

**STUDY OF SERUM SODIUM LEVEL AND ITS
PROGNOSTIC SIGNIFICANCE IN PATIENTS WITH
ACUTE STEMI IN TERTIARY CARE CENTER**

A Dissertation Submitted to

**THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY
CHENNAI**

In Partial Fulfilment of the Regulations

For the Award of the Degree of

M.D. (GENERAL MEDICINE) - BRANCH – I

REG. NO: 200120102502



HANJAVUR MEDICAL COLLEGE AND HOSPITAL,

THANJAVUR-613004

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***“A STUDY OF SERUM SODIUM LEVEL AND ITS PROGNOSTIC
SIGNIFICANCE IN PATIENTS WITH
ACUTE STEMI IN TERTIARY CARE CENTER”***

IS THE BONAFIDE ORIGINAL WORK DONE BY **DR CAUVERY N**, IN PARTIAL
FULFILMENT OF THE UNIVERSITY REGULATIONS OF THE TAMIL NADU DR.
M.G.R. MEDICAL UNIVERSITY, CHENNAI, FOR **M.D BRANCH - GENERAL
MEDICINE** EXAMINATION OF THE TAMILNADU DR M.G.R MEDICAL
UNIVERSITY TO BE HELD IN **MAY 2023**.

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M.G.R. MEDICAL UNIVERSITY, CHENNAI, FOR **M.D GENERAL MEDICINE** TO
BE HELD IN **MAY 2023**.

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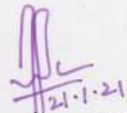
CERTIFICATE No: 848/2021

TITLE OF THE STUDY:
**A study of serum sodium level and its prognostic significance
in patients with acute STEMI in tertiary care center.**

PRINCIPAL INVESTIGATOR:
**Dr. N. Cauvery, Postgraduate in Department of General
Medicine, Thanjavur Medical College.**

This to certify that the protocol submitted by the principal
investigator of the above mentioned study has been reviewed as
per standard ethical guidelines and the same has been
APPROVED by the members of the Institutional ethical
committee at its meeting held on **21.01.2021**.




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DECLARATION

I, DR.CAUVERY N, SOLEMNLY DECLARE THAT THIS DISSERTATION TITLED "*A STUDY OF SERUM SODIUM LEVEL AND ITS PROGNOSTIC SIGNIFICANCE IN PATIENTS WITH ACUTE STEMI IN TERTIARY CARE CENTER*" IS A BONAFIDE RECORD OF WORK DONE BY ME AT THE DEPARTMENT OF GENERAL MEDICINE, THANJAVUR MEDICAL COLLEGE AND HOSPITAL, UNDER THE GUIDANCE OF **PROF.DR.S.VETRIVEL, M.D.** UNIT CHIEF, DEPARTMENT OF GENERAL MEDICINE, THANJAVUR MEDICAL COLLEGE AND HOSPITAL, THANJAVUR.

THIS DISSERTATION IS SUBMITTED TO THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI IN PARTIAL FULFILLMENT OF THE RULES AND REGULATIONS FOR THE AWARD OF **M.D DEGREE GENERAL MEDICINE** EXAMINATION TO BE HELD IN **MAY 2023**.

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Document Information

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Submitted by	CAUVERY N
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Similarity	16%
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Sources included in the report

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SA	HYPONATREMIA IN STEMI.docx Document HYPONATREMIA IN STEMI.docx (D123637752)	 4
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ACKNOWLEDGEMENT

I would like to thank **DR.R. BALAJI NATHAN.**, Dean, Thanjavur medical college and hospital, for permitting me to utilize the facilities of Thanjavur Medical College and Hospital facilities for this dissertation.

I wish to express my respect, sincere gratitude and thanks to my beloved teacher and Head of The Department **PROF DR.C. PARANTHAKAN, M.D.**, and my Unit Chief, **PROF.DR. S. VETRIVEL, M.D.**, for his valuable guidance and encouragement during the study and also throughout my course period.

I am extremely thankful to Assistant Professors of Medicine of my Unit **DR.S.VENNILA M.D, DR. V.NARESHKUMAR M.D**, for their valid comments and suggestions.

I profusely thank the Department of Cardiology and the Department of Biochemistry for their timely help, cooperation and support.

I sincerely thank all the staffs of Department of Medicine and Department Of Cardiology and Biochemistry for their timely help rendered to me, whenever needed.

I extend my thanks to all my friends, batch mates, any senior and junior colleagues who have stood by me and supported me throughout my study and course period.

I express my gratitude to all the patients who participated in the study for their extreme patience and co-operation.

I am extremely thankful to my Parents for their continuous support.

LIST OF ABBREVIATION

AMI- Acute Myocardial Infarction

AF- Atrial Fibrillation

AVP- Arginine Vasopressin

CAD- Coronary Artery Disease

CCF- Congestive Cardiac Failure

CK-MB – Creatinine Kinase-MB

CVD- Cardiovascular Disease

ECF- Extra Cellular Fluid

ECG- Electrocardiogram

IHD- Ischemic Heart Disease

ICF- Intra cellular Fluid

LV- Left Ventricle

LVF- Left Ventricular Failure

MR- Mitral Regurgitation

PSVT- Paroxysmal SupraVentricular Tachycardia

SIADH- Syndrome of Inappropriate Antidiuretic Hormone Secretion

TR- Tricuspid Regurgitation

VF- Ventricular Fibrillation

VPC- Ventricular Premature Complex

VT- Ventricular Tachycardia

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ABSTRACT:

Introduction:

Following a Myocardial Infarction, hyponatremia is frequent, and a rise in plasma sodium contents coincides with clinical recovery. Hyponatremia was present in about one third of the congestive heart failure patients. It has been demonstrated that in patients with heart failure and STEMI, it is a predictor of cardiovascular death. Acute STEMI patients who presented with hyponatremia at the time of admission and within 72 hrs of admission had high mortality and so serum sodium level (hyponatremia) is a prognostic indicator. Patient at risk can be identified by a simple marker of plasma sodium level.

Objectives:

To study the prognostic importance of serum sodium level (hyponatremia) among 80 patients with acute STEMI.

Methodology:

This is a Cross sectional analytical study, among 80 patients admitted with acute STEMI under the Department of General Medicine, Thanjavur Medical College and hospital, Thanjavur. Relevant history, clinical findings, biochemical measurements especially the serum sodium levels were compared with the outcomes. Prognostic value of the serum sodium levels were analyzed.

Results:

Among the subjects, 71 (88.75%) were Recovered and 9 (11.25%) had Death. The mean Serum Sodium at admission, at 24 hours, at 48 hours and 72 hours (mEq/L) among Death

was lower and statistically significant compared to the Recovered. Serum Sodium on admission, at 24 hours, at 48 hours and 72 hours in predicting Death was significant, with the good diagnostic accuracy value.

Conclusion:

Serum sodium can be used as a prognostic parameter for predicting death among the acute STEMI patients. The role of correction of the hyponatremia in prevention of the mortality rate among the acute STEMI patients' needs to be explored.

Keywords:

Serum sodium level, Hyponatremia, Acute coronary syndrome, Myocardial infarction, acute STEMI.

1 INTRODUCTION

Acute myocardial infarction is one of the prevalent diseases with substantial side effects, causing significantly increased rates of death, morbidity, disability, and societal costs.(1) Acute MI is a major public health concern in industrialised nations and is becoming increasingly significant in developing nations despite excellent studies in diagnosis and management over the past 40 years.(2,3)

Developing countries, particularly India, are anticipated to have a dramatic rise in ischemic heart disease and myocardial infarction due to a drop in infectious disease-related mortality coupled with accelerated economic development and lifestyle changes that promote atherosclerosis..(4,5)

Of the acute coronary syndromes, Acute STEMI is a major cause of morbidity and mortality in the world. With changing lifestyles the incidence seems to increase forever. Nearly 3 million STEMI occur every year in India. Urgent attention and appropriate treatment is essential to save life of the patient. (6–8)

One prevalent electrolyte problem among hospitalised patients is hyponatremia. A serum sodium concentration (Na^+) of more than 135 mmol/liter is referred to as hyponatremia, which can result from either greater sodium loss from the body or increased fluid retention (dilution-hyponatremia).(9)

Since the majority of moderate hyponatremia's symptoms are non-specific, severe hyponatremia typically manifests as CNS abnormalities.(10,11) The mortality rate for hyponatremia varies from 5% to 50% depending on how severe it is and how long it lasts.(12)

Following a Myocardial Infarction, hyponatremia is frequent, and a rise in plasma sodium contents coincides with clinical recovery.(13,14) Hyponatremia was present in about one third of the congestive heart failure patients.(15) It has been demonstrated that in patients with heart failure and STEMI, it is a predictor of cardiovascular death.

These patients' low stroke volume, low cardiac output, which leads to underfilling of the arterioles and activation of the Renin Angiotensin Aldosterone System, the non-osmotic release of vasopressin and intense sympathetic stimulation, which causes water retention, and vasoconstriction, which results in dilutional hyponatremia are some of the mechanisms contributing to their low serum sodium levels. The transmembrane potentials in cardiac cells, the production of regulatory proteins and enzymes, and the excitation of muscles may all be impacted by the serum sodium concentration. (16–18)

Need for the study / Justification of the study:

Acute STEMI patients who presented with hyponatremia at the time of admission and within 72 hrs of admission had high mortality and so serum sodium level (hyponatremia) is a prognostic indicator. Patient at risk can be identified by a simple marker of plasma sodium level. Therefore this study aims to study the prognostic importance of serum sodium level (hyponatremia) among 80 patients with acute STEMI.

2 AIM AND OBJECTIVES

2.1 AIM:

To study the prognostic importance of serum sodium level (hyponatremia) among 80 patients with acute STEMI.

2.2 OBJECTIVES:

- **Primary Objectives:**

To study the prognostic importance of serum sodium level (hyponatremia) in acute STEMI (without NSTEMI, unstable angina) in a series of 80 cases in Thanjavur medical college

To study the prognostic importance of serum sodium level (hyponatremia) in acute STEMI with age and sex distribution and various risk factor.

- **Secondary Objectives:**

To study its usefulness in predicting its poor prognosis and short time survival

3 REVIEW OF LITERATURE

Review of Literature of this study on prognostic importance of serum sodium level (hyponatremia) in acute STEMI, is discussed with the following heads:

a. Myocardial infarction

- Classification of Myocardial Infarction
- Pathogenesis of Myocardial Infarction
- Stages of repair following Myocardial infarction:
- ST Elevation Myocardial Infarction
- Non-STEMI
- Management
- Complications

b. Hyponatremia

- Metabolism of Sodium
- Renin Angiotensin Aldosterone axis and Regulation of Sodium
- Hyponatremia -Definition
- Drugs causing Hyponatremia
- Pathogenesis of Hyponatremia
- Clinicals features of Hyponatremia
- Treatment of hyponatremia
- Relationship between Hyponatremia and Myocardial Infarction.

c. Studies focussing on prognostic importance of serum sodium level (hyponatremia) in acute STEMI

a. Myocardial Infarction:

A myocardial infarction (MI) is the term used to describe the death of myocardial cells brought on by insufficient oxygen delivery to the myocardium, most frequently as a result of the coronary thrombus that lies on the underlying pathology of a ruptured atherosclerotic plaque.

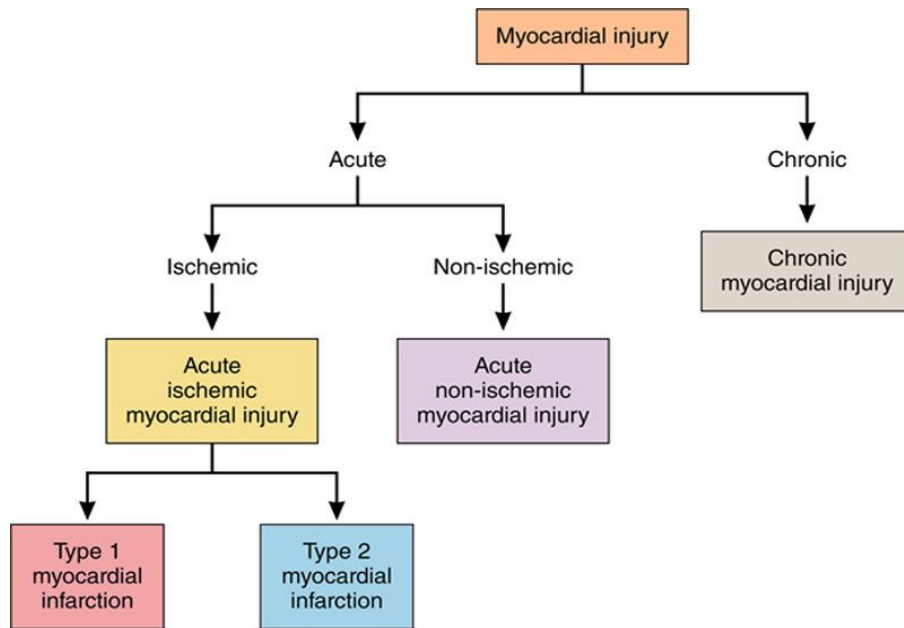
Classification of Myocardial infarction:

Cardiac troponin >99th percentile upper reference limit without any acute change indicates chronic myocardial damage. Either one of the immediate symptoms of myocardial ischemia and a cardiac troponin level >99th percentile upper reference limit constitute an acute myocardial damage.

- A. Symptoms of acute ischemia of myocardium.
- B. Newer ischemic changes in ECG.
- C. Appearance of pathological Q waves in ECG.
- D. new regional wall motion abnormalities or imaging evidence of a fresh loss of viable myocardium in a pattern that is consistent with an ischemia cause
- E. Confirmation of the presence of coronary thrombus either by angiography or autopsy (not for type 2-MI)

Acute nonischaemic myocardial damage is defined as the lack of a primary cause with a rise and decrease in cardiac biomarkers. Atherothrombotic coronary artery disease (CAD) is the primary cause of type 1 MI, which is typically brought on by the rupture of atherosclerotic plaque. Non-atherothrombotic causes of type 2 MI lead to an imbalance between oxygen supply and demand.(19) The following figure represents the categorisation of damage to the myocardium ,(19)

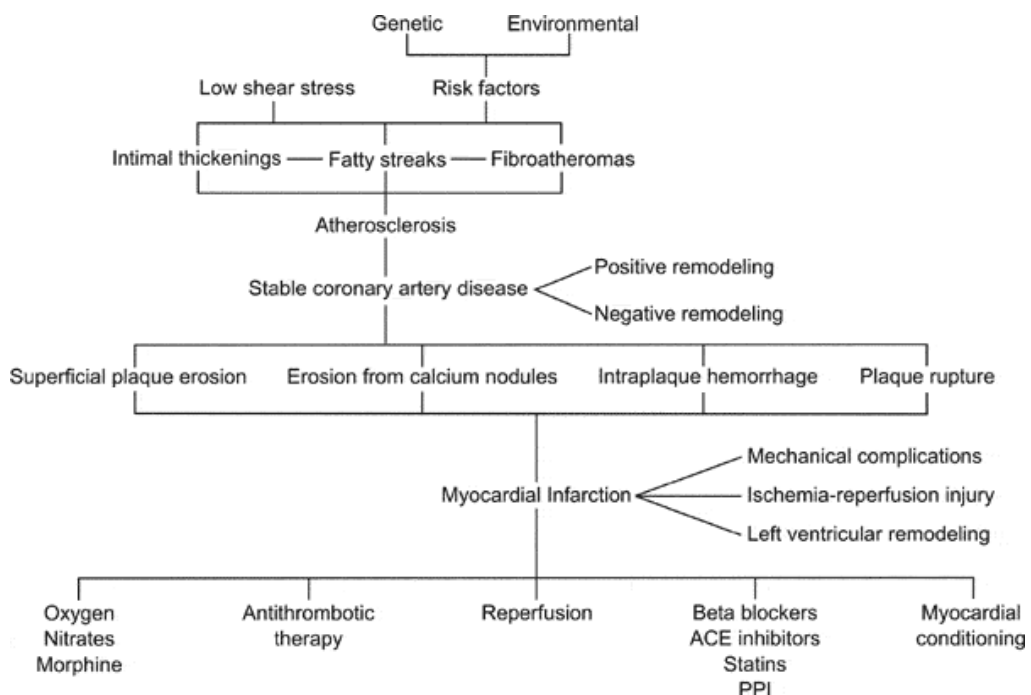
Figure 1. Categorisation of damage to the myocardium



Pathogenesis of Myocardial infarction:

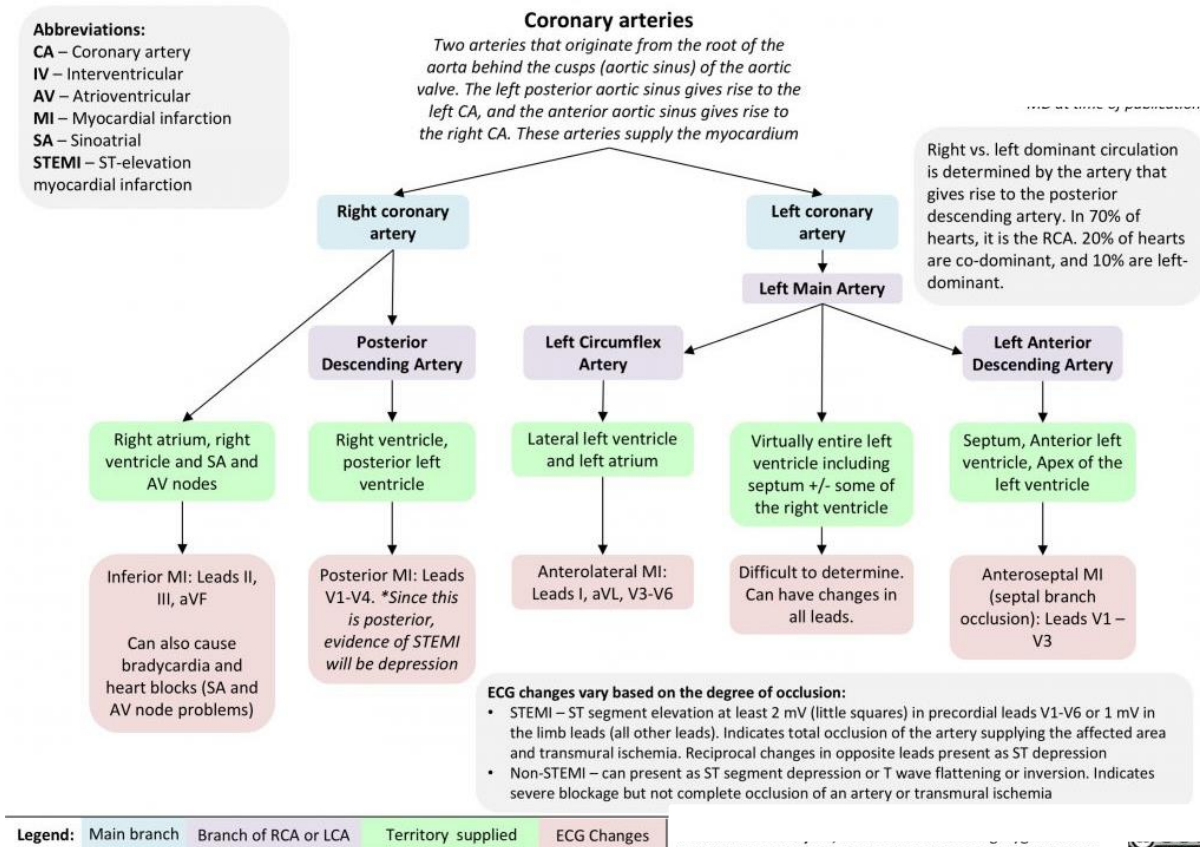
The below figure signifies the risk factors, Pathogenesis, clinical presentations, and management options myocardial infarction, (20)

Figure 2. Pathogenesis of Myocardial infarction.



