
48	740336F	1	1	IWMI, RVMI, PWMI	1	1	2	4	2	1
49	750456F	2		AWMI	1	2	2	4	2	1

50	750448F	1	3	AWMI	1	1	2	4	2	1
51	750500F	1	1	AWMI	1	1	2	4	2	1
52	744347D	2	2	NSTEMI	2	2	2	4	2	1
53	750411D	1	1	AWMI	1	1	2	4	2	1
54	720775F	1	1	AWMI	1	1	2	4	2	1
55	180276F	2		NSTEMI	2	2	2	4	2	1
56	591988C	2		NSTEMI	2	2	2	4	2	1
57	750175F	2		NSTEMI	2	2	2	4	2	1
58	448582F	1	3	STEMI	1	1	2	4	2	1
59	039957D	1	3	NSTEMI	2	2	2	4	2	1
60	750726F	1	3	NSTEMI	2	2	2	4	2	1
61	332918B	1	3	NSTEMI	2	1	2	4	2	1
62	750255F	2		IWMI	1	2	2	4	2	1
63	751213F	1	3	IWMI,RVMI	1	1	2	4	2	1
64	062059F	2		AWMI	1	2	2	4	2	1
65	751272F	2		NSTEMI	2	2	2	4	2	1
66	570803D	1	1	IWMI	1	1	2	4	2	1
67	984562D	1	3	IWMI	1	1	2	4	2	1
68	750818F	1	3	IWMI	1	2	2	4	2	1
69	751298F	1	2	IWMI, RVMI	1	1	2	4	2	1
70	166379F	1	3	NSTEMI	2	1, CABG	2	4	2	1
71	243871C	1	1	AWMI	1	1	2	4	2	1
72	751467F	1	MINOR CAD	NSTEMI	2	2	2	4	2	1
73	423954C	1	1	NSTEMI	2	1	2	4	2	1
74	209951D	1	2	NSTEMI	2	2	2	4	2	1
75	779922F	1	1	STEMI	1	1	2	4	2	1
76	751534F	1	3	NSTEMI	2	1	2	4	2	1
77	751786F	1	1	AWMI	1	1	2	4	2	1
78	751885F	2		NSTEMI	2	2	2	4	2	1
79	753122F	1	2	AWMI	1	2	2	1	1	1
80	713237F	1	3	NSTEMI	2	2	2	2	2	1
81	753181F	1	1	AWMI	1	1	2	4	2	1
82	709015F	1	3	AWMI	1	2	2	2	2	1
83	753193F	1	1	AWMI	1	1	2	4	2	1
84	753455F	1	1	IWMI	1	1	2	4	2	1
85	753174F	2		NSTEMI	2	2	2	1	1	1
86	520229B	1	3	NSTEMI	2	1 , CABG	2	4	2	1
87	922350F	1	2	NSTEMI	2	1	2	4	2	1
88	922802F	1	4	NSTEMI	2	2	2	4	2	1
89	923163F	1	1	NSTEMI	2	1	2	4	2	1
90	199091D	1	3,4	NSTEMI	2	2	2	4	2	1
91	505223F	2		NSTEMI	2	2	1	5	2	1
92	412556C	2		NSTEMI	2	2	2	4	2	1
93	310290C	1	2	AWMI	1	1	2	4	2	1
94	926377F	1	1	NSTEMI	1	2	2	4	2	1
95	926356F	1	2	IWMI	1	1	2	4	2	1
96	116703F	1	3	AWMI	1	1	2	4	2	1
97	928556F	1	1	NSTEMI	2	1	2	4	2	1
98	928322F	2		NSTEMI	2	2	2	4	2	1
99	928434F	1	3	NSTEMI	2	1	2	4	2	1
100	928609F	1	3,4	NSTEMI	2	2	2	4	2	1
101	929351F	2		NSTEMI	2	2	2	4	2	1

S.N	CMC HOSP.NO	AGE	Dur. (days)	SEX	DT. OF ADMISSION	DT. OF ENROLL	PLACE	CHEST PAIN	TYPE OF CHEST PAIN TYPICAL(1)/ ATYPICAL(2)NA (3)	DIAGNOSIS AT ADMISSION	HTN	DM II	SMk	HYPO THYRO IDISM	DYSLI PIDEMIA	PAST H/O CAD (ACS/CVA/ TIA)
1	662272F	55	17	F	10/11/2013	11/11/2013	MICU	2	3	HYPEROSMOLAR NON KETOTIC STATE, UTI	1	1	2	2	2	2
2	704940F	74	25	M	05/11/2014	06/11/2014	MICU	2	3	LRTI, ARDS, SEPTIC SHOCK	1	1	2	2	2	2
3	713714F	33	18	F	13/11/2013	14/11/2013	MHDU	2	3	RHD, SEVERE MS,MR, AF WITH FVR	2	2	2	2	2	2
4	700136B	80	8	M	25/11/2013	26/11/2013	MICU	2	3	ILD, PNEUMONIA, MYOCARDITIS	2	2	2	2	2	2
5	720640F	60	10	F	24/11/2013	25/11/2013	MICU	2	3	SCRUB TYPHUS, ARDS	1	2	2	2	2	2
6	305236C	77	13	M	22/11/2013	23/11/2013	MICU	2	3	ACUTE ON CHRONIC RENAL FAILURE, UROSEPSIS	1	1	2	2	1	1-CAD
7	726804F	40	20	F	08/12/2013	09/12/2013	MICU	2	3	ACUTE PULMONARY OEDEM ?CAUSE	1	2	2	2	2	2
8	740560F	65	15	F	14/12/2014	14/12/2014	MICU	1	1	? ACS/? PE	1	1	2	2	1	2
9	761706F	67	13	M	09/12/2013	10/12/2013	MICU	1	2	CKD	1	2	2	2	2	1- CAD
10	823549D	32	4	F	24/01/2014	24/01/2014	MICU	2	3	AGE WITH SHOCK	2	2	2	2	2	2
11	721237F	20	17	F	16/12/2013	17/12/2013	MICU	2	3	PNC DAY 1, ?ARDS, ?MYOCARDITIS	2	2	2	2	2	2
12	813874	68	17	F	17/12/2013	18/12/2013	MICU	2	3	CAD-TVD, S/P PPI FOR CHB, ACUTE ON CRF, MORBID OBESITY	2	1	2	2	1	1-CAD
13	740751	27	5	M	17/12/2013	18/12/2013	MHDU	2	3	?MYOCARDITIS, MENINGOENCEPHALITIS	2	2	2	2	2	2
14	089473F	51	5	M	20/12/2013	21/12/2013	MHDU	2	3	DKA, SEPSIS, SEPTIC SHOCK, CHRONIC PANCREATITIS	2	1	1	2	2	2
15	388529F	53	5	M	22/12/2013	23/12/2013	MICU	2	3	CLL WITHFEBRILE EUTROPENIA	2	2	1	2	2	2
16	750078F	65	4	F	23/12/2013	24/12/2013	MICU	2	3	SCRUB TYPHUS, ARDS,SHOCK	2	2	2	2	2	2
17	740702F	67	30	M	16/12/2013	17/12/2013	MHDU	2	3	PNEUMONIA, COPD, LGI BLEED	1	1	1	2	2	2
18	750738F	55	24	F	31/12/2013	31/12/2013	MICU	2	3	DKA, UTI, SEPSIS, O/C IWMI	1	1	2	2	1	1-CAD
19	740939F	26	18	F	01/01/2014	02/01/2014	MICU	2	3	ACUTE PULMONARYOEDEMA, PNC DAY 1	2	2	2	2	2	2
20	949255C	32	30	F	08/01/2014	08/01/2014	MICU	2	3	UROSEPSIS, ARDS, PNC 9 DAYS	2	2	2	2	2	2
21	876069C	54	9	F	10/01/2014	10/01/2014	MICU	2	3	CKD, DKA	2	1	2	1	2	2
22	290246D	55	10	M	10/01/2014	10/01/2014		2	3	CAD, CCF	1	1	2	2	1	1-CAD
23	782649F	72	10	M	12/01/2014	13/01/2014		2	3	CHB	1	2	1	2	2	2