The coronary anatomy of the ECG and the location of the ischemia are shown in the next figure, (21)

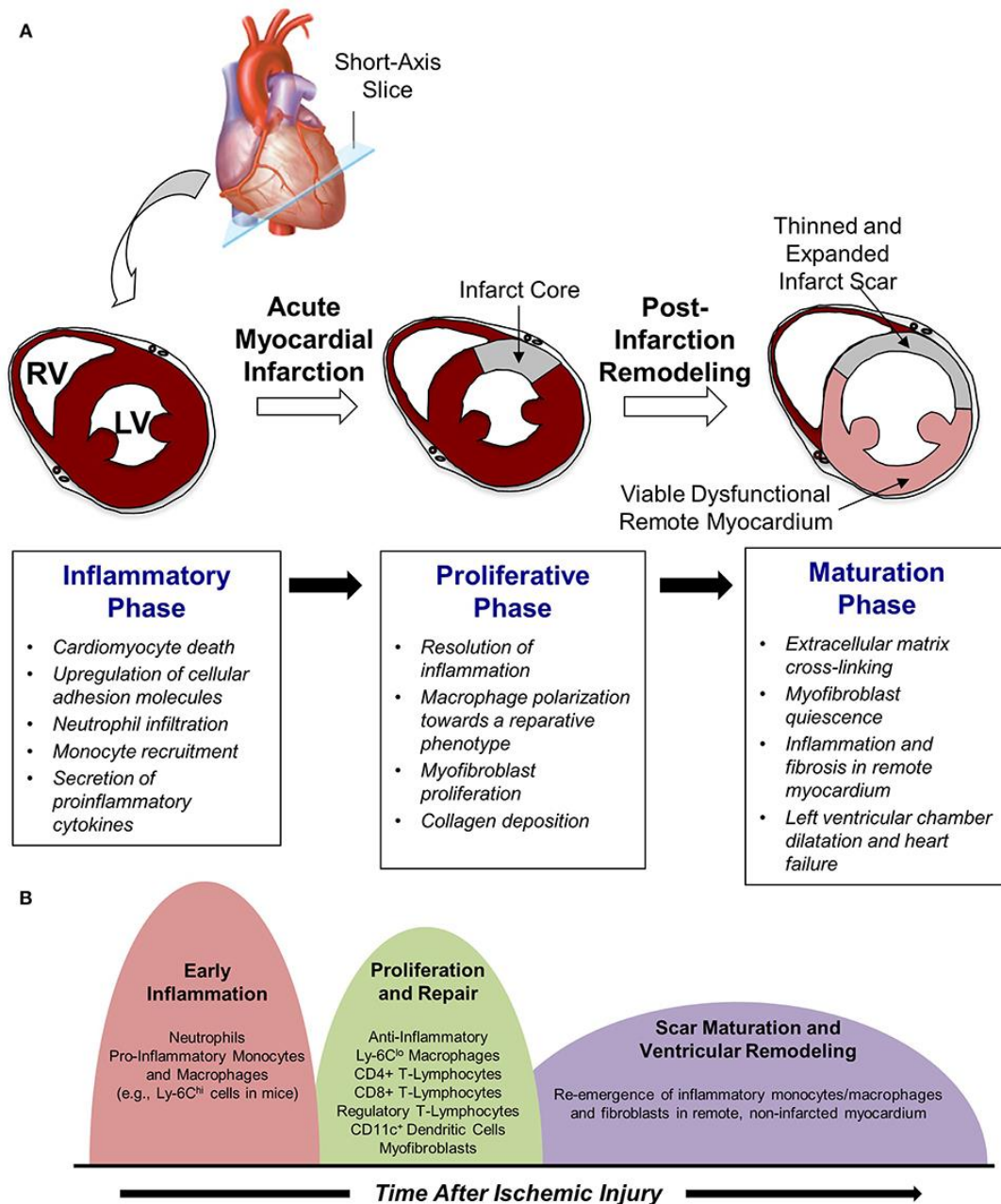
Figure 3. Coronary anatomy in ECG



Stages of repair following Myocardial infarction:

The three stages of cardiac repair following myocardial infarction are shown in the following illustration.,(22)

Figure 4. Three stages of cardiac repair following myocardial infarction



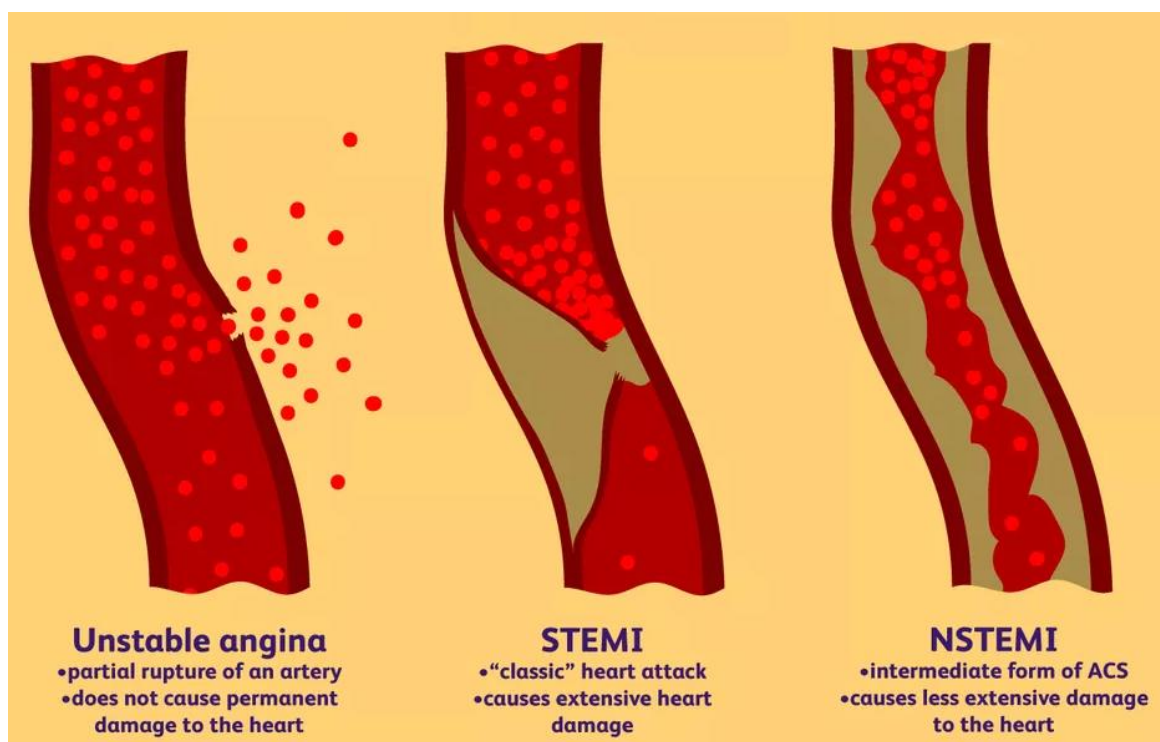
NON-ST Elevation Myocardial Infarction:

Acute Coronary Syndrome is divided into three categories based on the degree of the occlusion.

- I. Unstable angina (partial artery rupture does not result in permanent damage to the myocardium),
- II. STEMI (classic heart attack) in which a major coronary artery is partially or completely blocked by ruptured plaque, and
- III. NSTEMI (believed to be the "intermediate" form of ACS) in which a minor coronary artery or a major coronary artery is partially blocked, but in which the degree of heart tissue damage is much less severe.(23)

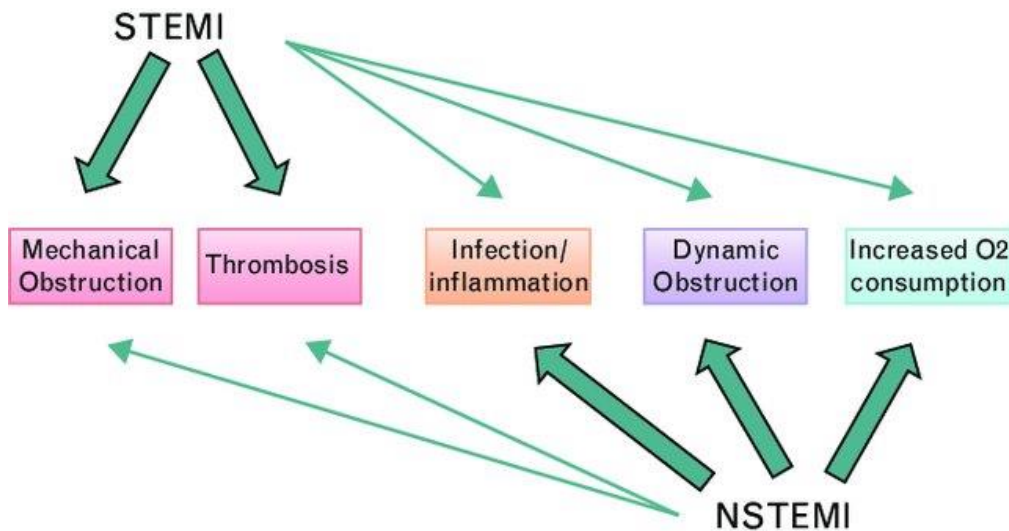
According to the severity of the obstruction, the following illustration depicts the many forms of acute coronary syndromes.,(23)

Figure 5. Forms of the acute coronary syndromes



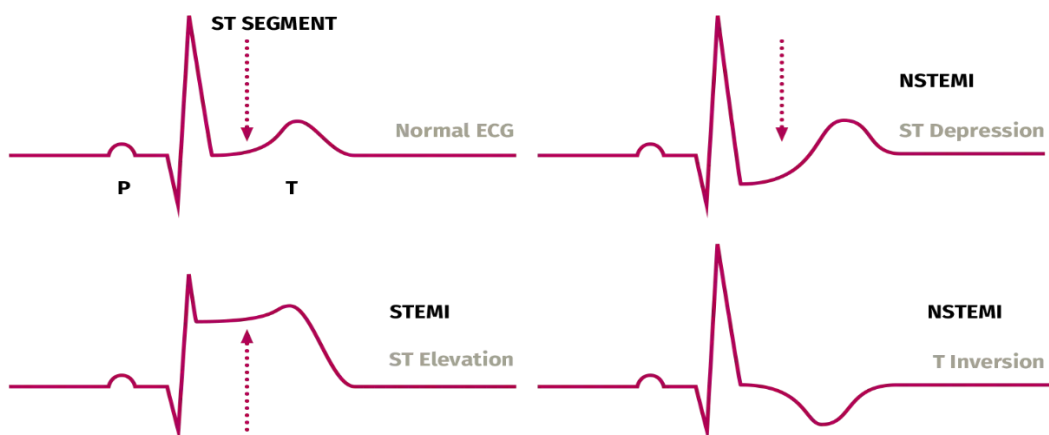
The pathophysiological pathways producing acute plaque rupture and thrombus development in ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) are shown in the accompanying image (dynamic imbalance between the oxygen demand and supply in the myocardium). (24)

Figure 6. Mechanism of STEMI and NSTEMI



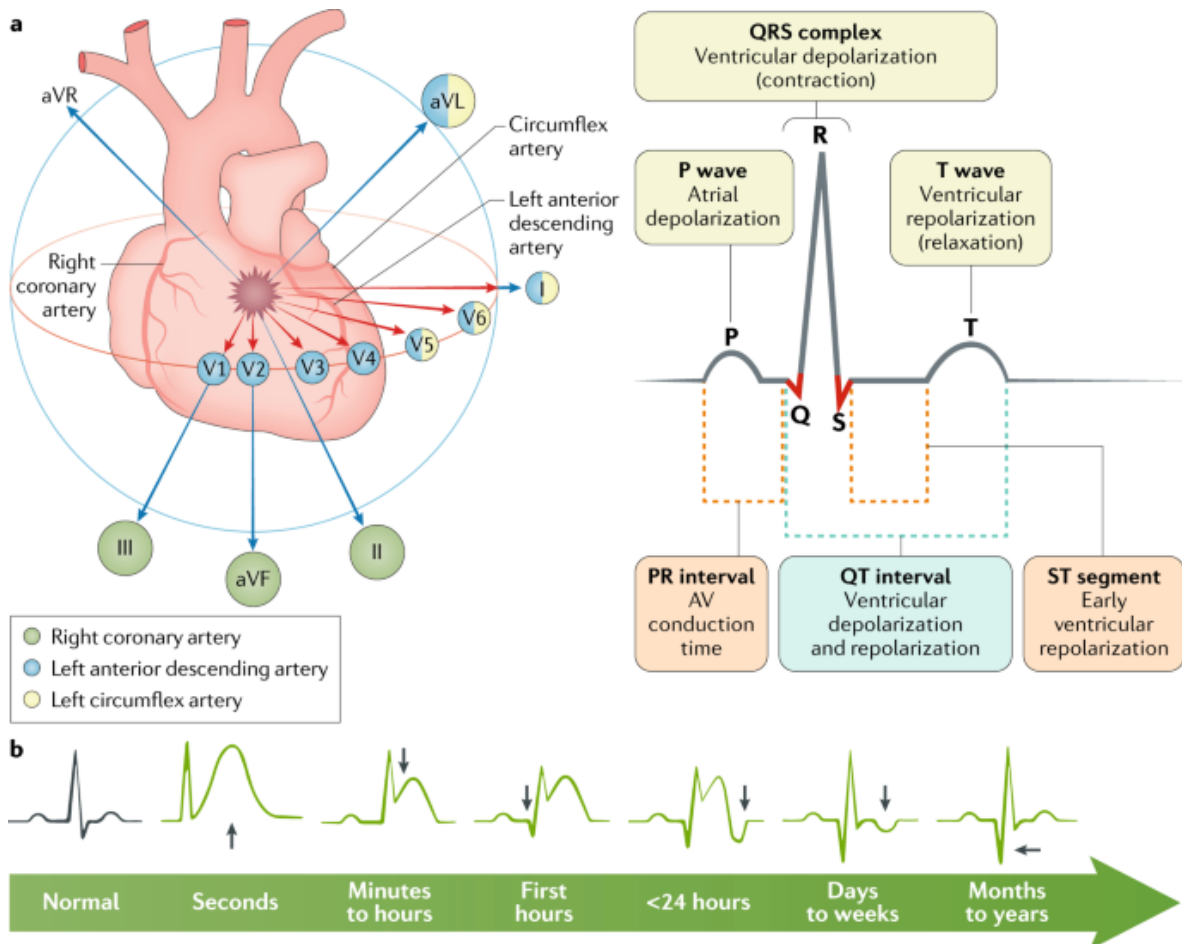
The ST segment is shown in the following images for normal, STEMI, and NSTEMI ECG.,(25)

Figure 7. ST segment in ECG



The ST-segment is seen in the following image. Relationship between normal ECG, ECG leads, and period of progression in myocardial infarction, (26)

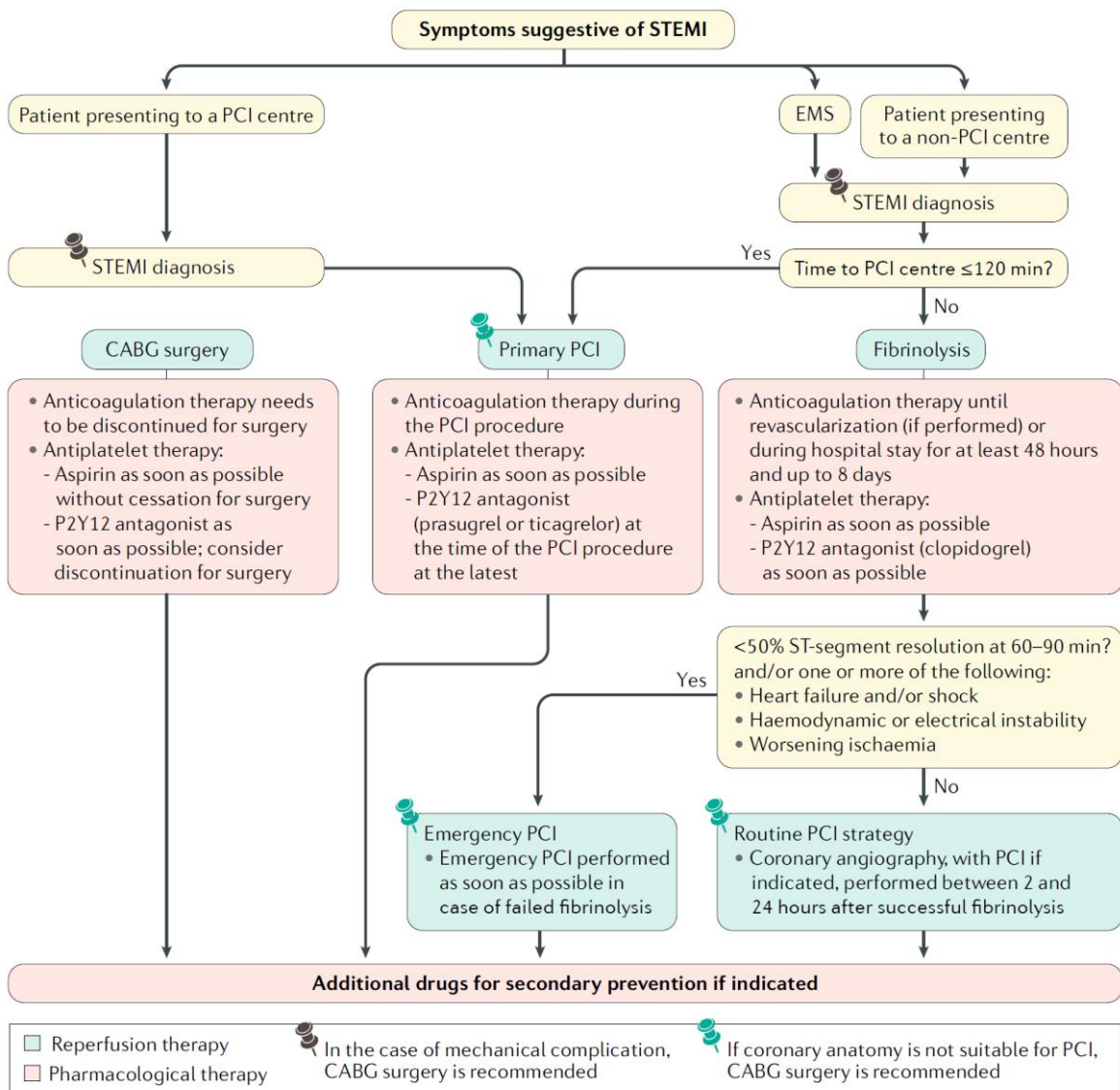
Figure 8. ST-segment in Myocardial infarction



Management:

The management algorithm for the ST elevation MI is represented by the following algorithm.,(26)

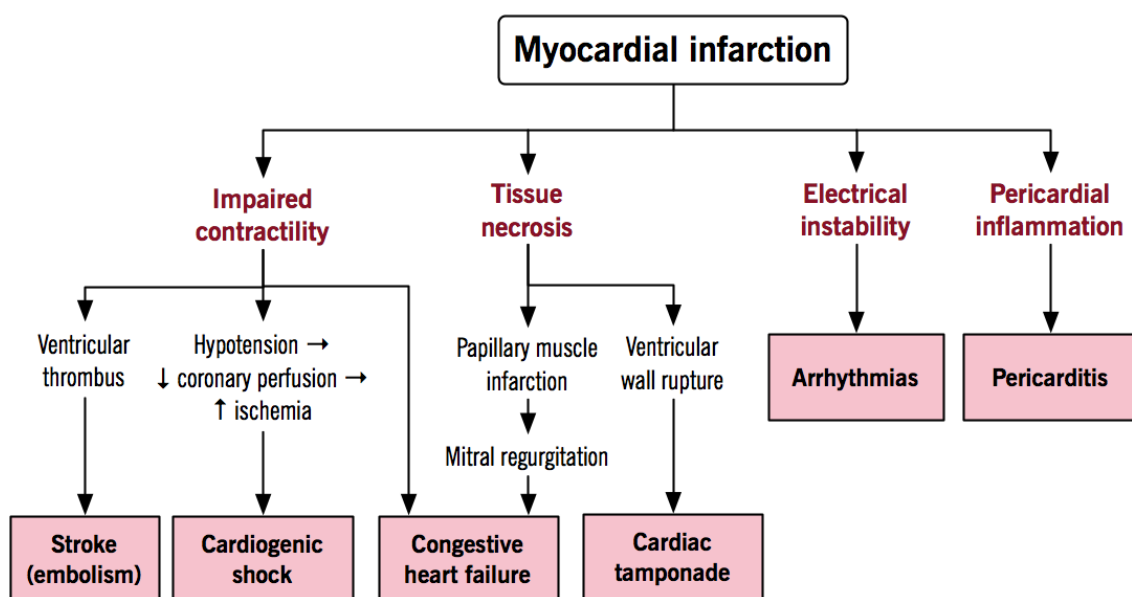
Figure 9. Algorithm for Management in ST elevation MI



Complications:

Myocardial necrosis causes the early complications, and inflammation of necrotic tissue and subsequent recovery cause the later complications. The following illustration shows the underlying mechanism and complications of myocardial infarction., (27)

Figure 10. Complications associated with Myocardial infarction



b. Hyponatremia:

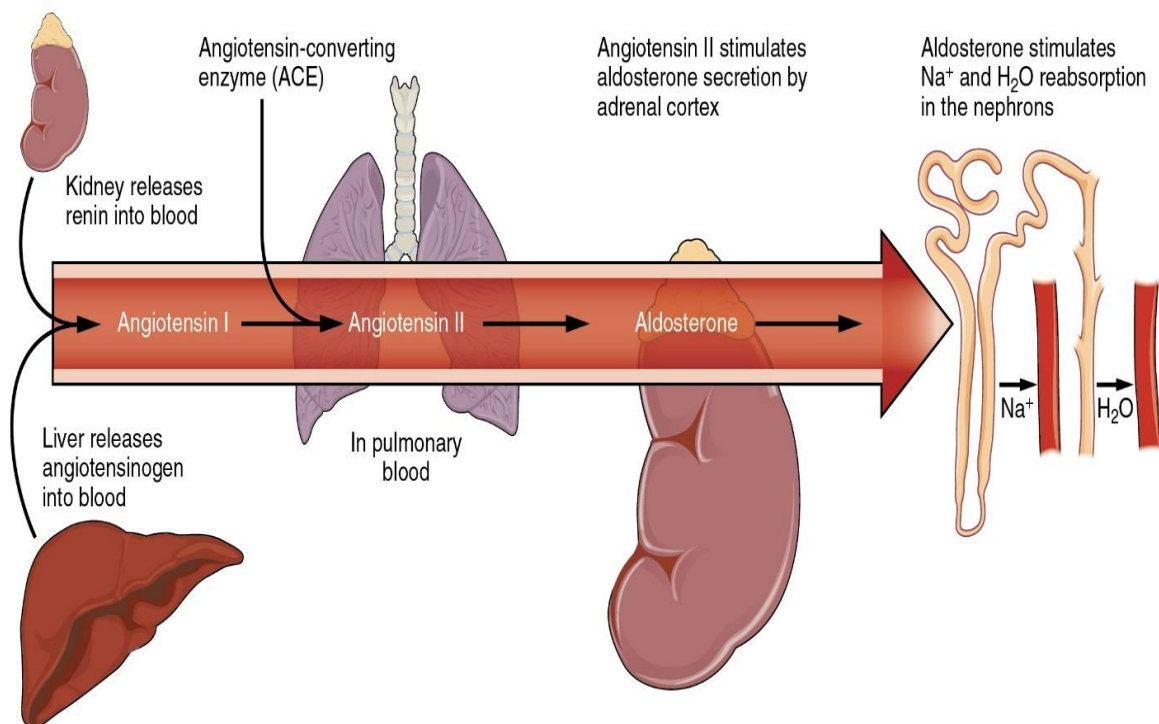
Metabolism of Sodium in body:

All living things require sodium, which is provided from food through salt, which is chemically Sodium-Chloride, to function. (28) Sodium is crucial for maintaining blood volume, osmotic equilibrium, blood pressure, pH level, and appropriate nerve and muscle transmission. It also plays a crucial function in the regulation of fluid and electrolyte balance. (29–31)

Renin-Angiotensin -Aldosterone axis:

Aldosterone, a hormone secreted by the adrenal gland, aids in the reabsorption of Na^+ and subsequently the reabsorption of water, which is crucial for controlling the amount of sodium in the blood.(32) The Renin-Angiotensin System is depicted in the following figure. (Angiotensin II promotes the adrenal cortex's release of aldosterone.).)(32)

Figure 11. Renin-Angiotensin-Aldosterone System:



Hyponatremia:

A serum sodium concentration (Na⁺) of less than 135 mmol/l is referred to as hyponatremia, which can result from either greater sodium loss from the body or increased fluid retention (dilution-hyponatremia). the following are clinical signs of mild gradual hyponatremia,

- i. Nausea,
- ii. Vomiting,
- iii. Headache,
- iv. Cramps in muscle,
- v. Easy fatigability. (33,34)

The following are possible side effects of severe and quickly developing hyponatremia:

- i. Cerebral oedema,
- ii. Coma,
- iii. Unconsciousness,
- iv. Seizures,
- v. Permanent brain damage.

Without immediate and appropriate medical therapy of the disease, acute or severe hyponatremia may result in mortality for the patient.(35)

Drugs resulting in Hyponatremia:

The list of medications that put patients at an elevated risk of hyponatremia is shown in the table below.(31,36)

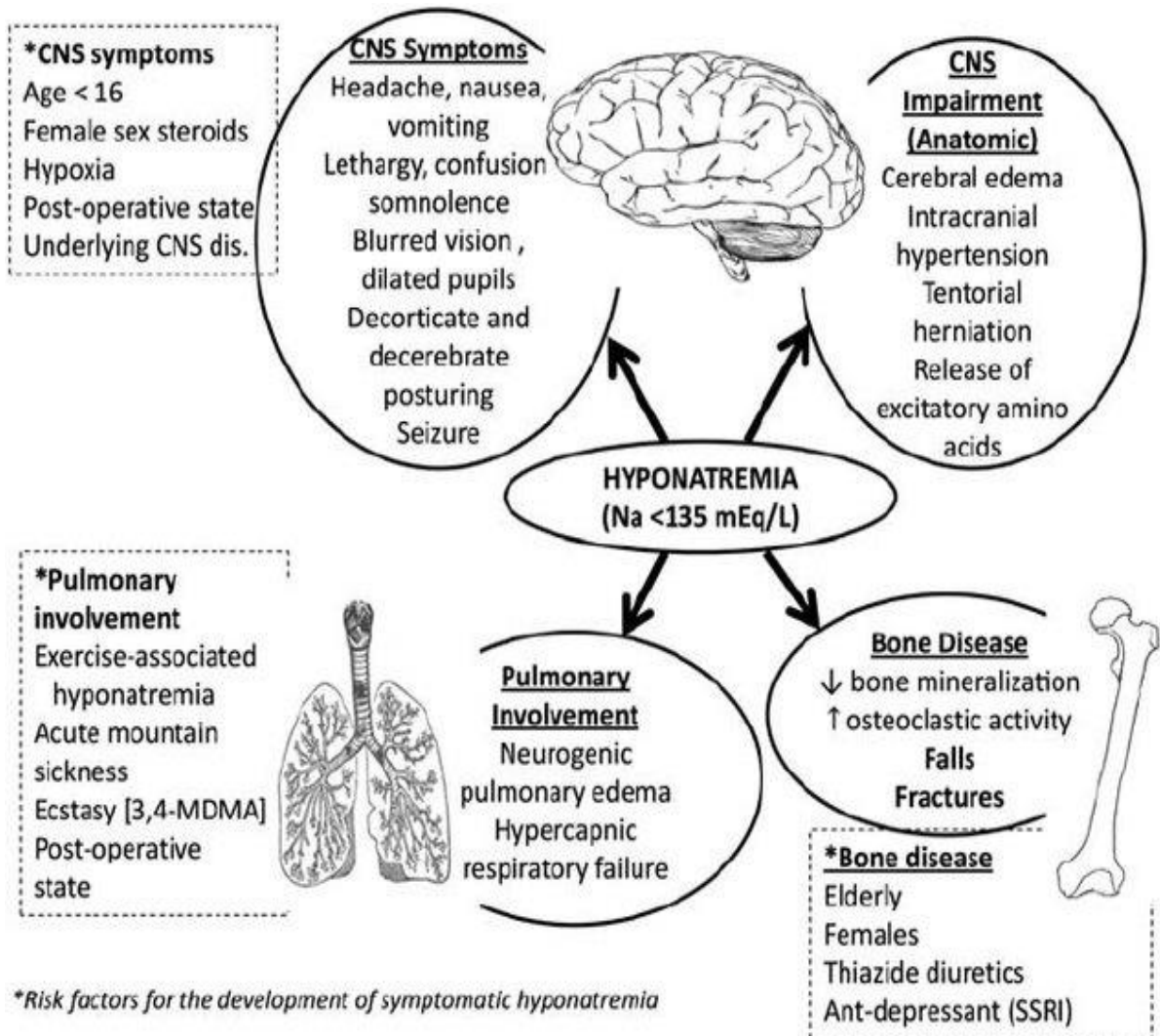
Table 1. Drugs increasing the Risk of Hyponatremia:

Medication Family	Examples
Diuretics	Hydrochlorothiazide, Furosemide (Lasix)
Non-steroidal anti-inflammatory drugs (NSAIDs)	Ibuprofen (Advil, Motrin), Naproxen sodium (Aleve)
Opiate derivatives	Codeine, Morphine
Phenothiazines	Prochlorperazine (Compazine), Promethazine (Phenergan)
Serotonin-reuptake inhibitors (SSRIs)	Fluoxetine (Prozac), Paroxetine (Paxil)
Tricyclic antidepressants	Amitriptyline (Elavil), Imipramine (Tofranil)
Individual Medications Associated with Hyponatremia	
Carbamazepine (Tegretol)	
Chlorpropamide (Diabinese)	
Clofibrate (Atromid-S)	
Cyclophosphamide (Cytoxan)	
Desmopressin (DDAVP; nasal or oral)	
Lamotrigine (Lamictal)	
Oxytocin (Pitocin)	
Vincristine (Oncovin)	

Pathogenesis of hyponatremia:

The next picture shows an overview of the pathogenesis of hyponatremia, including risk factors, symptoms, and indicators in various organs..(37)

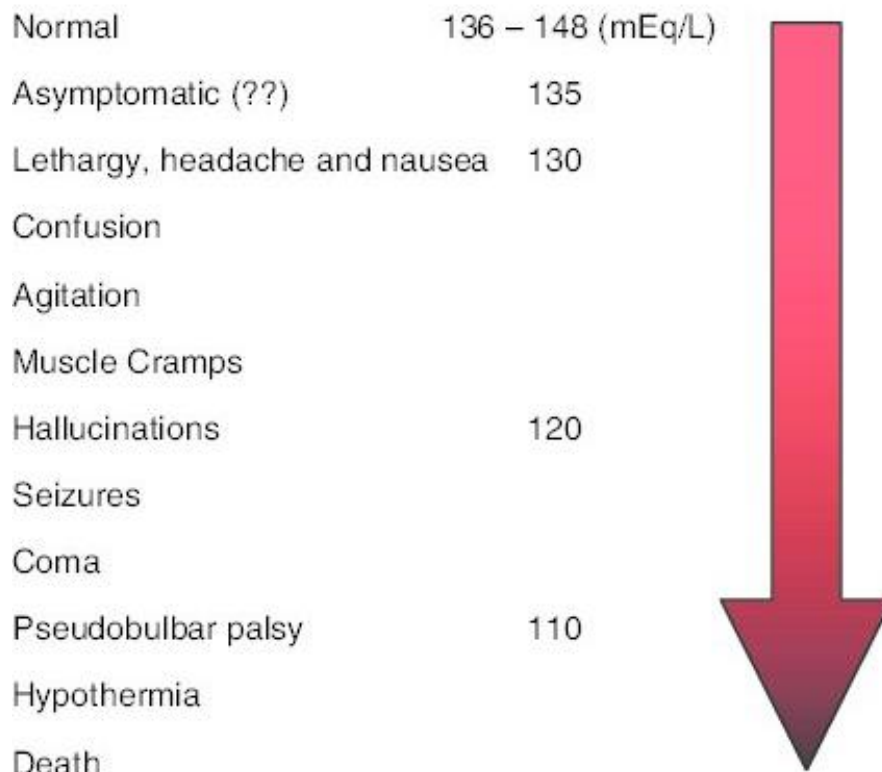
Figure 12. Pathogenesis of hyponatremia:



Clinical features associated with hyponatremia:

The following illustration depicts hyponatremia symptoms in relation to falling serum sodium levels.:(38)