24	751722F	29	30	F	12/01/2014	12/01/2014	MHDU	2	3	SEPSIS, ? MENINGITIS	2	2	2	2	2	2
25	751571F	74	11	M	11/01/2014	12-Jan	MHDU	2	3	SEPSIS, MODS, COPD	2	2	1	2	2	2
26	957810B	46	6	F	14/01/2014	15/01/2014	MICU	2	3	AGE WITH SHOCK, ARF	2	2	2	2	2	2
27	751841F	51	4	M	14/01/2014	15/01/2014	MICU	2	3	SCRUB TYPHUS	2	2	2	2	2	2
28	753144F	74	7	F	19/01/2014	19/01/2014	MICU	2	3	PNEUMONIA, HTN	1	2	2	2	2	2
29	890488C	67	3	F	22/01/2014	23/01/2014		2	3	CAD, CCF	1	1	2	2	1	1-CAD
30	289659C	60	11	M	19/01/2014	20/01/2014	MICU	2	3	COPD, NSTEMI	1	1	1	2	1	2
31	713588F	48	24	M	14/01/2014	15/01/2014	MICU	2	3	ALL, SIRS, SHOCK	2	2	2	2	2	2

32	308044C	74	15	M	20/01/2014	21/01/2014	MHDU	2	3	SEPSIS, SHOCK, PNEUMONIA	1	1	2	2	1	1-CAD
33	1887883D	46	15	M	16/01/2014	17/01/2014	MHDU	2	3	ARF ON CRF, DKA, SEPSIS	2	1	2	2	1	2
34	783729F	49	7	M	22/01/2014	23/01/2014	MHDU	2	3	CCF, THYROTOXICOSIS	2	2	1	2	1	2
35	199939B	77	9	M	22/01/2014	23/01/2014	MICU	2	3	COPD, PNEUMONIA, SHOCK	1	1	1	2	2	1- CAD
36	753753F	75	31	F	27/01/2014	27/01/2014	MHDU	2	3	ACS, PULMONARY OEDEMA, ACIDOSIS-METABOLIC	1	2	2	2	2	2
37	605557B	53	6	M	25/01/2014	26/01/2014		2	3	ACUTE PULMONARY OEDEM ?CAUSE	2	1	2	1	1	2
38	753826F	63	9	M	28/01/2014	29/01/2014	MHDU	2	3	SCRUB TYPHUS, ARDS	1	1	1	2	1	2
39	896263D	63	18	M	27/01/2014	27/01/2014	MICU	2	3	CKD-V, PNEUMONIA	1	1	2	2	1	2
40	785276	69	27	M	16/01/2014	16/01/2014	MICU	2	3	NSTEMI, CCF	1	1	2	2	1	2
41	754589F	54	42	F	08/02/2014	09/02/2014	MICU	2	3	AIDP, RESPIRATORY FAILURE	2	2	2	1	2	2
42	753994F	61	15	F	05/02/2014	06/02/2014	MHDU	2	3	SEPSIS, CARDIAC ARREST, SHOCK	1	1	2	2	2	2
43	784167F	49	52	F	13/02/2014	13/02/2014	MICU	2	3	SEPSIS, SHOCK	1	2	2	2	2	2
44	789657F	59	7	M	29/01/2014	29/01/2014	MHDU	2	3	SEPSIS, ARF	1	1	1	2	1	2
45	759299F	69	11	F	11/03/2014	11/03/2014	MHDU	1	1	NSTEMI, CCF	1	1	2	2	1	2
46	594716D	75	12	M	3/03/201	04/03/2014	MHDU	2	3	PNEUMOIA, SHOCK, COPD	2	1	1	2	1	2
47	813883F	74	9	M	10/03/2014	11/03/2014	MHDU	1	2	NSTEMI, SEPTIC SHOCK	1	2	2	2	1	1- CAD
48	759379F	57	27	M	12/03/2014	13-Mar	MICU	2	3	DCLD, POHTN,CAD	2	1	2	2	2	1-CAD
49	850924D	67	6	M	16/03/2014	16/03/2014	MICU	1	1	CVA, PULMONARY OEDEMA	1	1	1	2	1	1-CVA
50	261226C	68	21	M	15/03/2014	15/03/2014	MICU	2	3	CA LUNG, ?ACS	1	1	1	2	1	2
51	759519F	46	6	F	15/03//2014	15/03/2014	MHDU	2	3	DCMY CARDIOGENIC SHOCK	2	2	2	2	2	2
52	448884F	24	32	F	23/03/2014	24/03/2014	MICU	2	3	SLI, CNS LUPUS, NEPHRITIS	2	2	2	2	2	2
53	901238F	30	7	F	27/03/2014	27/03/2014	MICU	2	3	MORBID OBESITY, OSA	2	2	2	2	2	2
54	875664D	67	10	M	27/03/2014	27/03/2014	MHDU	2	3	CARDIAC ARREST, SEVERE AS, AR	1	2	1	2	2	2
55	759769F	71	9	M	30/03/2014	31/03/2014	MICU	2	3	UROSEPSIS, ANAEMIA	2	1	2	2	2	2
56	901125F	53	9	F	30/03/2014	30/03/2014	MICU	2	3	ACUTE PULMONARY OEDEM ?CAUSE	2	2	2	2	2	2
57	222689B	80	16	F	03/04/2014	03/04/2014	MICU	2	3	CKD, AGE, ARF	1	1	2	2	2	2
58	896168B	67	16	M	30/03/2014	30/03/2014	MHDU	2	3	PULMONARY OEDEMA	1	1	2	2	1	1-CAD
59	829444F	53	4	M	05/04/2014	06/04/2014	MICU	2	3	CARDIAC SRREST, CAD, CKD	1	1	2	2	1	1-CAD
60	901844F	18	6	F	09/04/2014	09/04/2014	MICU	2	3	PARTIAL HANGING	2	2	2	2	2	2
61	901173F	66	18	F	31/03/2014	01/04/2014	MHDU	1	2	CAP, SEPSIS, SHOCK	2	1	1	2	1	2
62	292927	78	8	F	05/04/2014	06/04/2014	MICU	2	3	ILD, ACS	1	1	2	2	1	1-CAD
63	901914F	29	6	M	10/04/2014	10/04/2014	MHDU	2	3	SNAKE BITE, ARF	2	2	2	2	2	2
64	817024F	48	12	F	11/04/2014	11/04/2014	MICU	2	3	MULTIPLE MYELOMA, LRTI	2	2	2	2	2	2
65	989990C	74	8	F	16/04/2014	16/04/2014	MICU	2	3	NSTEMI	2	2	2	2	2	2

66	902088F	75	13	M	13/04/2014	13/04/2014	MICU	2	3	COPD, CAP, AF	1	1	1	2	1	2
67	728679F	50	2	F	18/04/22014	18/04/2014	MHDU	2	3	?AMYLOIDOSIS, CARDIAC ARREST	1	2	2	2	2	2
68	902424F	50	8	M	19/04/2014	19/04/2014	MHDU	2	3	CAP	1	2	1	2	2	2
69	983853D	57	13	F	19/04/2014	20/04/2014	MHDU	2	3	NSTEMI	1	1	2	2	1	2
70	743464D	38	8	M	25/04/2014	25/04/2014	MHDU	2	3	HEMMORRHAGIC STROKE	1	2	2	2	2	2
71	502643F	64	25	M	23/04/2014	24/04/2014	MHDU	2	3	ANAEMIA, GI MALIGNANCY	2	2	1	2	2	2
72	910746F	25	18	F	25/06/2014	25/06/2014	MICU	2	3	PERIPARTUM CARDIOMYOPATHY	1	2	2	2	2	2
73	509840C	63	4	M	03/07/2014	03/07/2014	MICU	1	2	ACUTE PULMONARY OEDEMA	1	1	2	2	1	1
74	858816A	59	4	F	05/07/2014	05/07/2014	MICU	1	2	CKD, DM II HTN, ACUTE PULMONARY OEDEMA	1	1	2	2	1	1-CAD