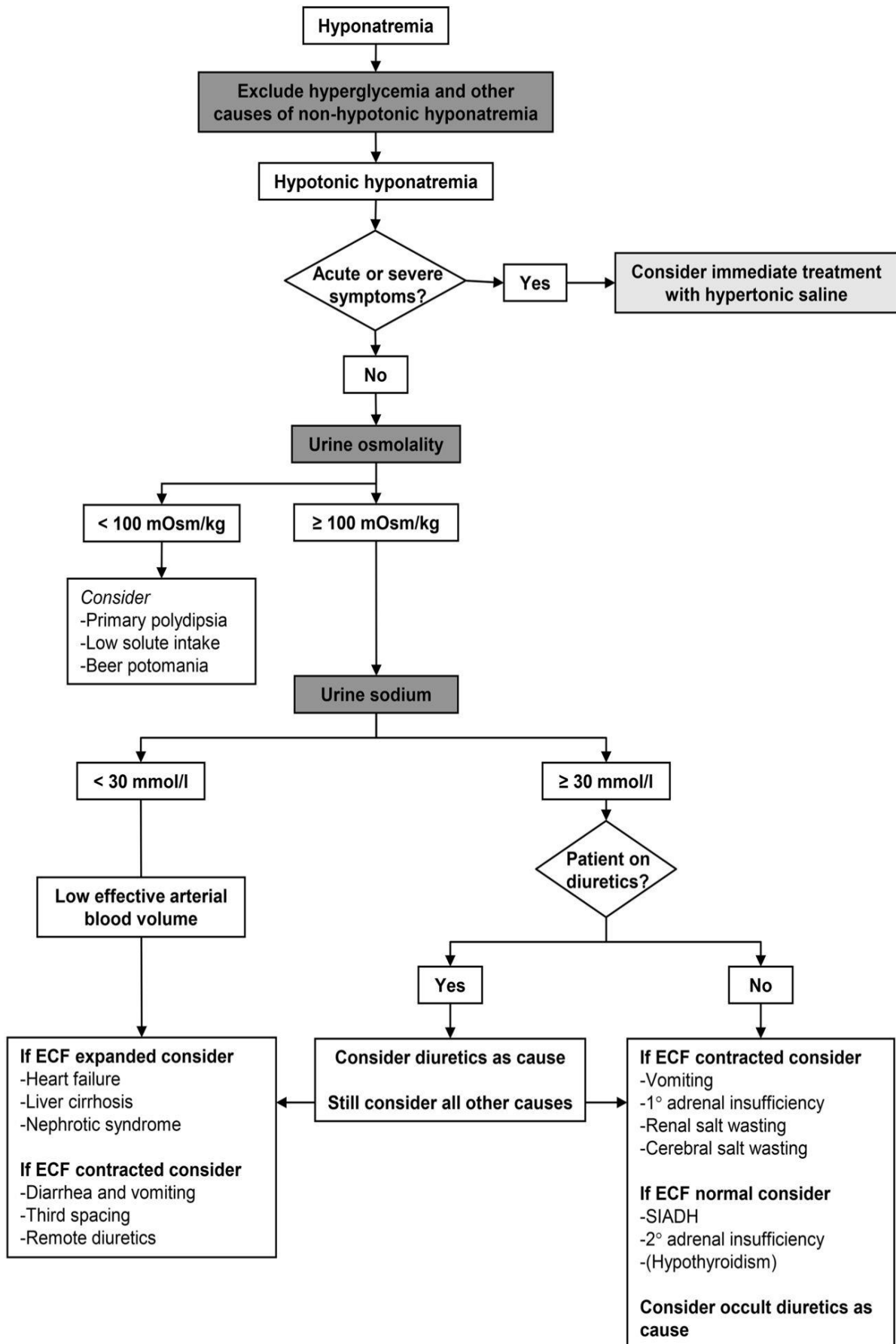
Figure 13. Clinical features associated with Hyponatremia:



Algorithm for management of hyponatremia:

The algorithm for treating patients who come with hyponatremia is shown in the accompanying graphic. (The initial stage in assessing hyponatremia is taking a patient's history. The clinician's primary responsibility comes after determining the plasma osmolality: determining the bodily fluid state. The FENa computation aids in the final diagnosis assessment.)(39)

Figure 14. Algorithm for management of hyponatremia:



Formula for infusion of Sodium:

The formula used to calculate the impact of infusates and fluid losses on Na⁺ are shown in the following table:(40)

Table 2. Formula for calculation of Sodium infusion

Infusate Formula	Fluid-Loss Formula
$\Delta[\text{Na}^+]_s = \frac{[\text{Na}^+ + \text{K}^+]_{\text{inf}} - [\text{Na}^+]_s}{\text{TBW} + 1}$	$\Delta[\text{Na}^+]_s = \frac{[\text{Na}^+]_s - [\text{Na}^+ + \text{K}^+]_{\text{fl}}}{\text{TBW} - 1}$
Projects the effect of gaining 1 L of any infusate (inf) on the patient's $[\text{Na}^+]_s$	Projects the effect of losing 1 L of any fluid (fl) on the patient's $[\text{Na}^+]_s$

Treatment Guidelines for hyponatremia:

The following table details the exact ailment and therapy used to treat hyponatremia.(41–43)

Table 3. Summary of Treatment of Hyponatremia:

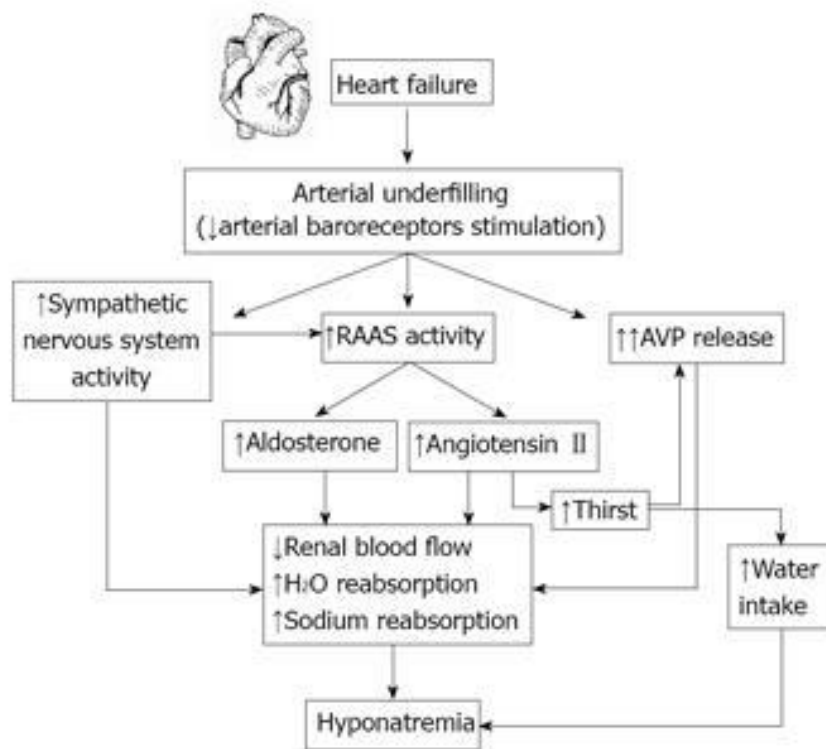
Condition	Therapy
Acute or symptomatic hyponatremia	<ul style="list-style-type: none"> • Severe symptoms: Bolus 3% saline 100 mL x 3 as needed • Moderate symptoms: Continuous infusion 3% saline 0.5-2 mL/kg/hour
Chronic hyponatremia Syndrome of inappropriate antidiuretic hormone secretion	<ul style="list-style-type: none"> • Fluid restriction (first-line) • Loops, diuretics, urea, vaptans, salt tablets, demeclocycline (second-line)
Hypovolemic hyponatremia	<ul style="list-style-type: none"> • Isotonic saline or balanced crystalloids solutions
Hypervolemic hyponatremia	<ul style="list-style-type: none"> • Fluid restrictions, loop diuretics
Sodium correction rates	<ul style="list-style-type: none"> • Minimum: 4-8 mmol/L/day, 4-6 mmol/L/day if high risk for osmotic demyelination syndrome (ODS) • Limits: 10-12 mmol/L/day, 8 mmol/L/day if high risk for ODS
Management of overcorrection	<ul style="list-style-type: none"> • Baseline SNA > 120 mmol/L: Start once limit of Na correction is exceeded • Baseline SNA < 120 mmol/L: Start relowering with electrolyte-free water (10 mL/kg) with or without desmopressin 2µg IV after correction exceeds 6-8

Relationship between Myocardial Infarction and Hyponatremia:

Patients' with myocardial infarction have low stroke volume, low cardiac output, which leads to underfilling of the arterioles and activation of the Renin Angiotensin Aldosterone System, the non-osmotic release of vasopressin and intense sympathetic stimulation, which causes water retention, and vasoconstriction, which results in dilutional hyponatremia are some of the mechanisms contributing to their low serum sodium levels. The transmembrane potentials in cardiac cells, the production of regulatory proteins, enzymes, and the stimulation of muscles may all be impacted by the serum sodium level..(16–18)

Since the majority of moderate hyponatremia's symptoms are non-specific, severe hyponatremia typically manifests as CNS abnormalities..(10,11) The mechanisms for hyponatremia to develop in people with heart failure are depicted in the next picture,(44,45)

Figure 15. Mechanisms of hyponatremia in heart failure.



c. Studies focussing on prognostic importance of serum sodium level (hyponatremia) in acute STEMI:

In 2004, Israeli researchers **Alexander Goldberg et al.** examined the prevalence of hyponatremia among the 1047 individuals who had had an acute ST-elevation myocardial infarction and assessed the prognostic significance of the condition. Serum sodium levels were measured during admission and on a daily basis following that. Hyponatremia (135 mmol/L) was present in 12.5% of patients at admission and 19.9% of patients after 72 hours. Hyponatremia (135 mmol/L) upon admission was linked to a 2.0 odds ratio for 30-day death; (95 percent CI: 1.0 to 3.9). Hyponatremia (130 mmol/L) upon admission was associated with a 30-day mortality rate with an odds ratio of 3.4 (95 percent confidence interval: 1.5 to 7.8).(46)

Israel's **Alexander Goldberg et al.** examined the prevalence of hyponatremia among the 978 individuals who had acute ST-elevation myocardial infarction in 2006 and assessed its predictive significance for the length of hospitalisation and long-term mortality of the condition. Serum sodium levels were measured during admission and on a daily basis following that. At admission, 11 percent of the patients had hyponatremia (135 mmol/L). They found that for every 1-mEq/L decrease in serum sodium, the adjusted Hazard Ratio for mortality or heart failure was 1.12.(47)

In a thorough analysis of more than 1400 papers, **Vraj Shah et al.** looked at the prevalence of hyponatremia in the 1047 individuals who had acute ST-elevation myocardial infarction and assessed its predictive significance for the course of the illness. They noted that hyponatremia worsens short-term and long-term mortality, rehospitalization rates, lengthened hospital stays, and decreased ejection fraction rates.(16)

In their EPHEBUS experiment, **Pieter Martens et al.** looked at how serum sodium and eplerenone were used by the 6632 patients who had myocardial infarction, left ventricular dysfunction, or heart failure. They saw that eplerenone's positive effects persisted regardless of baseline serum sodium levels or the onset of hyponatremia..(48)

In a study of 100 patients with acute ST-elevation myocardial infarction, **Konsam Biona Devi et al.** from Manipur examined the prevalence of hyponatremia and assessed the prognostic significance of the condition. At admission, they discovered a 44 percent frequency of hyponatremia (135 mmol/L). Serum sodium levels were measured during admission and on a daily basis following that. They found that a considerably greater proportion of Killip III and IV, a higher level of CK-MB, and a higher degree of ST-elevation were all related to hyponatremia. Out of six hyponatremia patients, four passed away.(49)

Italian researchers **Chiara Lazzeri et al.** examined the prevalence of hyponatremia among the 1231 patients who had acute ST-elevation myocardial infarction and assessed the prognostic significance of the condition. At admission, they discovered a prevalence of hyponatremia (135 mmol/L) of 23.2%. They noted that a considerable proportion of the aged population, diabetics, Killip III and IV, Triple Vein Disease, and increased rates of both long-term and short-term mortality were all associated with hyponatremia..(50)

In a study of 10 patients with acute ST-elevation myocardial infarction, **Makrand B. Mane et al.** examined the prevalence of hyponatremia and assessed the prognostic significance of the condition. They found that a considerably larger percentage of elderly people, tobacco use, diabetes, hypertension, ejection fraction, and both long-term and short-term mortality were all linked to hyponatremia.(51)

In a study by **Vikas et al.**, 200 patients with acute ST-elevation myocardial infarction were examined for the prevalence of hyponatremia and the prognostic significance of the condition. Hyponatremia (135 mmol/L) was seen in 14% of patients at admission and 20% of patients after 72 hours. They discovered that hyponatremia had a strong correlation with both long- and short-term mortality, a greater number of risk factors, a higher Killip class, and a lower ejection percentage.(52)

A total of 100 patients with acute ST-elevation myocardial infarction were analysed by **Suprabhat Giri et al.** to determine the prevalence of hyponatremia and to assess the prognostic significance of the condition. They found that the prevalence of hyponatremia (less than 135 mmol/L) was 29%. Hyponatremia was found to be significantly correlated with both hospital mortality and 7-day MACE in multivariate analysis.(53)

In a study of 100 patients with acute ST-elevation myocardial infarction, **Sidhnath Singh et al.** from Ranchi examined the prevalence of hyponatremia and assessed the prognostic significance of the condition. At admission, 81 percent of patients had hyponatremia (135 mmol/L), and at 72 hours, 77 percent did. They found that long- and short-term mortality, as well as risk variables like smoking, diabetes, anterior infarction, higher Killip class, and reduced ejection fraction, were all strongly correlated with hyponatremia.(54)

RESEARCH QUESTION OR HYPOTHESIS

3.1 **RESEARCH QUESTION:**

What is the prognostic importance of serum sodium level (hyponatremia) in acute STEMI

3.2 **NULL HYPOTHESIS:**

There is no significant relationship between the serum sodium level (hyponatremia) and the prognosis in acute STEMI.

3.3 **RESEARCH OR ALTERNATE HYPOTHESIS:**

There is a significant relationship between the serum sodium level (hyponatremia) and the prognosis in acute STEMI.

4 METHODOLOGY

4.1 STUDY SUBJECTS:

80 patients admitted with acute STEMI under the Department of General Medicine, Thanjavur Medical College and hospital, Thanjavur.

4.2 STUDY DESIGN:

Cross sectional analytical study.

4.3 STUDY PARAMETERS:

- Relevant history
- Smoking, alcoholism
- Diabetes, hypertension
- Previous history of CAD, diuretic therapy
- Serum CKMB (IU/L)
- KILIP Class
- Ejection Fraction (%)
- Plasma sodium on admission and 24,48 and 72 hrs.

4.4 STUDY PERIOD:

Data collection – 11/2 years (2020 December to 2022 July).

4.5 STUDY SETTING:

Department of General Medicine, Thanjavur Medical College and hospital, Thanjavur.

4.6 **SAMPLING PROCEDURE:**

Consecutive Sampling.

4.7 **INCLUSION CRITERIA:**

- ✓ More than 20 minutes of chest pain.
- ✓ Age >18 years
- ✓ ECG alteration consisting of new pathological Q waves or ST segment or T wave changes which are diagnosis of MI in ECG.
- ✓ Both males and females
- ✓ Elevation of cardiac enzymes such as creatinine kinase (CKMB) or cardiac troponin T and I levels
- .

4.8 **EXCLUSION CRITERIA:**

- ☒ Acute coronary syndrome without ST elevation
- ☒ Unstable ANGINA
- ☒ Patients with acute MI and cardiac failure with diuretic therapy
- ☒ Age less than 18 years
- ☒ Pre-existing renal disease
- ☒ Patients not capable of giving consent (psychiatric patients).
- ☒ Patients not willing to participate in the study (who refused to consent)

4.9 SAMPLE SIZE:

According to **Ish Singla et al** study,(55) considering the prevalence of hyponatremia (sodium <135 mEq/L) among ST-elevation myocardial infarction (STEMI) patients as 23.1% with a precision of 9.3% and 95% confidence interval, the minimum required sample size is calculated with the following formula,

$$N = Z_{1-\alpha/2}^2 * p * (1 - p) / d^2$$

p (%) - prevalence of hyponatremia (sodium <135 mEq/L) among ST-elevation myocardial infarction (STEMI) patients = 0.231

$Z_{1-\alpha/2}$ - two tailed probability for 95% confidence interval = 1.96

d (%) - precision or allowable error for hyponatremia (sodium <135 mEq/L) among ST-elevation myocardial infarction (STEMI) patients = 0.093

$$N = 1.96^2 * 0.231 * (1 - 0.231) / 0.093^2$$

$$N = 78.9$$

Thus the total sample size required for the study is 79, rounded off to 80.

4.10 ETHICAL CONSIDERATION:

Institutional Ethical Committee approval, from Thanjavur Medical College and hospital, Thanjavur, was applied and granted before the start of the study. Informed written consent was obtained.

Source of Funding: None declared

Conflict of Interest: None declared

4.11 STUDY PROCEDURE:

80 consecutive patients admitted with acute STEMI under the Department of General Medicine, Thanjavur Medical College and hospital, Thanjavur, who fulfilled the eligibility criteria (inclusion and exclusion criteria) were included in the study.

After explaining the study rationale, informed written consent was obtained from all the participants. Relevant history, clinical findings, biochemical measurements especially the serum sodium levels were compared with the outcomes of the study population. Prognostic value of the serum sodium levels were analysed.

4.12 BUDGET:

Self. (No added investigation or intervention)

4.13 STATISTICAL METHODS:

I. Descriptive Statistics:

1. Numerical variables like levels of CKMB, Age, Na levels on Admission, Na levels at 24hrs, Na levels at 48hrs, Na levels at 72 hrs, Ejection Fraction, etc., are represented in mean, SD, median, and mode. Histograms are used wherever necessary.
2. Categorical variables like presence of smoking, alcoholism, diabetes mellitus, systemic hypertension, previous coronary artery disease, prior diuretic therapy, KILIP score, type of MI, gender, outcomes, etc., are represented in frequency and percentage. Pie-charts and bar diagrams are used as necessary.

3. Data was entered in MS Excel sheet and analysed using SPSS software version 16.

II. Inferential Statistics:

1. When a Numerical variable (eg., Na levels on Admission, at 24hrs, at 48hrs, at 72 hrs) are compared with the another Numerical variable, Pearson's correlation test is used.
2. When a Categorical Variable is compared with a serum sodium levels, test of significance, used will be student t test/ ANOVA test as appropriate.
3. When a Categorical Variable is compared with a presence of hyponatremia, the variables are represented in both by tables and bar diagrams. For test of significance, chi-square test is used.
4. P-values less than 0.05 were considered statistically significant.

5 RESULTS

Results of the study, on prognostic importance of serum sodium level (hyponatremia) in acute STEMI, is discussed with the following headings:

I. Age (years)

II. Age group

III. Gender

IV. Smoker

V. Alcoholic

VI. Diabetes Mellitus

VI. Hypertension

VII. Prior CAD

VIII. Prior Diuretic Therapy

IX. Serum CKMB (IU/L)

X. KILIP Class

XI. Serum Sodium

XII. Ejection Fraction (%)

XIII. Myocardial Infarction Type

XIV. Outcome

XV. Serum Sodium on admission (mEq/L) with Outcome

XVI. Serum Sodium at 24 hours (mEq/L) with Outcome

XVII. Serum Sodium at 48 hours (mEq/L) with Outcome

XVIII. Serum Sodium at 72 hours (mEq/L) with Outcome

XIX. Serum Sodium with Myocardial Infarction Type

XX. Serum Sodium with KILIP Class

XXI. Correlation of Serum Sodium with CKMB

XXII. Correlation of Serum Sodium with Ejection Fraction

XXX. ROC for predicting Death among STEMI using Serum Sodium

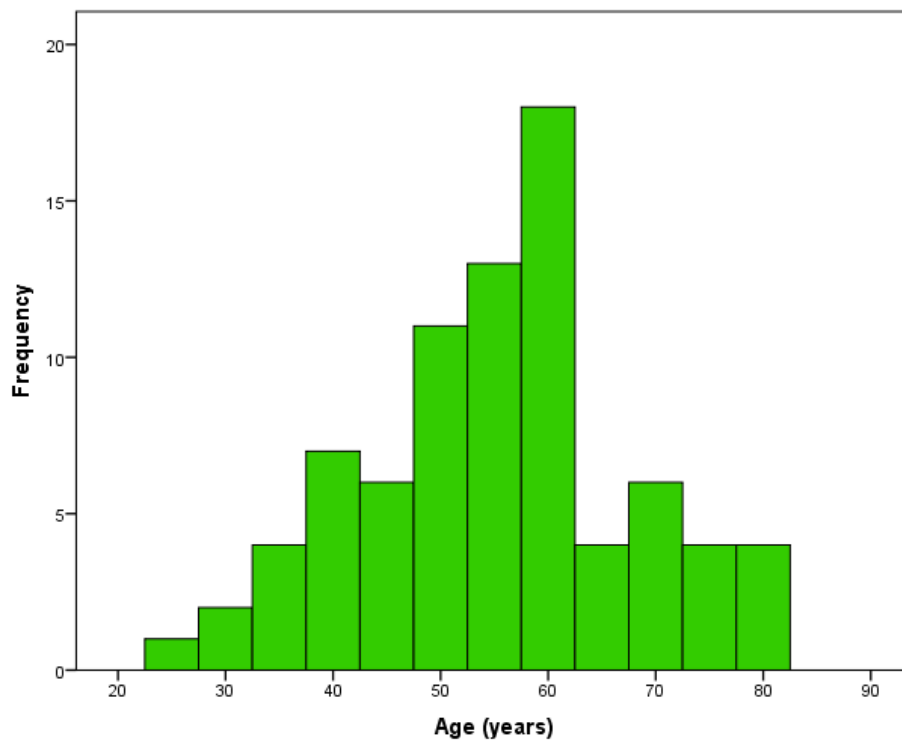
I. Age (years)

The mean Age (years) among the subjects was 55.33 (± 12.62) ranging from 25 to 82 years.

Table 4. Age (years)

Age (years)	
Mean	55.33
Median	55
Std. Deviation	12.62
Range	57
Minimum	25
Maximum	82

Figure 16. Age (years)



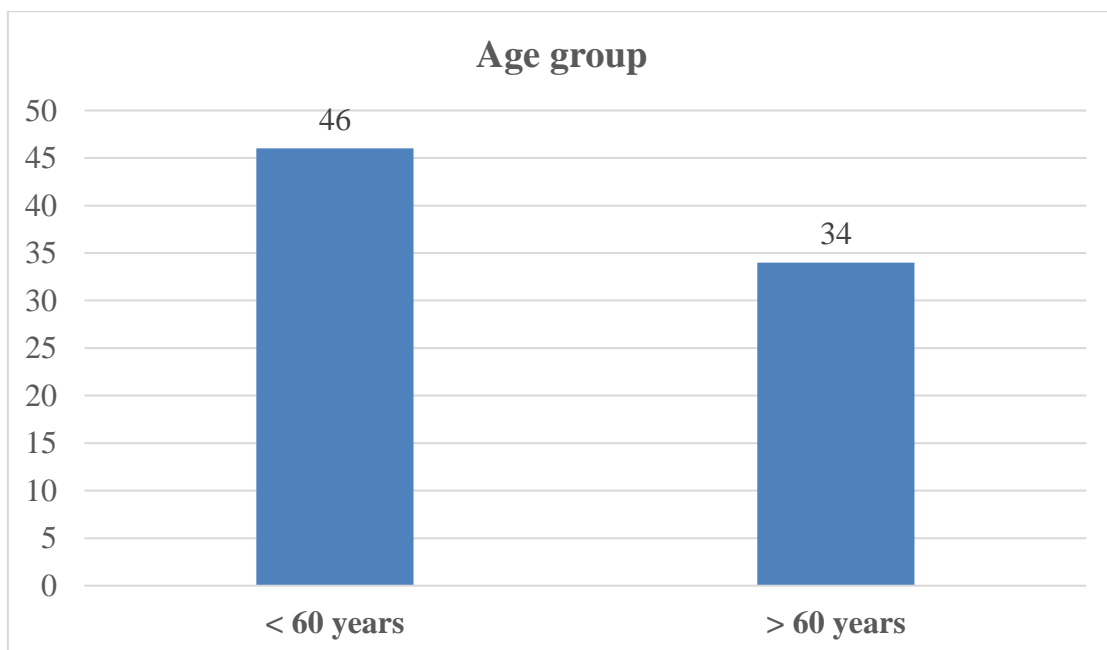
II. Age group

Among the subjects, 46 (57.5%) were in < 60 years and 34 (42.5%) were in > 60 years

Table 5. Age group

Age group	Frequency	Percent
< 60 years	46	57.50
> 60 years	34	42.50
Total	80	100.00