75	216179D	73	11	F	02/07/2014	03/07/2014	MHDU	2	3	OP POISONING, ASPIRATION PEUMONIA	2	1	2	2	2	2
76	912219F	75	11	M	02/07/2014	02/07/2014	MICU	2	3	ACUTE FEBRILE ILLNESS	2	2	1	2	2	2
77	881568F	56	9	M	08/07/2014	09/07/2014	MHDU	2	3	CVA, MUCORMYCOIS	1	1	2	2	2	2
78	912129F	38	25	M	30/06/2014	30/06/2014	MHDU	2	2	SUPERVASMOL POISONING	2	2	2	2	2	2
79	914052F	21	7	F	16/07/2014	17/07/2014	MHDU	2	3	VIPER BITE	2	2	2	2	2	2
80	657643F	45	29	M	18/07/2014	18/07/2014	MICU	2	3	EPHROPATHY, RESPIRATORY FAILURE	2	2	1	2	2	2
81	914004F	78	16	M	21/07/2014	22/07/2014	MHDU	2	3	DELIRIUM, IGA NEPHROPATHY	2	2	2	2	2	2
82	914041F	64	18	M	17/07/2014	17/07/2014	MHDU	2	3	UTI, UROSEPSIS, CKD	1	1	2	2	1	1-CAD
83	008830G	70	20	F	24/07/2014	24/07/2014	MICU	2	3	PUO, PULMONARY OEDEMA	1	2	2	2	2	2
84	914547F	50	7	F	24/07/2014	24/07/2014	MHDU	2	3	COPD, AKI, DM II	2	1	2	2	2	2
85	914852F	38	7	F	28/07/2014	28/07/2014	MHDU	2	3	SCRUB TYPHUS,ARDS, AKI	2	2	2	2	2	2
86	376245D	48	17	F	01/08/2014	01/08/2014	MICU	1	1	ILD, LUPUS NEPHRITIS	1	2	2	2	2	2
87	915301F	68	7	M	04/08/2014	04/08/2014	MHDU	2	3	HAEMOTOXIC SNAKE BITE, ASV HYPERSENSITIVITY	2	2	2	2	2	2
88	915403F	21	11	M	06/08/2014	06/08/2014	MHDU	2	3	OP POISONING	2	2	2	2	2	2
89	915900F	55	13	M	13/08/2014	14/08/2014	MHDU	1	1	NSTEMI	2	1	1	2	1	1-CAD
90	895558F	65	21	M	09/08/2014	09/08/2014	MICU	2	3	NSTEMI, PULMONARY OEDEMA	1	1	2	2	1	2
91	750969F	61	9	F	15/08/2014	15/08/2014	MHDU	2	3	UTI, SEPSIS	2	1	2	2	2	2
92	260906F	51	14	F	01/09/2014	01/09/2014	MHDU	2	3	CKD, UROSEPSIS	1	1	2	2	1	2
93	913941F	24	17	F	13/08/2014	13/08/2014	MHDU	2	3	POSTPARTUM, PPCM	2	2	2	2	2	2
94	666298D	54	11	F	25/08/2014	25/08/2014	MHDU	2	3	DCMY, LVD, DM II NEPHROPATHY	1	1	2	2	1	2
95	917841F	79	17	M	28/08/2014	28/08/2014	MHDU	2	3	CVA, ASPIRATION PNEUMONIA	1	2	1	2	1	2
96	135775D	67	8	F	17/07/2014	17/07/2014	MHDU	2	3	ACUTE PULMONARY OEDEMA	2	3	2	2	2	2
97	888205A	67	26	M	03/09/2014	03/09/2014	MHDU	2	3	SCRUB TYPHUS, ARDS, VP	2	3	2	2	2	2
98	040852G	42	29	M	06/09/2014	06/09/2014	MICU	2	3	CELLULITIS, SEPSIS,VT, CARDIAC ARREST		1	1	2	2	2
99	511979F	20	7	F	06/09/2014	06/09/2014	MHDU	2	3	PAH, PMND	1	2	2	2	2	2
100	740029F	76	4	F	07/12/2013	08/12/2013	CCU	2	3	NSTEMI	2	2	2	2	1	2

S.N	CMC HOSP.NO	RENAL FAILURE	INTUBATED (CPAP-3)	IF INTUBATED - PEEP	Hb	HCT	TLC	Na	K	S.CREATININE	B.UREA	GRBS AT ADMISSION	CKMB1	CKMB2	TROP T1	CMC HOSP.NO	TROP T2	TROPONIN DELTA HANGE >20%	ECG AT ADMMISSION- NOT S/O ACS (1), ABNORMAL S/O ACS(2)	ECHO- NOT S/O OF ACS(1), ABNORMAL S/O ACS(2)
1	662272F	1	1	8	10.3	31	16300	133	3.2	2.02	94	1237	2.15	1.99	18.17	662272F	23	2	1	1
2	704940F	1	1	8	11.6	35	11500	128	3.8	1.75	42	201	4.86	12.51	172.6	704940F	94.83	1	1	1
3	713714F	1	1	8	13.1	39	21900	113	5.2	2	75	98	10	11	42	713714F	20	1	1	1
4	700136B	2	2	NA	13.1	40	13100	129	4.2	0.79	68	102	3.31	3.42	872.6	700136B	900.3	2	1	1
5	720640F	1	1	10	11.5	35.5	16500	137	3.8	1.46	127	108	21.3	21.46	155	720640F	167.4	2	2	1
6	305236C	1	1	7	6.1	19	7000	138	4	4.33	119	180	2.3	1.86	248.7	305236C	216.1	2	2	2
7	726804F	1	1	11	13.8	41	24300	139	3.3	1.44	33	190	15.31	36.88	229.7	726804F	774.7	1	2	2
8	740560F	2	1	10	13.2	40	14400	142	4.1	0.75	21	188	2.61	9.6	22.84	740560F	401	1	1	2
9	761706F	1	1	8	6.2	17.8	12500	122	5.9	16.92	142	210	8.82	12.16	291.7	761706F	1273	1	2	2
10	823549D	1	1	6	11	33	12000	130	3	1.66		148	1.99	13.1	8.97	823549D	121.9	1	2	2
11	721237F	2	3	8	9.7	27.3	6300	131	4.7	0.75	19	108	7.52	7.78	32.73	721237F	61.88	1	1	1
12	813874	1	1	10	9	27	11700	150	4.3	2	57	202	5.24	7.48	84.27	813874	69.44	2	1	1
13	740751	2	1	7	14.5	42	8000	131	4.1	0.8	38	118	18	22.83	600	740751	900.7	1	2	2
14	089473F	2	1	8	10.7	33	12600	164	3.6	1.21	58	176	1.26	10.63	46.95	089473F	206.1	1	2	2
15	388529F	2	2	NA	6.4	19	30500	125	4	1.19	20	90	0.859	3.29	15.68	388529F	43.68	1	2	2
16	750078F	1	1	10	12.5	35.7	5300	128	3.7	2.14	116	136	4.75	6	39.53	750078F	70	1	2	2
17	740702F	1	1	10	9.3	28.1	23400	139	4	2.32	56	166	1.93	1.98	75.89	740702F	72.26	2	1	1
18	750738F	1	2	NA	17.2	39	11000	122	3.1	1.94	34	455	54.18	252	72.33	750738F	876.1	1	2	2
19	740939F	1	1	6	8.9	28	24000	131	4.5	6.9	62	110	2.24	5	91.27	740939F	200	1	2	2
20	949255C	2	2	NA	10.9	31	15200	141	3.8	0.84	22	80	2.88	1.91	26.5	949255C	17.94	1	1	1
21	876069C	1	2	NA	8.5	25.4	6300	124	4.6	2.75	71	600	15.3	17.73	291	876069C	353	1	2	2
22	290246D	1	2	NA	12	34.7	12200	131	5	1.79	49	221	4.84	5	40.42	290246D	51.81	1	2	2
23	782649F	1	2	NA	12.8	38.2	13700	135	3.3	1.53	27	112	2.95	3.67	40.89	782649F	55.1	1	1	1
24	751722F	1	1	9	10.5	31.9	5500	134	2.8	1	16	68	5.26	7.09	81.55	751722F	74.34	2	1	1
25	751571F	2	1	8	11.7	36.8	57200	113	2.7	0.66	20	221	8	10.83	54.84	751571F	37.09	1	1	1
26	957810B	1	2	NA	10.3	30.1	6400	138	3.3	1.78	64	112	4.54	4.78	50.01	957810B	107.5	1	2	2
27	751841F	2	2	NA	14.4	39.9	13400	126	3.8	1.32	50	102	4.1	3.44	25.65	751841F	28.65	2	1	1
28	753144F	1	3	8	12.4	38.1	24600	135	3.3	1.48	31	132	6.48	6.84	75.91	753144F	81.42	2	1	1
29	890488C	1	2	NA	10.7	31.4	11900	135	5.5	1.74	35	144	3.39	3.66	42.36	890488C	43.73	2	1	1
30	289659C	1	1	8	11.4	33.6	29400	127	4.3	1.86	74	301	8	12.55	150.2	289659C	435	1	2	2
31	713588F	2	1	8	8.9	26.2	1800	116	4	0.75		112	6	6.56	22.33	713588F	23.58	2	1	1

32	308044C	1	1	7	12.2	37	7700	133	4.8	2.53	55	132	14.48	11.96	47.48	308044C	96.11	1	2	2
33	1887883D	1	1	8	8.4	26.4	11200	138	3.4	2.72	102	588	12.41	13.52	486.8	1887883D	544.9	2	2	2
34	783729F	2	2	NA	13.2	43.4	13700	142	4.6	0.69	20	112	2.61	3.6	11.02	783729F	49.56	1	1	1
35	199939B	2	2	NA	13.6	40	29700	116	3.9	1.18	22	201	27.83	17.56	66.55	199939B	48.5	1	1	1
36	753753F	2	1	9	12.6	43	14500	128	3.8	1.31	54	146	5.72	13.37	299.5	753753F	545	1	2	2
37	605557B	2	2	NA	14.8	43.6	13700	141	3.2	0.61	20	221	22.43	20.32	679.8	605557B	755.4	1	2	2
38	753826F	2	1	7	12.9	38.7	10400	121	2.7	1.03	37	144	4.85	3.96	71.6	753826F	90.97	1	1	1
39	896263D	1	1	10	10.3	30.8	31500	136	3.4	3.05	109	309	10.46	24.13	38.17	896263D	59.33	1	1	1
40	785276	1	1	7	12.7	36.2	10800	126	5.4	1.68	45	289	2.37	4.57	20.93	785276	24.8	2	2	2
41	754589F	2	1	8	13.6	40.2	21600	127	3.5	0.61	47	112	6.73	8.7	89.01	754589F	71.67	2	1	1
42	753994F	1	1	9	11.4	33	9600	137	35	2.6	33	110	1.26	1.83	57.57	753994F	53.78	2	1	1
43	784167F	1	1	7	9.1	27	24600	139	3.4	1.68	30	189	6.52	14.77	384.7	784167F	203.7	1	1	1
44	789657F	1	1	9	10.9	31	17200	132	6	3.12	188	301	1.88	2.31	35.64	789657F	49.5	1	1	1
45	759299F	2	1	8	10	29.8	30800	130	4.6	0.83	42	205	5.33	4.95	18.41	759299F	146.7	1	2	2
46	594716D	1	1	9	14.3	31.7	18600	136	4.4	1.57	65	130	34.82	29.88	709.2	594716D	2989	1	2	2
47	813883F	1	1	10	11.2	33.4	5300	151	3.3	1.83	51	112	4.13	214	14.77	813883F	2520	1	2	2
48	759379F	1	1	8	6.5	19.3	9400	116	4.2	0.82	35	98	3.8	2.89	28.87	759379F	26.8	2	1	1
49	850924D	1	1	8	12	36	17700	136	3.9	3.46	59	201	4.09	8.21	76.24	850924D	202	1	2	2
50	261226C	2	1	7	10.8	31.6	15400	130	4	0.91	64	201	8.33	4.81	94.69	261226C	216.2	1	2	2
51	759519F	2	1	10	9.6	29.5	7600	131	3	0.95	29	128	1.85	1.98	19.56	759519F	21.45	2	1	1
52	448884F	1	1	9	7.1	20.4	5800	134	3.4	5.25	194	78	2.14	2.12	229.2	448884F	192	2	1	1
53	901238F	1	1	10	12.6	38.9	18300	131	4.7	1.81	146	192	2.82	2.14	21.94	901238F	24.15	2	1	1
54	875664D	1	1	8	10.8	33.1	15600	136	5.4	2.22	53	200	6.66	16.67	71.18	875664D	164.1	1	2	2
55	759769F	1	3	7	5.7	16.4	5400	142	3	2.8	88	190	3.79	6.09	58.16	759769F	<3	1	1	1
56	901125F	2	1	7	13.2	43.4	42400	137	3.8	1.08	42	112	5.66	16.04	42.55	901125F	73.7	1	1	1
57	222689B	1	2		9.4	28	10200	136	4.2	2.57	69	255	21.79	36.13	103.4	222689B	578.6	1	2	2
58	896168B	1	1	8	11.8	35.6	3300	125	4.3	1.49	43	280	1.63	1.34	17	896168B	15	2	1	1
59	829444F	1	1	8	8.3	24.8	28000	135	4.8	6.66	81	180	15.65	4.07	2062	829444F	1474	1	1	1
60	901844F	2	1	7	15.7	46.1	15700	132	3	0.96	22	121	6.94	10.24	360	901844F	284.1	1	1	1
61	901173F	1	1	8	12.8	38.2	6200	138	4	1.62	45	200	6.04	4.64	12.89	901173F	16.18	2	1	1
62	292927	1	1	6	10.7	33	2800	128	3.3	1.42	25	198	1.85	3.28	15.63	292927	32.42	1	1	1
63	901914F	1	1	7	10.4	30.5	16900	140	4.3	1.48	83	122	8.98	11.85	67.96	901914F	98.53	1	1	1
64	817024F	2	2		8.1	25	2100	132	2.6	0.49	20	132	4.96	10.85	272	817024F	671.6	1	2	2
65	989990C	2	2		12.4	36.9	9400	126	3.5	0.97	27	138	3.66	6.17	101.8	989990C	177	1	2	2

66	902088F	1	1	8	11.7	33.3	6800	135	4.1	3.5	108	142	1.74	1.56	38.22	902088F	27.94	1	1	1