Figure 17. Age group



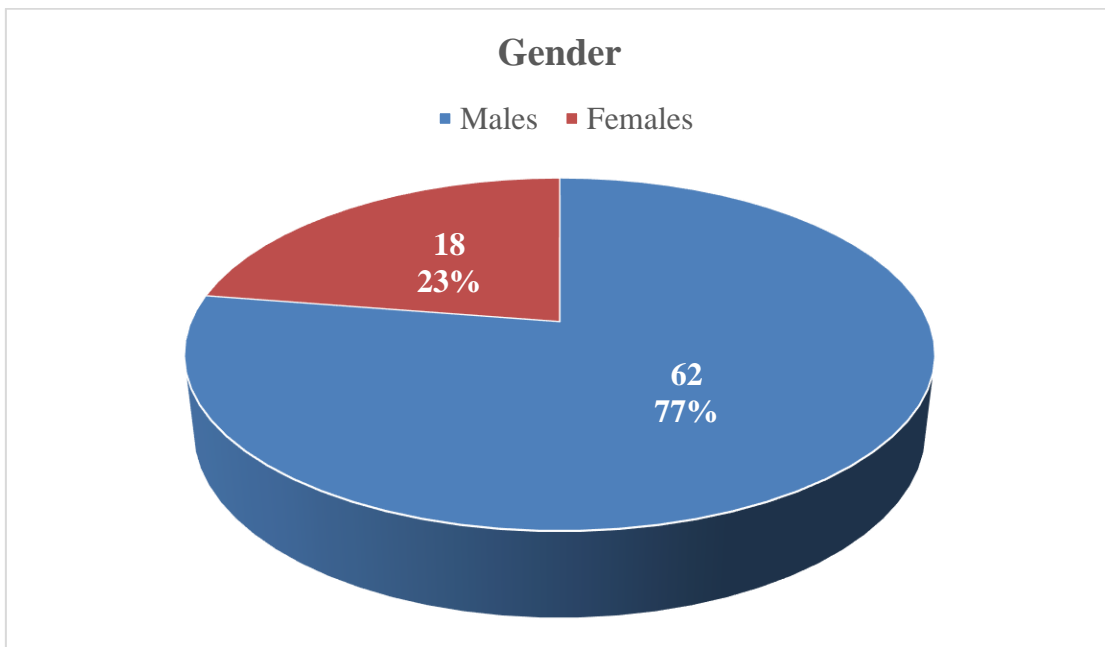
III. Gender

Among the subjects, 62 (77.5%) were Males and 18 (22.5%) were Females

Table 6. Gender

Gender	Frequency	Percent
Males	62	77.50
Females	18	22.50
Total	80	100.00

Figure 18. Gender



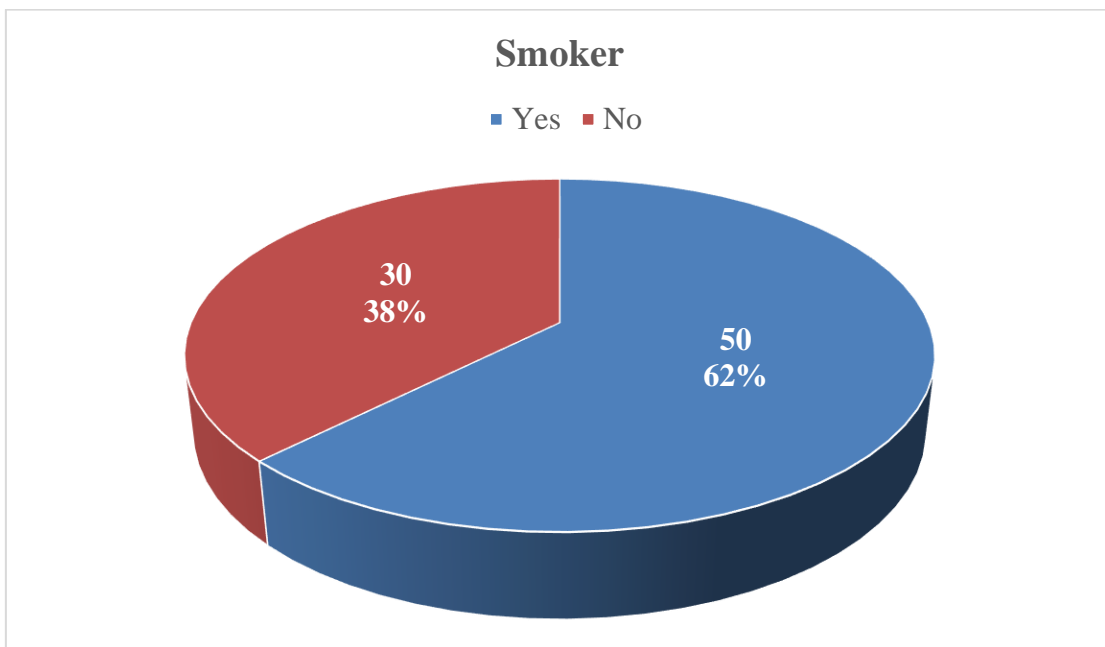
IV. Smoker

Among the subjects, 50 (62.5%) were Smoker

Table 7. Smoker

Smoker	Frequency	Percent
Yes	50	62.50
No	30	37.50
Total	80	100.00

Figure 19. Smoker



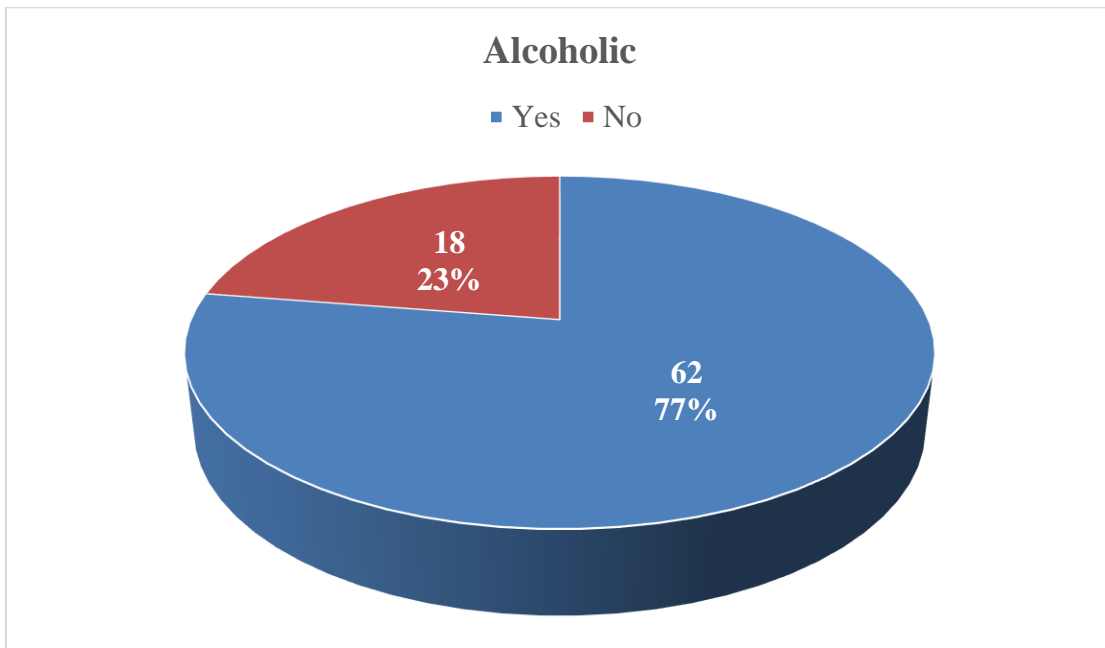
V.Alcoholic

Among the subjects, 62 (77.5%) were Alcoholic

Table 8.Alcoholic

Alcoholic	Frequency	Percent
Yes	62	77.50
No	18	22.50
Total	80	100.00

Figure 20.Alcoholic



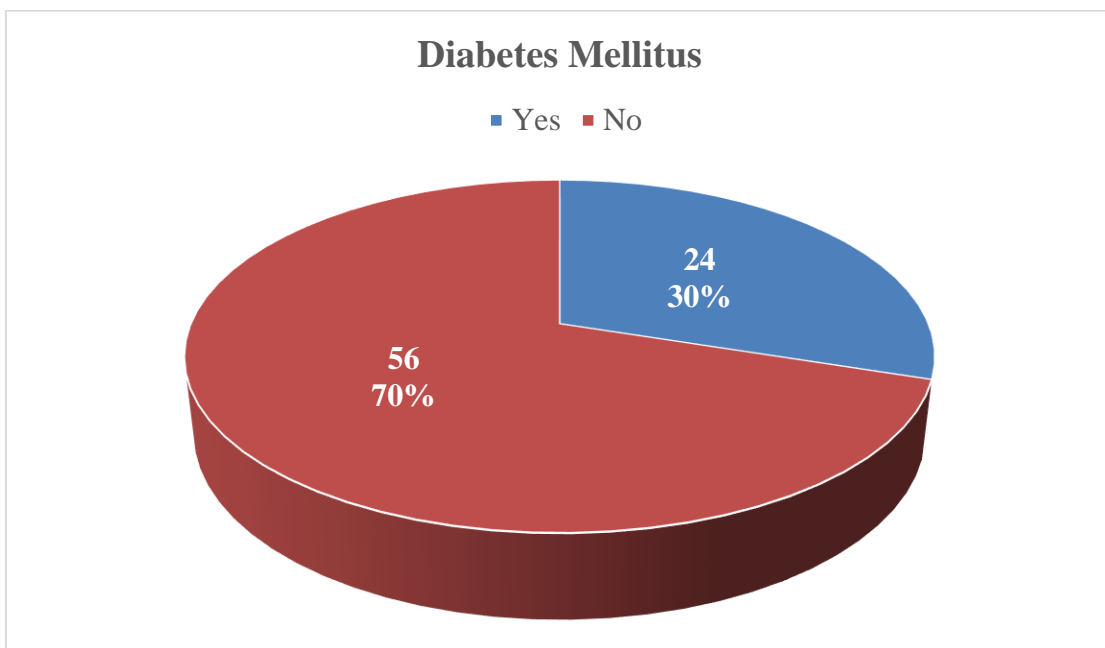
VI. Diabetes Mellitus

Among the subjects, 24 (30%) had Diabetes Mellitus

Table 9. Diabetes Mellitus

Diabetes Mellitus	Frequency	Percent
Yes	24	30.00
No	56	70.00
Total	80	100.00

Figure 21. Diabetes Mellitus



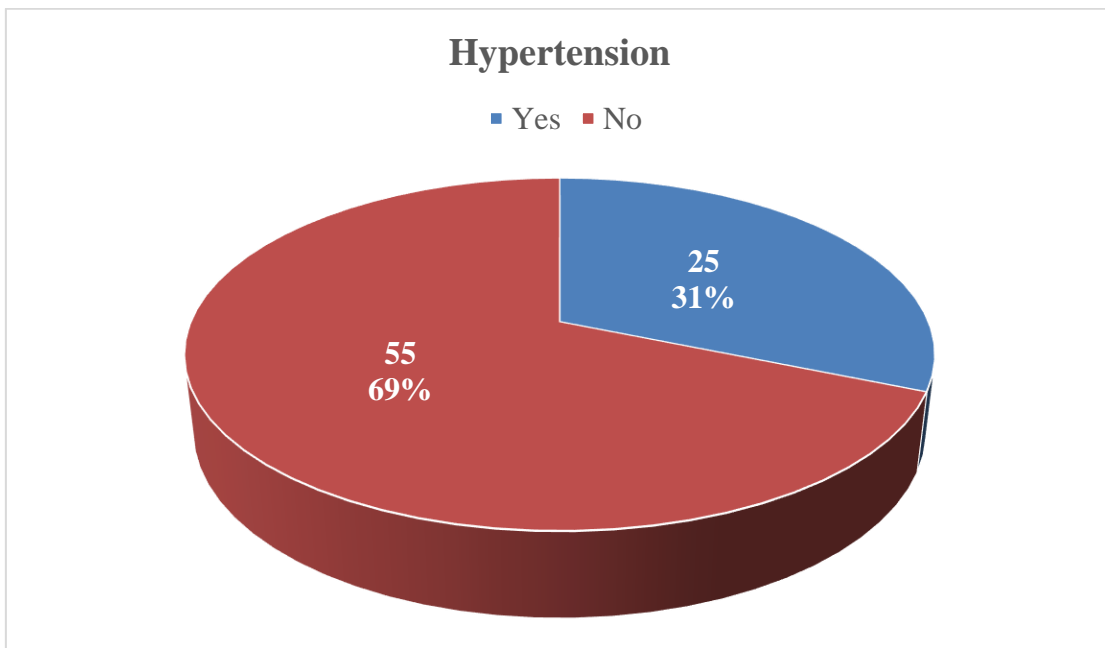
VII. Hypertension

Among the subjects, 25 (31.25%) had Hypertension

Table 10. Hypertension

Hypertension	Frequency	Percent
Yes	25	31.25
No	55	68.75
Total	80	100.00

Figure 22. Hypertension



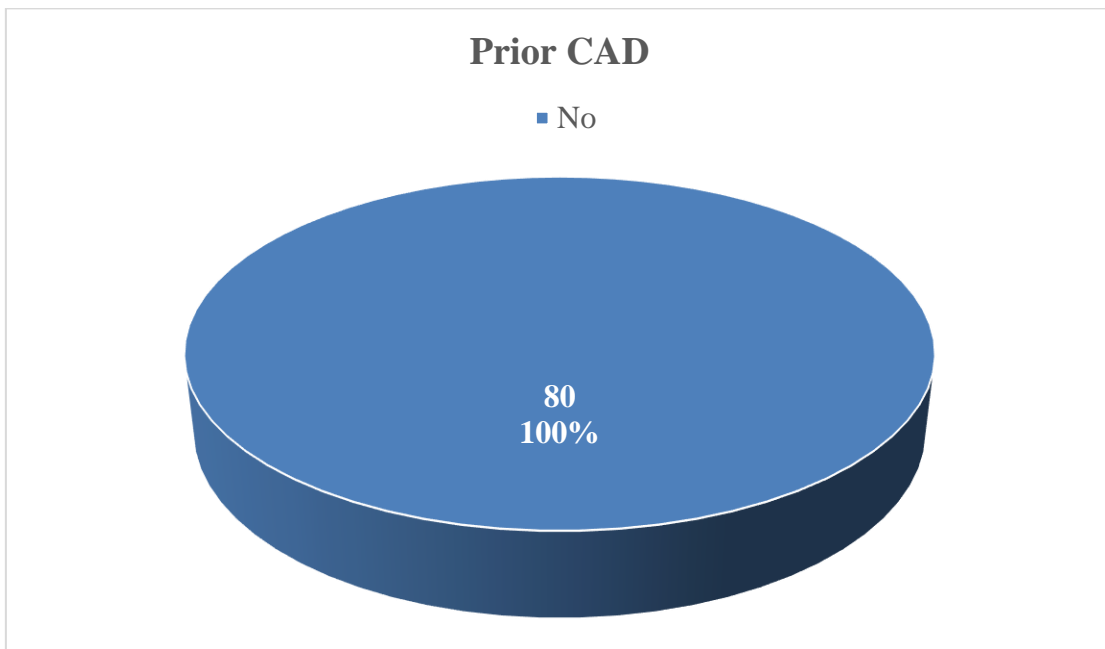
VIII. Prior CAD

Among the subjects, none had Prior CAD

Table 11. Prior CAD

Prior CAD	Frequency	Percent
No	80	100.00

Figure 23. Prior CAD



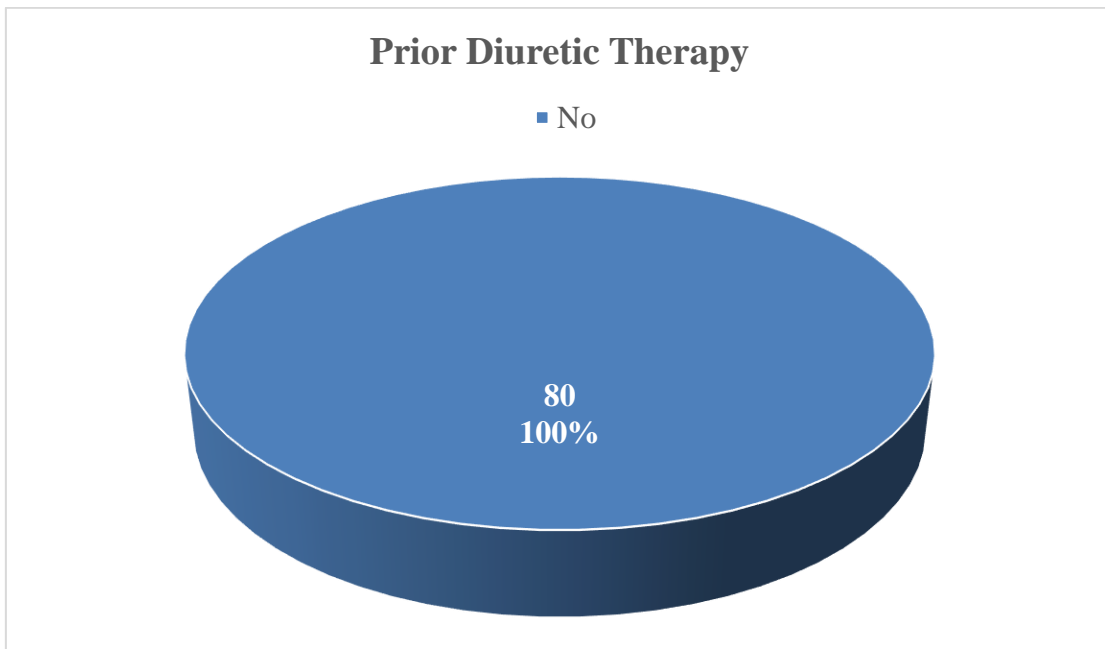
IX. Prior Diuretic Therapy

Among the subjects, none had Prior Diuretic Therapy

Table 12. Prior Diuretic Therapy

Prior Diuretic Therapy	Frequency	Percent
No	80	100.00

Figure 24. Prior Diuretic Therapy



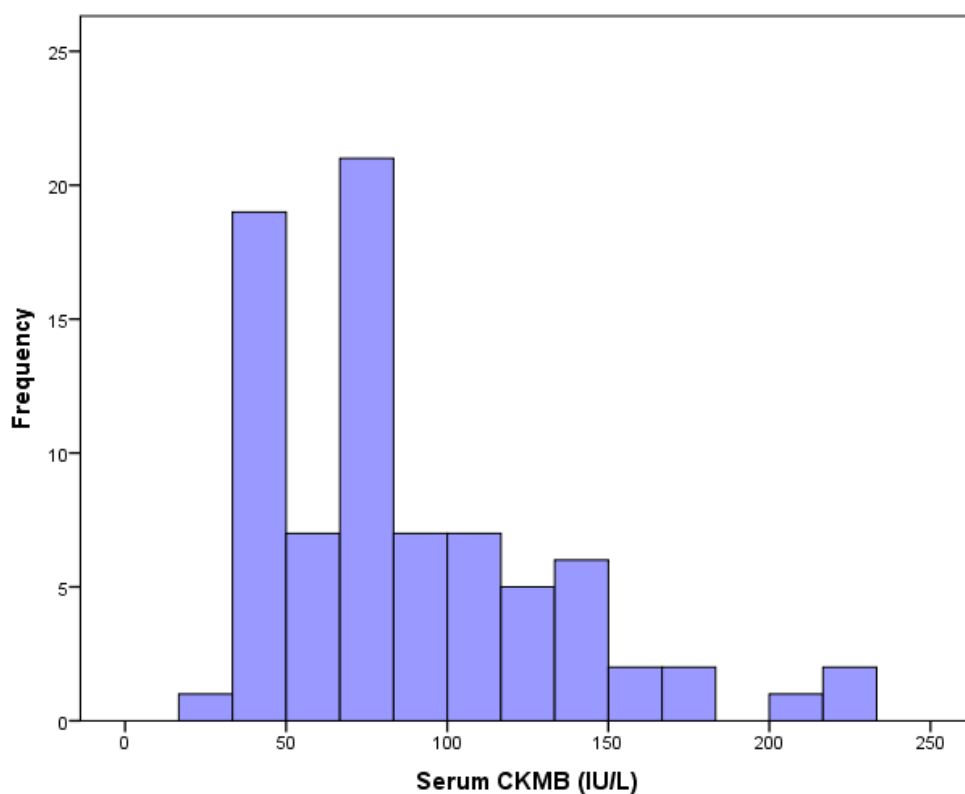
X. Serum CKMB (IU/L)

The mean Serum CKMB (IU/L) among the subjects was 87.36 (\pm 44.76) ranging from 31 to 221 IU/L.

Table 13. Serum CKMB (IU/L)

Serum CKMB (IU/L)	
Mean	87.36
Median	75.5
Std. Deviation	44.76
Range	190
Minimum	31
Maximum	221

Figure 25. Serum CKMB (IU/L)



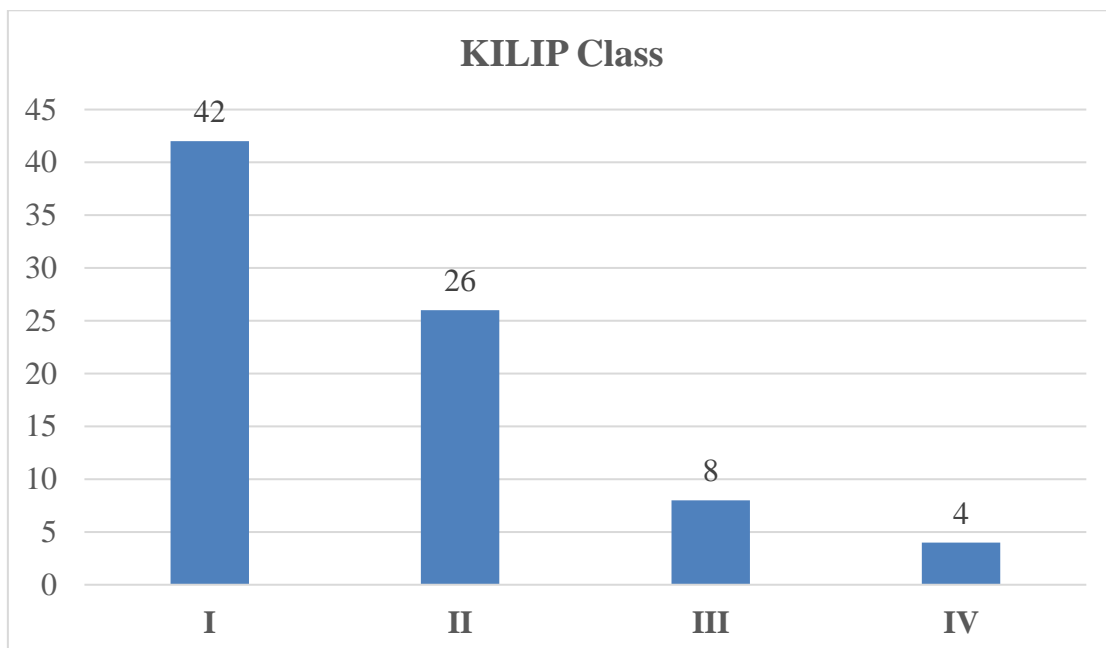
XI. KILIP Class

Among the subjects, 42 (52.5%) were in Class I, 26 (32.5%) were in Class II, 8 (10%) were in Class III and 4 (5%) were in Class IV.

Table 14. KILIP Class

KILIP Class	Frequency	Percent
I	42	52.50
II	26	32.50
III	8	10.00
IV	4	5.00
Total	80	100.00

Figure 26. KILIP Class



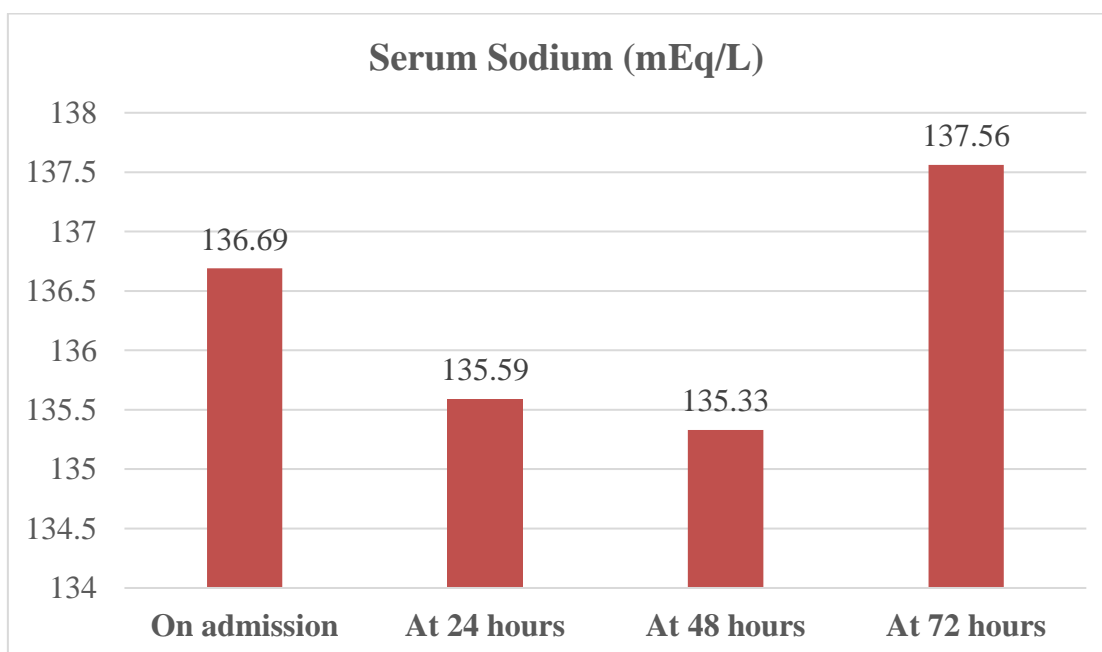
XII. Serum Sodium

The mean Serum Sodium on admission (mEq/L) among the subjects was 136.69 (\pm 3.12) mEq/L ranging from 126 to 143 mEq/L. The mean Serum Sodium at 24 hours (mEq/L) among the subjects was 135.59 (\pm 2.54) mEq/L ranging from 130 to 140 mEq/L. The mean Serum Sodium at 48 hours (mEq/L) among the subjects was 135.33 (\pm 2.39) mEq/L ranging from 130 to 140 mEq/L. The mean Serum Sodium at 72 hours (mEq/L) among the subjects was 137.56 (\pm 3.46) mEq/L ranging from 130 to 146 mEq/L.

Table 15. Serum Sodium

Serum Sodium (mEq/L)	N	Mean	S.D.	Minimum	Maximum
On admission	80	136.69	3.12	126.0	143.0
At 24 hours	80	135.59	2.54	130.0	140.0
At 48 hours	80	135.33	2.38	130.0	140.0
At 72 hours	80	137.56	3.46	130.0	146.0

Figure 27. Serum Sodium



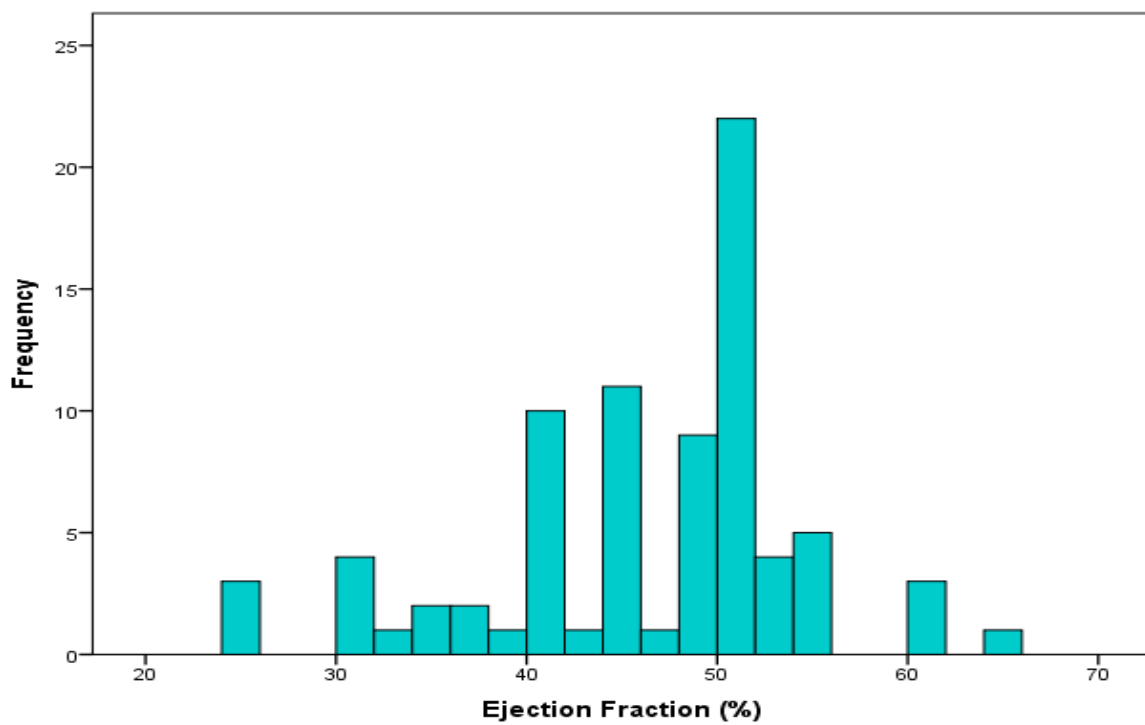
XIII. Ejection Fraction (%)

The mean Ejection Fraction (%) among the subjects was 45.56 (\pm 8.13) ranging from 25 to 65 %

Table 16. Ejection Fraction (%)

Ejection Fraction (%)	
Mean	45.56
Median	48
Std. Deviation	8.13
Range	40
Minimum	25
Maximum	65

Figure 28. Ejection Fraction (%)



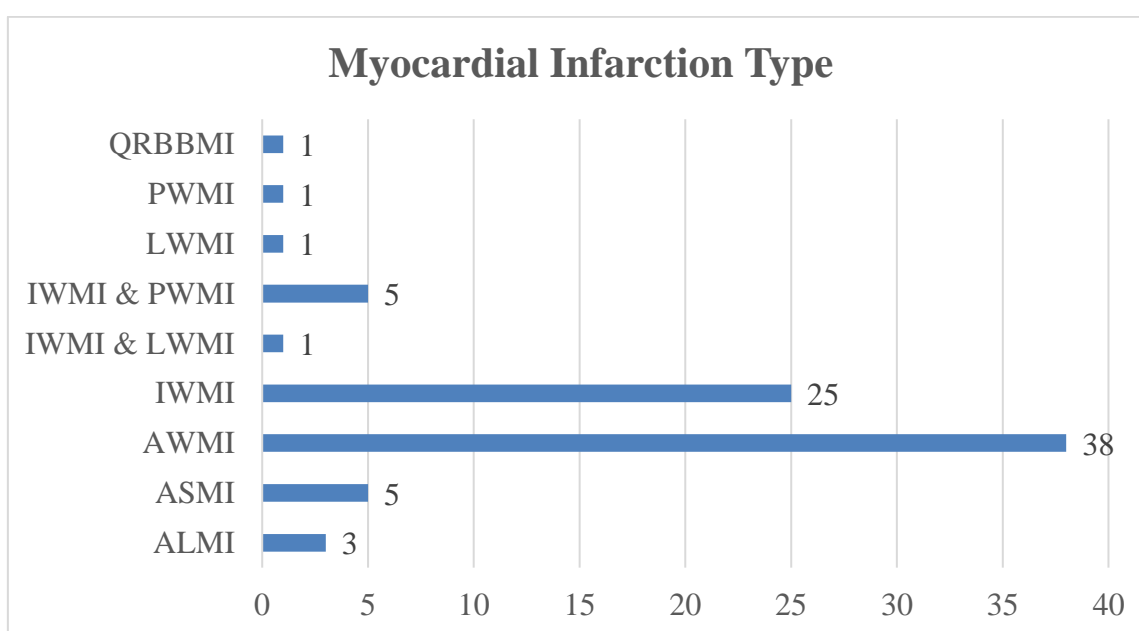
XIV. Myocardial Infarction Type

Among the subjects, 38 (47.5%) had AWMI, 25 (31.25%) had IWMI and 5 (6.25%) had ASMI

Table 17. Myocardial Infarction Type

Myocardial Infarction Type	Frequency	Percent
ALMI	3	3.75
ASMI	5	6.25
AWMI	38	47.50
IWMI	25	31.25
IWMI & LWMI	1	1.25
IWMI & PWMI	5	6.25
LWMI	1	1.25
PWMI	1	1.25
QRBBMI	1	1.25
Total	80	100.00

Figure 29. Myocardial Infarction Type



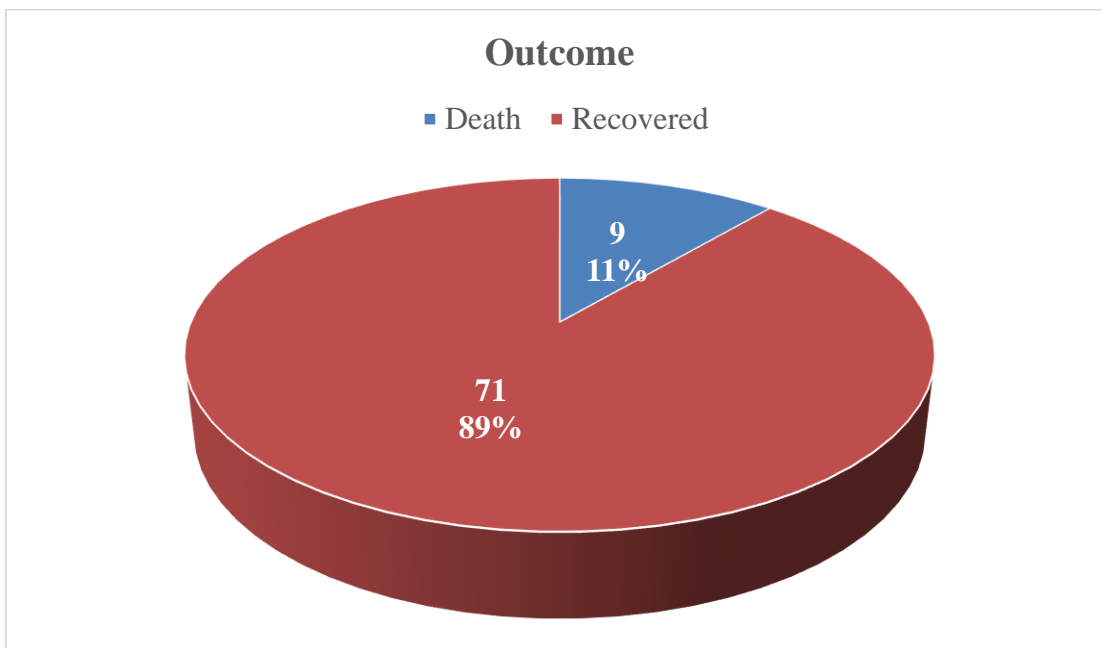
XV. Outcome

Among the subjects, 71 (88.75%) were Recovered and 9 (11.25%) had Death

Table 18. Outcome

Outcome	Frequency	Percent
Death	9	11.25
Recovered	71	88.75
Total	80	100.00

Figure 30. Outcome



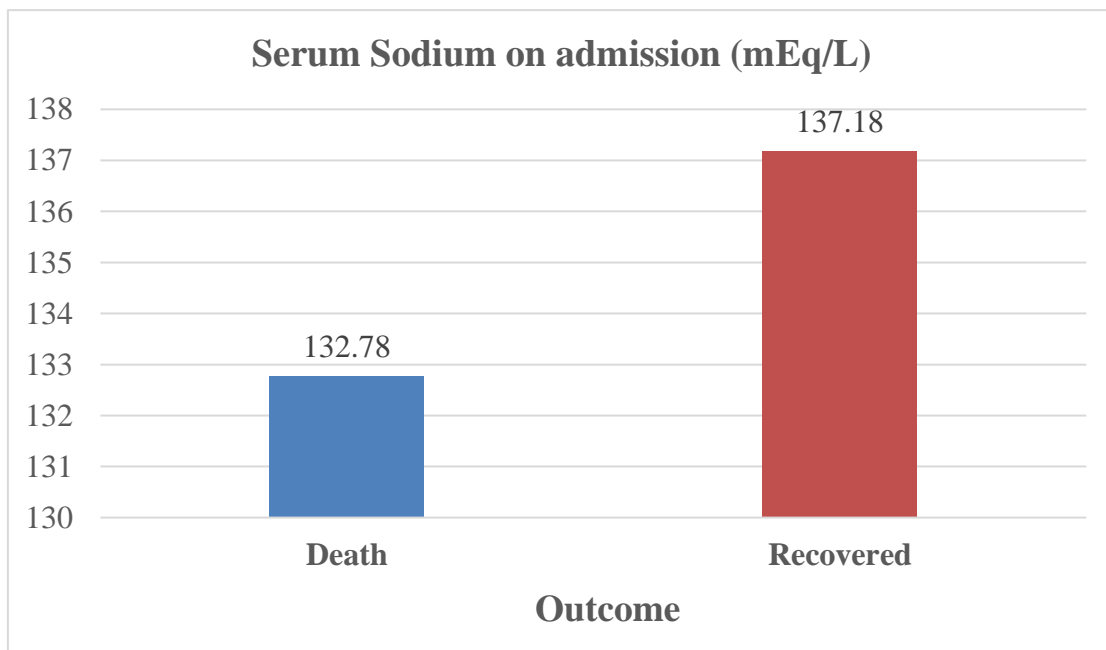
XVI. Serum Sodium on admission (mEq/L) with Outcome

The mean Serum Sodium on admission (mEq/L) among Death was 132.78 (± 4.71) which is lower by 4.41 and statistically significant compared to 137.18 (± 2.49) in Recovered

Table 19. Serum Sodium on admission (mEq/L) with Outcome

	Outcome	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Serum Sodium on admission (mEq/L)	Death	9	132.78	4.71	4.405	0.023
	Recovered	71	137.18	2.49		

Figure 31. Serum Sodium on admission (mEq/L) with Outcome



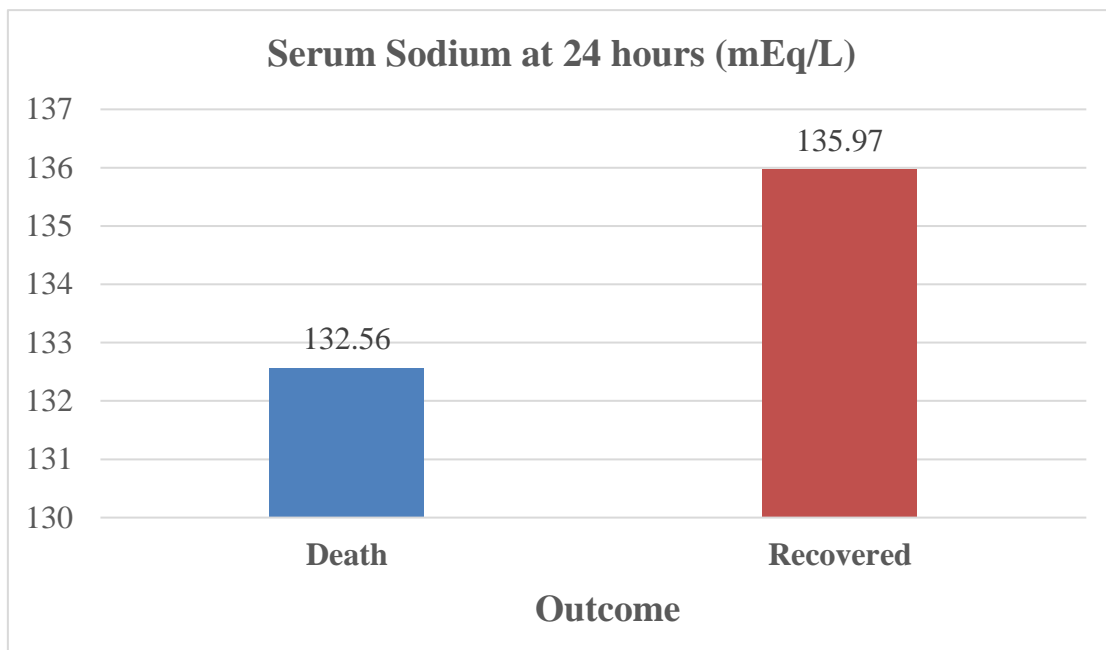
XVII. Serum Sodium at 24 hours (mEq/L) with Outcome

The mean Serum Sodium at 24 hours (mEq/L) among Death was 132.56 (\pm 2.3) which is lower by 3.42 and statistically significant compared to 135.97 (\pm 2.31) in Recovered

Table 20. Serum Sodium at 24 hours (mEq/L) with Outcome

	Outcome	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Serum Sodium at 24 hours (mEq/L)	Death	9	132.56	2.30	3.416	0.001
	Recovered	71	135.97	2.31		

Figure 32. Serum Sodium at 24 hours (mEq/L) with Outcome



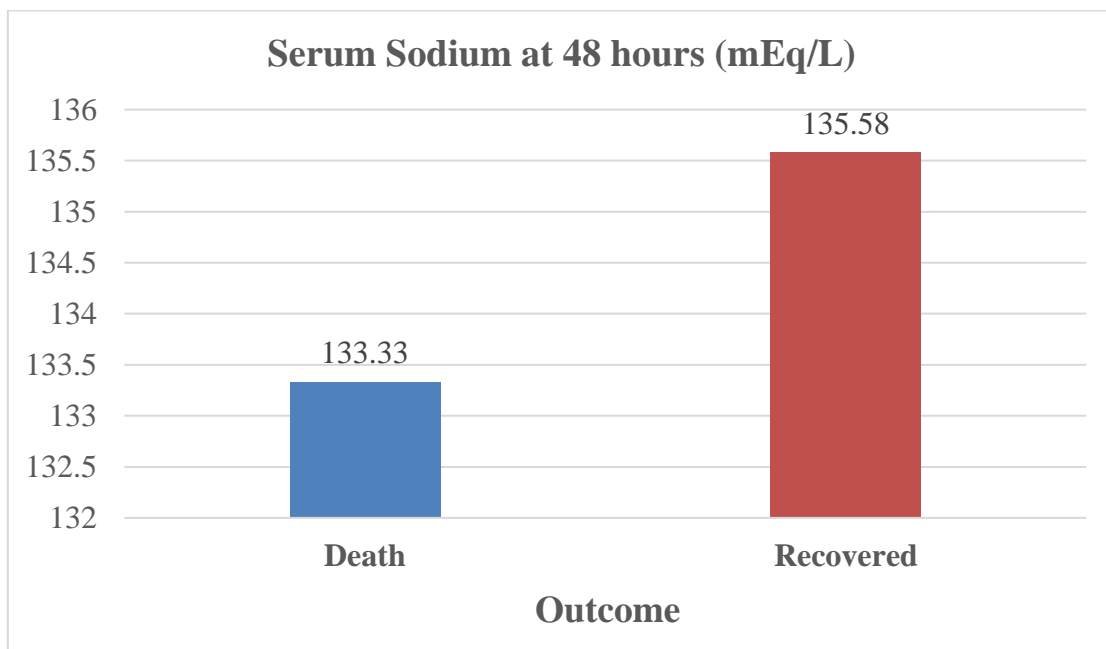
XVIII. Serum Sodium at 48 hours (mEq/L) with Outcome

The mean Serum Sodium at 48 hours (mEq/L) among Death was 133.33 (\pm 2.65) which is lower by 2.24 and statistically significant compared to 135.58 (\pm 2.25) in Recovered

Table 21. Serum Sodium at 48 hours (mEq/L) with Outcome

	Outcome	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Serum Sodium at 48 hours (mEq/L)	Death	9	133.33	2.65	2.244	0.007
	Recovered	71	135.58	2.25		

Figure 33. Serum Sodium at 48 hours (mEq/L) with Outcome



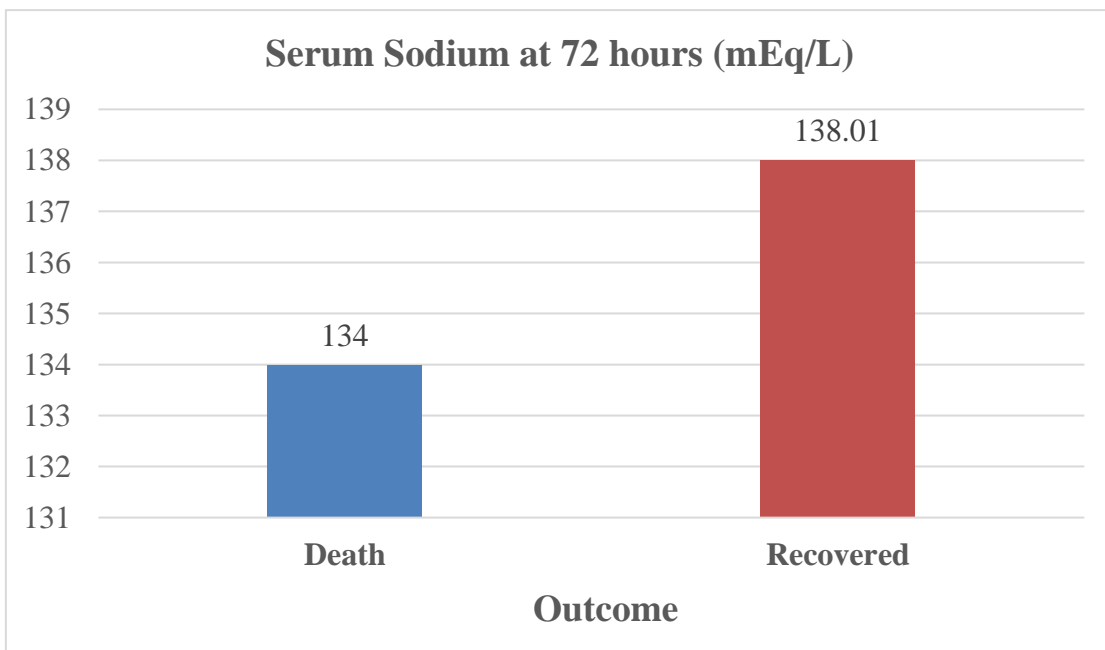
XIX. Serum Sodium at 72 hours (mEq/L) with Outcome

The mean Serum Sodium at 72 hours (mEq/L) among Death was 134 (± 2.65) which is lower by 4.01 and statistically significant compared to 138.01 (± 3.3) in Recovered

Table 22. Serum Sodium at 72 hours (mEq/L) with Outcome

	Outcome	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Serum Sodium at 72 hours (mEq/L)	Death	9	134.00	2.65	4.014	0.001
	Recovered	71	138.01	3.30		

Figure 34. Serum Sodium at 72 hours (mEq/L) with Outcome



XX. Serum Sodium with Myocardial Infarction Type

The mean Serum Sodium on admission (mEq/L) among Other MI was 136.91 which is higher than mean among Anterior Wall MI which was 136.71 followed by Inferior Wall MI with a mean of 136.58 but the difference was not statistically significant ($p > 0.05$).