67	728679F	2	1	12	11.8	35.8	13800	135	3.4	0.44	13	188	11.61	10.8	122	728679F	306.6	1	2	2
68	902424F	2	3	9	14	47	12700	121	4	1.03	34	130	6.31	7.76	31.78	902424F	20.23	1	1	1
69	983853D	2	1	7	13.1	37.2	17500	135	3.7	0.83	30	148	3.1	2.58	16.46	983853D	124.4	1	2	2
70	743464D	1	1	6	18	52.6	19900	133	3.8	1.92	29	146	31.31	21.7	423.8	743464D	552.6	1	2	2
71	502643F	1	1	8	6.1	17.8	23700	133	4.5	2.15	53	110	2	1.19	67	502643F	60	2	1	1
72	910746F	1	1	8	13.8	42.9	21350	146	4	1.55	101	188	23.1	15.54	7879	910746F	6549	2	1	1
73	509840C	1	1	8	13	39	17300	135	3.9	1.49	37	201	1.94	2.33	30.77	509840C	43.93	1	2	2
74	858816A	1	1	10	11	33	31780	125	5	2.73	68	190	35.98	35.59	7302	858816A	7431	2	2	2
75	216179D	1	1	7	17.7	53.1	22000	142	2.7	1.73	71	130	20.31	16.71	14.48	216179D	13.7	2	1	1
76	912219F	1	1	8	12.5	37.2	8400	140	4.7	4.52	184	200	36.51	23.47	20.24	912219F	17.61	2	1	1
77	881568F	2	1	8	8	24	9200	140	3.8	1.2	27	110	4.34	7	47.85	881568F	80	1	2	2
78	912129F	1	1	8	13.3	39	18050	141	3.5	4	26	176	27.78	14.69	360	912129F	1559	1	1	1
79	914052F	2	2		10.1	30.8	24900	147	3.1	1.18	62	118	45.43	19.27	651.3	914052F	503.6	2	1	1
80	657643F	1	1	7	10.6	32	23400	136	4.5	3.83	50	180	2.84	3.09	116	657643F	128.4	2	2	2
81	914004F	2	3	8	8.4	25	19000	137	3.9	0.71	55	112	2.09	2.62	1243	914004F	866.6	1	2	2
82	914041F	1	1	7	7.5	22	23500	137	5.2	5.63	194	190	2.67	2	414.5	914041F	416.7	2	2	2
83	008830G	2	1	8	11.3	33.6	33800	132	3.9	0.85	20	118	2.82	8.8	206.8	008830G	317	1	2	2
84	914547F	2	1	5	11.3	37.6	24400	131	4.6	1.28	36	110	2.39	2.09	21.94	914547F	26.6	2	1	1
85	914852F	1	1	8	9.8	28.7	15700	138	3.5	1.33	71	108	2.92	3.12	85.33	914852F	35.52	1	1	1
86	376245D	1	1	12	7.3	21.9	13000	128	5.9	2.59	81	178	1.38	4.11	32.95	376245D	336.8	1	2	2
87	915301F	2	1	8	13.8	40.2	15400	139	3.8	1.04	42	122	17.86	14.14	917.8	915301F	406.3	1	1	1
88	915403F	2	1	10	16.5	46	26600	138	3.9	1.08	22	102	12.53	12.01	251	915403F	209.8	2	1	1
89	915900F	1	2		14.5	46	18100	128	4.8	1.92	51	202	2.1	>300	37.17	915900F	3641	1	2	2
90	895558F	1	1	8	10.5	31	20100	129	3.6	1.71	30	306	2.42	39.26	57	895558F	700	1	2	2
91	750969F	1	2		8.5	25.4	9700	135	4	2.05	78	112	3.13	12.77	125	750969F	180	1	2	2
92	260906F	1	1	10	7.9	23.3	16800	133	4.3	4.75	108	174	0.709	0.832	66.99	260906F	59.9	2	1	1
93	913941F	2	2		9.2	27.5	12500	130	4.5	0.9	30	102	4.19	2.89	26.26	913941F	15.05	1	1	1
94	666298D	1	1	12	14.3	42.6	6900	113	5.1	1.75	77	98	5.7	5.07	219	666298D	182	2	1	1
95	917841F	2	1	10	10.6	31	9200	134	3.4	1.35	55	152	10.16	8	16.65	917841F	89	1	2	2
96	135775D	1	2		6.5	19	13390	142	6.6	1.54	55	124	4.85	3.74	114.4	135775D	74.13	1	1	1
97	888205A	1	1	8	9	28.6	15700	131	5	2.18	85	98	21.39	19.97	48.48	888205A	55.91	2	1	1
98	040852G	1	1	7	13.5	40.2	6100	132	4.9	1.5	28	420	4.11	2.3	110.7	040852G	106.2	2	1	1
99	511979F	2	1	4	18.4	61.2	8700	134	3.9	0.58	28	131	6.09	4.64	28.23	511979F	25.62	2	1	1
100	740029F	2	2		12.8	39	14300	133	3.6	0.59	24	223	11.04	14.91	585.8	740029F	735.2	1	2	2

S.N	CMC HOSP.NO	APACHE II SCORE	Apache II score-12 TO 17=1, 18 T0 24=2, ≥25=3	CAG	FINAL DIAGNOSIS	URGENT REVASCULARISATION YES (1)/NO(2)	INHOSPITAL- (1)-MORTALITY , (2) MYOCARDIAL INFARCTION, (A)-STEMI, (B) NSTEMI, (3) NONE	OUTCOME ≤30 DAYS AFTER DISCHARGE-(1)-MORTALITY, (2)-REHOSPITALISATION, (3)-REINFARCTION, (4)- NONE, (5) INHOSPITAL DEATH	≤30 DAY MORTALITY	≤30 DAY REHOSPITALISATION	
1	662272F	24	2	2	UTI, NOSOCOMIAL PNEUMONIA, HYPEROSMOLAR ON KETOTIC STATE	2	3	4	2	2	
2	704940F	34	3	2	NOSOCOMIAL PNEUMONIA, H3N1 INFECTION, TYPE I RESPIRATORY	2	1	5	2	2	
3	713714F	21	2	2	RHD, SEVERE MS, MR,AF WITH FVR, LRTI	2	3	4	2	2	
4	700136B	17	1	2	ARDS SECONDARY TO VIRAL MYOCARDITIS, ILD	2	1	5	2	2	
5	720640F	28	3	2	ARDS, SCRUB TYPHUS,HTN	2	3	4	2	2	
6	305236C	29	3	2	CKD, MRSA SEPTICEMIA, MODS, DIC	2	1	5	2	2	
7	726804F	35	3	2	ACS-NSTEMI, HTN, CARDIAC ARREST	2	2B	4	2	2	
8	740560F	21	2	2	NSTEMI, CARDIOGENIC SHOCK,HIE	2	1	5	2	2	
9	761706F	12	1	2	CKD STAGE V, PNEUMONIA, STEMI	2	2B	4	2	2	
10	823549D	18	2	2	ACS-NSTEMI, CMC MYOCARDITIS, PNEUMONIA, HIV	2	2B	1	1	2	
11	721237F	12	1	2	MYOCARDITIS, ARDS	2	3	4	2	2	
12	813874	36	2	2	SEPSIS, UTI	2	3	4	2	2	
13	740751	23	2	2	ACS-NSTEMI,TB MENINGITIS, CVA,	2	1,2B	5	2	2	
14	089473F	29	3	2	NSTEMI	2	1, 2B	5	2	2	
15	388529F	27	3	2	PNEUMONIA, CLL, SEPSIS, SHOCK	2	2B	1	1	2	
16	750078F	28	3	2	ACS-NSTEMI,SCRUB TYPHUS	2	1,2B	5	2	2	
17	740702F	27	3	2	AF,COPD, PNEUMONIA	2	1	5	2	2	
18	750738F	25	3	2	NSTEMI, SEPSIS, SHOCK, AKI	2	2B	4	2	2	
19	740939F	33	3	2	ACS-NSTEMI,ARF, RENAL CORTICAL NECROSIS	2	2B	4	2	2	
20	949255C	12	1	2	ARDS, UROSEPSIS	2	3	4	2	2	
21	876069C	23	2	2	CKD, DKA, CCF	2	2B	4	2	2	
22	290246D	23	2	2	CAD, CCF	2	3	4	2	2	
23	782649F	18	2	2	CHB	2	3	4	2	2	
24	751722F	17	1	2	CNS VASCULITIS, SEPSIS, ARDS, MYOCARDITIS	2	1	5	2	2	
25	751571F	35	3	2	SEPSIS, SHOCK	2	1	5	2	2	
26	957810B	18	2	2	SHOCK, ARF	2	2B	4	2	2	
27	751841F	15	1	2	SHOCK	2	3	4	2	2	
28	753144F	17	1	2	SHOCK, ARDS	2	3	4	2	2	
29	890488C	23	2	2	CCF, CAD	2	3	4	2	2	
30	289659C	30	3	2	COPD, NSTEMI	2	2B	4	2	2	
31	713588F	23	2	2	SEPSIS, SHOCK	2	3	1	1	2	

32	308044C	29	3	2	SEPSIS, SHOCK, PNEUMONIA	2	2B	4	2	2	
33	1887883D	35	3	2	UTI, SEPSIS, AFR ON CRF	2	2B	4	2	2	
34	783729F	12	1	2	HYPERTHYROIDISM, LVD	2	3	2	2	1	
35	199939B	24	2	2	ARDS, PNEUMONIA, SEPSIS, SHOCK	2	1	1	1	2	
36	753753F	22	2	2	NSTEMI, HF	2	2B	4	2	2	
37	605557B	12	1	2	NSTEMI, CCF, PAPILLARY CARCINOMA THYROID	2	2B	2	2	1	
38	753826F	16	1	2	SCRUB TYPHUS, LVD	2	3	4	2	2	
39	896263D	37	3	2	CKD, SEPSIS, SHOCK,PNEUMONIA	2	1	5	2	2	
40	785276	30	3	2	NSTEMI,SEPSIS,SHOCK	2	1	5	2	2	
41	754589F	15	1	2	AIDP	2	3	4	2	2	
42	753994F	30	3	2	CPR, SEPSIS, SHOCK	2	3	4	2	2	
43	784167F	23	2	2	CVA, SEPSIS	2	3	4	2	2	
44	789657F	32	3	2	SEPSIS, SHOCK,ARF	2	1	5	2	2	
45	759299F	19	2	2	NSTEMI, CAP	2	1, 2B	5	2	2	
46	594716D	28	3	2	NSTEMI	2	2B	1	1	2	
47	813883F	34	3	2	STEMI, ARF	2	2A	4	2	2	
48	759379F	30	3	2	DCLD, POHT, SEPSIS, ARF	2	1	5	2	2	
49	850924D	33	3	2	CKD, NSTEMI	2	2B	4	2	2	
50	261226C	29	3	2	NSTEMI, VAP, SEPSIS	2	2B, 1	5	2	2	
51	759519F	21	2	2	DCMY, CARDIOGENIC SHOCK	2	1	5	2	2	
52	448884F	28	3	2	MYOCARDITIS, LUPUS NEPHRITIS, CNS LUPUS	2	1	5	2	2	
53	901238F	17	1	2	OSA, SHOCK	2	1	5	2	2	
54	875664D	30	3	2	NSTEMI	2	2B	1	1	2	
55	759769F	35	3	2	SEPSIS, ANAEMIA	2	3	2	2	1	
56	901125F	12	1	2	RHD, SEVERE MS,PAH, LRTI	2	3	4	2	2	
57	222689B	30	3	2	NSTEMI	2	2B	4	2	2	
58	896168B	18	2	2	PNEUMONIA, VAP, GTCS	2	1	5	2	2	
59	829444F	35	3	2	CPR, CKD	2	1	5	2	2	
60	901844F	14	1	2	MYOCARDIAL STUNNING, PARTIAL HANGING	2	3	4	2	2	
61	901173F	25	3	2	CAP, ARDS	2	3	4	2	2	
62	292927	26	3	2	VAP, SEPSIS, SHOCK, ARDS	2	1	5	2	2	
63	901914F	20	2	2	VIPER BITE	2	3	4	2	2	
64	817024F	23	2	2	NSTEMI	2	2B	4	2	2	
65	989990C	16	1	2	NSTEMI	2	2B	4	2	2	

66	902088F	28	3	2	AF, PNEUMONIA, COPD	2	3	4	2	2	
67	728679F	30	3	2	NSTEMI, CARDIOGENIC SHOCK	2	1	5	2	2	
68	902424F	22	2	2	HIV, PNEUMONIA	2	3	4	2	2	
69	983853D	26	3	1	NTTE, PNEUMONIA	2	3	1,2	1	1	
70	743464D	23	2	2	PONTINE HEMORRHAGE	2	1, 2B	5	2	2	
71	502643F	27	3	2	PROBABLE GI MALIGNANCY	2	3	4	2	2	
72	910746F	18	2	2	PERIPARTUM CARDIOMYOPATHY	2	1	5	2	2	
73	509840C	12	1	1	NSTEMI	2	2B	2	2	1	
74	858816A	34	3	2	NSTEMI	2	1, 2B	5	2	2	
75	216179D	30	3	2	ASPIRATION PNUMONIA	2	3	1	1	2	
76	912219F	30	3	2	ARF, ARDS, SEPSIS	2	1	5	2	2	
77	881568F	19	2	2	NSTEMI,CAVERNOUS SINUS THROMBOSIS	2	1,2B	5	2	2	
78	912129F	12	1	2	RHABDOMYLYSIS, ARF	2	3	4	2	2	
79	914052F	12	1	2	VIPER BITE	2	3	4	2	2	
80	657643F	21	2	2	CKD, NSTEMI	2	2B	4	2	2	
81	914004F	20	2	2	NSTEMI,UROSEPSIS, DELIRIUM	2	2B	4	2	2	
82	914041F	31	3	2	NSTEMI,UROSEPSIS, ARF	2	2B	1	1	2	
83	008830G	16	1		NSTEMI	2	2B	4	2	2	
84	914547F	15	1	2	COPD, PNEUMONIA	2	3	4	2	2	
85	914852F	18	2	2	SCRUB TYPHUS, ARDS, AKI	2	3	4	2	2	
86	376245D	35	3	2	NSTEMI	2	2B	4	2	2	
87	915301F	12	1	2	MYOCARDITIS, HAEMOTOXIC SNAKE BITE	2	3	4	2	2	
88	915403F	23	2	2	OP POISONING, MYOCARDITUS	2	3	4	2	2	
89	915900F	21	2	2	NSTEMI	2	2B	4	2	2	
90	895558F	28	3	2	NSTEMI	2	2B	4	2	2	
91	750969F	26	3	2	NSTEMI	2	2B	4	2	2	
92	260906F	24	2	2	PYELONEPHRITIS, DM II	2	3	4	2	2	
93	913941F	12	1	2	PPCM	2	3	4	2	2	
94	666298D	29	3	2	DCMY, CKD	2	3	4	2	2	
95	917841F	36	3	2	NSTEMI,CVA, AF, ASPIRATION PNEUMONIA	2	1, 2B	5	2	2	
96	135775D	27	3	2	ANAEMIA, CCF	2	3	4	2	2	
97	888205A	32	3	2	SRUB TYPHUS, ARDS, VAP	2	3	4	2	2	
98	040852G	16	1	2	CELLULITIS, SEPSIS, CPR	2	3	4	2	2	
99	511979F	27	3	2	ALS, PAH, RESPIRATORY FAILURE, POLYCYTHEMIA	2	3	2	2	2	
100	740029F	20	2	1	NSTEMI	2	2B	4	2	2	

S.N	CMC HOSP.NO	TOTAL MORTALITY (IN-HOSPITAL AND ≤30DAYS MORTALITY)	THROMBOTIC (1) /NONTHROMBOTIC (2)	STEMI (1) NSTEMI(2), NONE (3)	CAUSE OF NON-THROMBOTIC TROPONIN ELEVATION	CPR	PNEUMONIA	SEPSIS	TACHYARRHYTHMIA	BRADYARRHYTHMIA	MYOCARDITIS	ARDS	DKA	SHOCK	ARF	CKD	COPD	CHF	AIDP	CVA	ANEMIA	RHD	GTCS	SNAKE BITE
1	662272F	2	2	3	SEPSIS, PNEUMONIA, KETOSIS	2	1	1	2	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2
2	704940F	1	2	3	PNEUMONIA	2	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
3	713714F	2	2	3	PNEUMONIA, TACHYARRHYTHMIA, SHOCK,SEPSIS	2	1	1	1	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2
4	700136B	1	2	3	MYOCARDITIS, ARDS	2	2	1	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2
5	720640F	2	2	3	ARDS, SEPSIS, SHOCK	2	2	1	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	2
6	305236C	1	2	3	SEPSIS, ACUTE ON CKD	2	2	2	2	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2
7	726804F	2	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
8	740560F	1	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
9	761706F	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
10	823549D	1	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
11	721237F	2	2	3	MYOCARDITIS, ARDS	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	1	2	2	2
12	813874	2	2	3	ARF, SHOCK,	2	2	2	2	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2
13	740751	1	1	2	NA	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
14	089473F	1	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
15	388529F	1	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
16	750078F	1	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
17	740702F	1	2	3	AF, COPD, PNEUMONIA, SHOCK	2	2	1	1	2	2	2	2	1	1	2	1	2	2	2	1	2	2	2
18	750738F	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
19	740939F	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
20	949255C	2	2	3	URROSEPSIS, ARDS	2	2	1	2	2	2	1	2	2	2	2	2	2	2	2	1	2	2	2
21	876069C	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
22	290246D	2	2	3	HF	2	2	1	2	2	2	2	2	2	1		2	1	2	2	2	2	2	2
23	782649F	2	2	3	CHB	2	2	1	2	1	2	2	2	2	1	2	2	2	2	2	2	2	2	2
24	751722F	1	2	3	SEPSIS, ARDS, MYOCARDITIS	2	2	1	2	2	1	1	2	2	1	2	2	2	2	2	1	2	2	2
25	751571F	1	2	3	SEPSIS, SHOCK	2	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2
26	957810B	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
27	751841F	2	2	3	SHOCK	2	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2
28	753144F	2	2	3	SHOCK, ARDS	2	2	1	2	2	2	1	2	1	1	2	2	2	2	2	2	2	2	2
29	890488C	2	2	3	HF	2	2	2	2	2	2	2	2	2	1	2	2	1	2	2	2	2	2	2
30	289659C	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
31	713588F	1	2	3	SEPSIS, SHOCK	2	2	1	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2

32	308044C	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
33	1887883D	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
34	783729F	2	2	3	HYPERTHYROIDISM, HF	2	2	1	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2
35	199939B	1	1	3	ARDS, PNEUMONIA, SEPSIS, SHOCK	2	1	1	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	2
36	753753F	2	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
37	605557B	2	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
38	753826F	2	2	3	NA	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
39	896263D	1	2	3	CKD, SEPSIS, PNEUMONIA, SHOCK	2	1	1	2	2	2	2	2	1	1	1	2	2	2	2	1	2	2	2
40	785276	1	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
41	754589F	2	2	3	AIDP	2	2	1	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2
42	753994F	2	2	3	SEPSIS, SHOCK, CPR	1	2	1	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2
43	784167F	2	2	3	SEPSIS, CVA, ARF	2	2	1	2	2	2	2	2	2	1	2	2	2	2	1	1	2	2	2
44	789657F	1	2	3	SEPSIS, SHOCK, ARF	2	2	1	2	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2
45	759299F	1	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
46	594716D	1	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
47	813883F	2	1	1	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
48	759379F	1	2	3	DCLD, SEPSIS, SHOCK, ARF	2	2	1	2	2	2	2	2	1	1	2	2	2	2	2	1	2	2	2
49	850924D	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
50	261226C	1	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
51	759519F	1	2	3	CARDIOGENIC SHOCK	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2
52	448884F	1	2	3	ARF, MYOCARDITIS	2	2	2	2	2	1	2	2	2	1	2	2	2	2	2	1	2	2	2
53	901238F	1	2	3	SHOCK, SEPSIS, ARF	2	2	1	2	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2
54	875664D	1	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
55	759769F	2	2	3	ANAEMIA, ARF, SEPSIS	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
56	901125F	2	2	3	SEVERE MS, PAH, PNEUMONIA	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2
57	222689B	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
58	896168B	1	2	3	SEPSIS, PNEUMONIA, GTCS	2	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	1	2
59	829444F	1	2	3	CPR, CKD	1	2	1	2	2	2	2	2	2	1	1	2	2	2	2	1	2	2	2
60	901844F	2	2	3	MYOCARDIAL STUNNING	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
61	901173F	2	2	3	ARDS, ARF, PNEUMONIA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
62	292927	1	2	3	ARDS, SEPSIS, SHOCK, VAP	2	1	1	2	2	2	1	2	1	1	2	2	2	2	2	1	2	2	2
63	901914F	2	2	3	MYOCARDITIS, ARF	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
64	817024F	2	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
65	989990C	2	1	2	NA	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2

66	902088F	2	2	3	AF, COPD, PNEUMONIA	2	1	2	1	2	2	2	2	2	1	2	1	2	2	2	2	2	2	2
67	728679F	1	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
68	902424F	2	2	3	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
69	983853D	1	2	3	PNEUMONIA,ARDS, SEPSIS,SHOCK	2	1	1	2	2	2	1	2	1	2	2	2	2	2	2	2	2	2	2
70	743464D	1	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
71	502643F	2	2	3	ANAEMIA, ARF, SEPSIS	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
72	910746F	1	2	3	PERIPARTUM CARDIOMYOPATHY	2	2	1	2	2	1	2	2	2	1	2	2	1	2	2	2	2	2	2
73	509840C	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
74	858816A	1	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
75	216179D	1	2	3	PNEUMONIA	2	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
76	912219F	1	2	3	ARF, ARDS, SEPSIS	2	2	1	2	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2
77	881568F	1	1	2	NA	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
78	912129F	2	2	3	ARF	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
79	914052F	2	2	3	HEMOTOXIC SNAKE BITE	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	1
80	657643F	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
81	914004F	2	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
82	914041F	1	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
83	008830G	2	1	2	NA	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
84	914547F	2	2	3	PNEUMONIA, SEPSIS	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
85	914852F	2	2	3	ARDS, AKI	2	1	1	2	2	2	1	2	2	1	2	2	2	2	2	1	2	2	2
86	376245D	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
87	915301F	2	2	3	MYOCARDITIS, HAEMOTOXIC SNAKE BITE	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
88	915403F	2	2	3	OP POISONING, CARDIAC ARREST, MYOCARDITIS	1	2	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2
89	915900F	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
90	895558F	2	1	2	NA	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
91	750969F	2	1	2	NA	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
92	260906F	2	2	3	PYELONEPHRITIS, UROSEPSIS, CKD	2	1	1	2	2	2	2	2	2	1	1	2	2	2	2	1	2	2	2
93	913941F	2	2	3	PPCM	2	2	1	2	2	1	2	2	2	2	2	2	1	2	2	1	2	2	2
94	666298D	2	2	3	DCMY, CKD	2	2	2	2	2	2	2	2	2	1	1	2	1	2	2	2	2	2	2
95	917841F	1	1	2	NA	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2
96	135775D	2	2	3	ANAEMIA,ARF	2	2	1	2	2	2	2	2	2	1	2	2	2	2	2	1	2	2	2
97	888205A	2	2	3	SCRUB TYPHUS, ARDS, VAP	2	1	1	2	2	2	1	2	2	1	2	2	2	2	2	1	2	2	2
98	040852G	2	2	3	CPR, VT, ARF	1	2	2	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2	2
99	511979F	2	2	3	ALS, PAH	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2	2	2
100	740029F	2	1	2	NA	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2