The mean Serum Sodium at 24 hours (mEq/L) among Other MI was 136.18 which is higher than mean among Anterior Wall MI which was 135.79 followed by Inferior Wall MI with a mean of 135.13 but the difference was not statistically significant ($p > 0.05$).

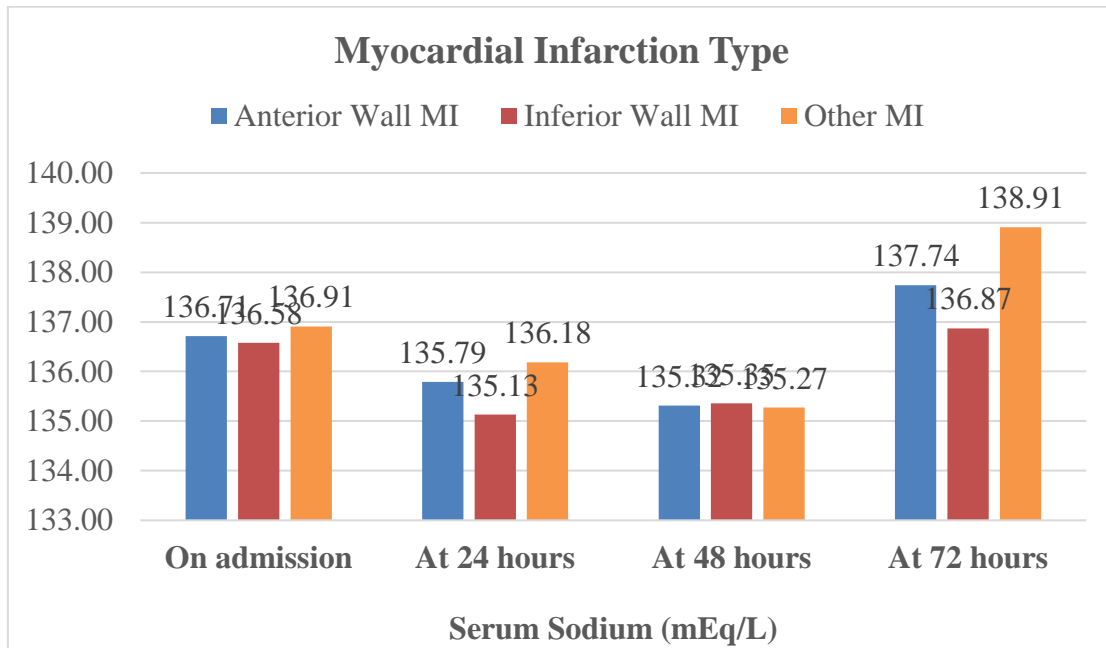
The mean Serum Sodium at 48 hours (mEq/L) among Inferior Wall MI was 135.35 which is higher than mean among Anterior Wall MI which was 135.32 followed by Other MI with a mean of 135.27 but the difference was not statistically significant ($p > 0.05$).

The mean Serum Sodium at 72 hours (mEq/L) among Other MI was 138.91 which is higher than mean among Anterior Wall MI which was 137.74 followed by Inferior Wall MI with a mean of 136.87 but the difference was not statistically significant ($p > 0.05$).

Table 23. Serum Sodium with Myocardial Infarction Type

Serum Sodium (mEq/L)	Myocardial Infarction Type			ANOVA p value
	Anterior Wall MI	Inferior Wall MI	Other MI	
On admission	136.7 (± 3.1)	136.6 (± 3.2)	136.9 (± 3)	0.955
At 24 hours	135.8 (± 2.4)	135.1 (± 2.7)	136.2 (± 2.6)	0.401
At 48 hours	135.3 (± 2.8)	135.4 (± 2.2)	135.3 (± 1.5)	0.995
At 72 hours	137.7 (± 3.7)	136.9 (± 2.8)	138.9 (± 4.3)	0.225

Figure 35. Serum Sodium with Myocardial Infarction Type



XXI. Serum Sodium with KILIP Class

Class I had higher mean of Serum Sodium on admission (mEq/L) with 137.02 followed by Class II with 136.62 and least in Class IV with 134 but the difference is not statistically significant

Class II had higher mean of Serum Sodium at 24 hours (mEq/L) with 135.92 followed by Class I with 135.76 and least in Class IV with 131.5 and the difference is statistically significant

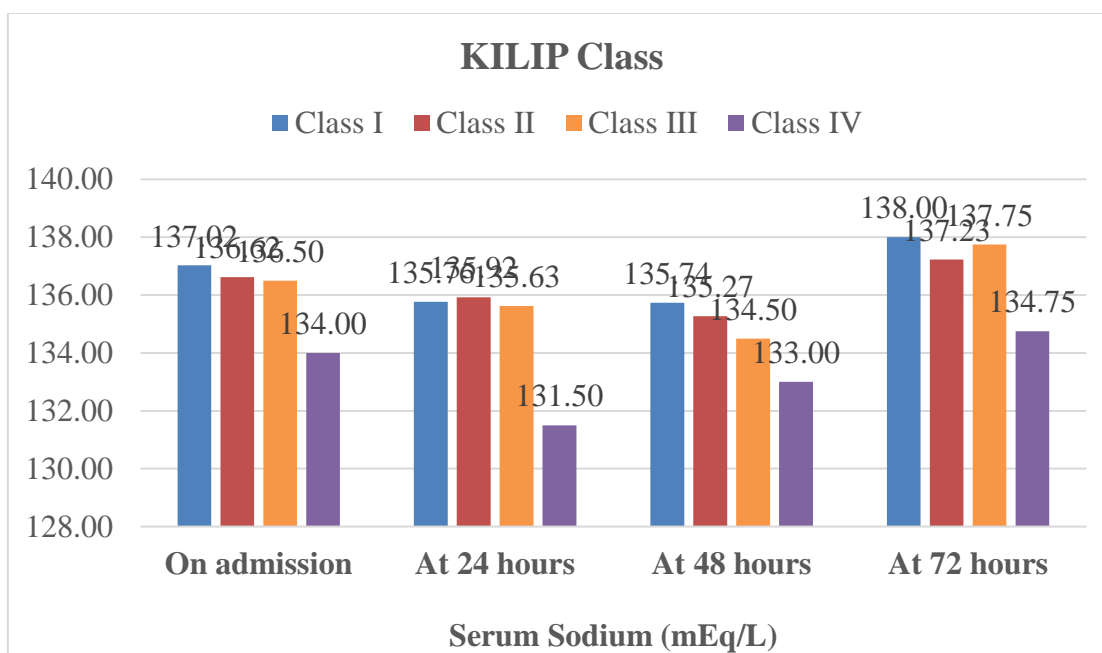
Class I had higher mean of Serum Sodium at 48 hours (mEq/L) with 135.74 followed by Class II with 135.27 and least in Class IV with 133 but the difference is not statistically significant

Class I had higher mean of Serum Sodium at 72 hours (mEq/L) with 138 followed by Class III with 137.75 and least in Class IV with 134.75 but the difference is not statistically significant

Table 24. Serum Sodium with KILIP Class

Serum Sodium (mEq/L)	KILIP Class				ANOVA p value
	I	II	III	IV	
On admission	137 (± 2.8)	136.6 (± 2.6)	136.5 (± 4.8)	134 (± 5.4)	0.324
At 24 hours	135.8 (± 2.4)	135.9 (± 2)	135.6 (± 3.6)	131.5 (± 1.9)	0.009
At 48 hours	135.7 (± 2.6)	135.3 (± 1.8)	134.5 (± 2.3)	133 (± 2)	0.108
At 72 hours	138 (± 3.4)	137.2 (± 3.7)	137.8 (± 2.9)	134.8 (± 3.4)	0.315

Figure 36. Serum Sodium with KILIP Class



XXII. Correlation of Serum Sodium with CKMB

There was no significant correlation between serum sodium and CKMB levels from admission to 72 hours.

Table 25. Correlation of Serum Sodium with CKMB

Serum Sodium (mEq/L)	N	Correlation coefficient "r"	p value
On admission	80	0.038	0.737
At 24 hours	80	0.163	0.148
At 48 hours	80	0.064	0.573
At 72 hours	80	0.015	0.897

XXIII. Correlation of Serum Sodium with Ejection Fraction

Serum Sodium on admission (mEq/L) has a significant positive correlation with Ejection Fraction (%) with a correlation coefficient of 0.357. Serum Sodium at 24 hours (mEq/L) has a significant positive correlation with Ejection Fraction (%) with a correlation coefficient of 0.374. Serum Sodium at 48 hours (mEq/L) has a significant positive correlation with Ejection Fraction (%) with a correlation coefficient of 0.483. Serum Sodium at 72 hours (mEq/L) has a significant positive correlation with Ejection Fraction (%) with a correlation coefficient of 0.288.

Table 26. Correlation of Serum Sodium with Ejection Fraction

Serum Sodium (mEq/L)	N	Correlation coefficient "r"	p value
On admission	80	0.357	0.001
At 24 hours	80	0.374	0.001
At 48 hours	80	0.483	0.001
At 72 hours	80	0.288	0.001

XXIV. ROC for predicting Death among STEMI using Serum Sodium

The area under the curve for Serum Sodium on admission (mEq/L) in predicting Death is 0.807 (0.663 - 0.951). The area under the curve for Serum Sodium at 24 hours (mEq/L) in predicting Death is 0.855 (0.739 - 0.972). The area under the curve for Serum Sodium at 48 hours (mEq/L) in predicting Death is 0.751 (0.564 - 0.938). The area under the curve for Serum Sodium at 72 hours (mEq/L) in predicting Death is 0.833 (0.731 - 0.936).

The cut off of Serum Sodium on admission (mEq/L) for predicting Death is 129.5 which had a sensitivity of 33.33%, specificity of 100%, positive predictive value of 100%, negative predictive value of 92.21% and a diagnostic accuracy of 92.5%.

The cut off of Serum Sodium at 24 hours (mEq/L) for predicting Death is 131 which had a sensitivity of 33.33%, specificity of 97.18%, positive predictive value of 60%, negative predictive value of 92% and a diagnostic accuracy of 90%.

The cut off of Serum Sodium at 48 hours (mEq/L) for predicting Death is 132.5 which had a sensitivity of 44.44%, specificity of 87.32%, positive predictive value of 30.77%, negative predictive value of 92.54% and a diagnostic accuracy of 82.5%.

The cut off of Serum Sodium at 72 hours (mEq/L) for predicting Death is 130.5 which had a sensitivity of 22.22%, specificity of 100%, positive predictive value of 100%, negative predictive value of 91.03% and a diagnostic accuracy of 91.25%.

Table 27. ROC for predicting Death among STEMI using Serum Sodium

Serum Sodium (mEq/L)	Area under the curve	95% Confidence Interval		p value
		Lower Bound	Upper Bound	
On admission	0.807	0.663	0.951	0.003
At 24 hours	0.855	0.739	0.972	0.001
At 48 hours	0.751	0.564	0.938	0.015
At 72 hours	0.833	0.731	0.936	0.001

Figure 37. ROC for predicting Death among STEMI using Serum Sodium

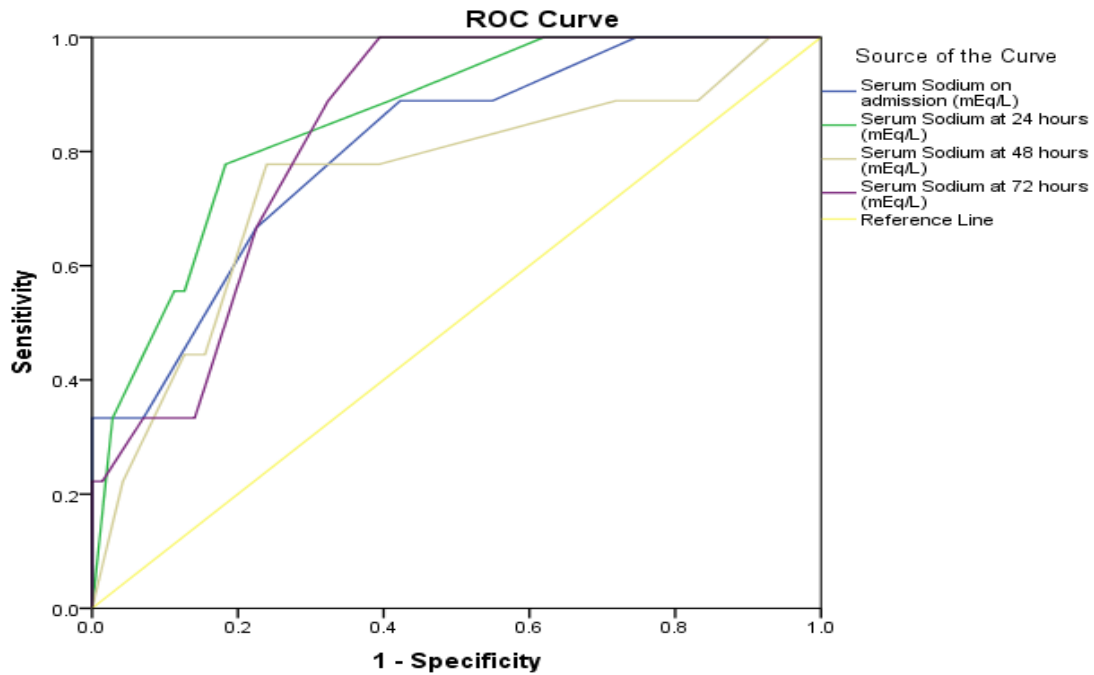


Table 28. Accuracy values of different cutoffs of Serum Sodium (mEq/L) on admission for prediction of prognosis.

Cut off	Sensitivity	Specificity	PPV	NPV	Accuracy
Serum Sodium (mEq/L) on admission					
125	0.00%	100.00%	#DIV/0!	88.75%	88.75%
127	22.22%	100.00%	100.00%	91.03%	91.25%
129.5	33.33%	100.00%	100.00%	92.21%	92.50%
131.5	33.33%	98.59%	75.00%	92.11%	91.25%
133.5	33.33%	92.96%	37.50%	91.67%	86.25%
135.5	66.67%	77.46%	27.27%	94.83%	76.25%
136.5	88.89%	57.75%	21.05%	97.62%	61.25%
137.5	88.89%	45.07%	17.02%	96.97%	50.00%
138.5	100.00%	25.35%	14.52%	100.00%	33.75%
139.5	100.00%	21.13%	13.85%	100.00%	30.00%
140.5	100.00%	7.04%	12.00%	100.00%	17.50%
141.5	100.00%	5.63%	11.84%	100.00%	16.25%
142.5	100.00%	1.41%	11.39%	100.00%	12.50%
144	100.00%	0.00%	11.25%	100.00%	11.25%

Table 29. Accuracy values of different cutoffs of Serum Sodium (mEq/L) at 24 hrs for prediction of prognosis.

Cut off	Sensitivity	Specificity	PPV	NPV	Accuracy
Serum Sodium (mEq/L) at 24 hours					
129	0.00%	100.00%	#DIV/0!	88.75%	88.75%
131	33.33%	97.18%	60.00%	92.00%	90.00%
132.5	55.56%	88.73%	38.46%	94.03%	85.00%
133.5	55.56%	87.32%	35.71%	93.94%	83.75%
134.5	77.78%	81.69%	35.00%	96.67%	81.25%
135.5	88.89%	59.15%	21.62%	97.67%	62.50%
136.5	100.00%	38.03%	16.98%	100.00%	45.00%
137.5	100.00%	29.58%	15.25%	100.00%	37.50%
138.5	100.00%	11.27%	12.50%	100.00%	21.25%
139.5	100.00%	7.04%	12.00%	100.00%	17.50%
141	100.00%	0.00%	11.25%	100.00%	11.25%

Table 30. Accuracy values of different cutoffs of Serum Sodium (mEq/L) at 48 hrs for prediction of prognosis.

Cut off	Sensitivity	Specificity	PPV	NPV	Accuracy
Serum Sodium (mEq/L) at 48 hours					
129	0.00%	100.00%	0.00%	88.75%	88.75%
131	22.22%	95.77%	40.00%	90.67%	87.50%
132.5	44.44%	87.32%	30.77%	92.54%	82.50%
133.5	44.44%	84.51%	26.67%	92.31%	80.00%
134.5	77.78%	76.06%	29.17%	96.43%	76.25%
135.5	77.78%	60.56%	20.00%	95.56%	62.50%
136.5	88.89%	28.17%	13.56%	95.24%	35.00%
137.5	88.89%	16.90%	11.94%	92.31%	25.00%
138.5	100.00%	7.04%	12.00%	100.00%	17.50%
139.5	100.00%	5.63%	11.84%	100.00%	16.25%
141	100.00%	0.00%	11.25%	100.00%	11.25%

Table 31. Accuracy values of different cutoffs of Serum Sodium (mEq/L) at 72 hrs for prediction of prognosis.

Cut off	Sensitivity	Specificity	PPV	NPV	Accuracy
Serum Sodium (mEq/L) at 72 hours					
129	0.00%	100.00%	#DIV/0!	88.75%	88.75%
130.5	22.22%	100.00%	100.00%	91.03%	91.25%
131.5	22.22%	98.59%	66.67%	90.91%	90.00%
132.5	33.33%	92.96%	37.50%	91.67%	86.25%
133.5	33.33%	88.73%	27.27%	91.30%	82.50%
134.5	33.33%	85.92%	23.08%	91.04%	80.00%
135.5	66.67%	77.46%	27.27%	94.83%	76.25%
136.5	88.89%	67.61%	25.81%	97.96%	70.00%
137.5	100.00%	60.56%	24.32%	100.00%	65.00%
138.5	100.00%	39.44%	17.31%	100.00%	46.25%
139.5	100.00%	38.03%	16.98%	100.00%	45.00%
141	100.00%	19.72%	13.64%	100.00%	28.75%
142.5	100.00%	5.63%	11.84%	100.00%	16.25%
143.5	100.00%	4.23%	11.69%	100.00%	15.00%
145	100.00%	1.41%	11.39%	100.00%	12.50%
147	100.00%	0.00%	11.25%	100.00%	11.25%

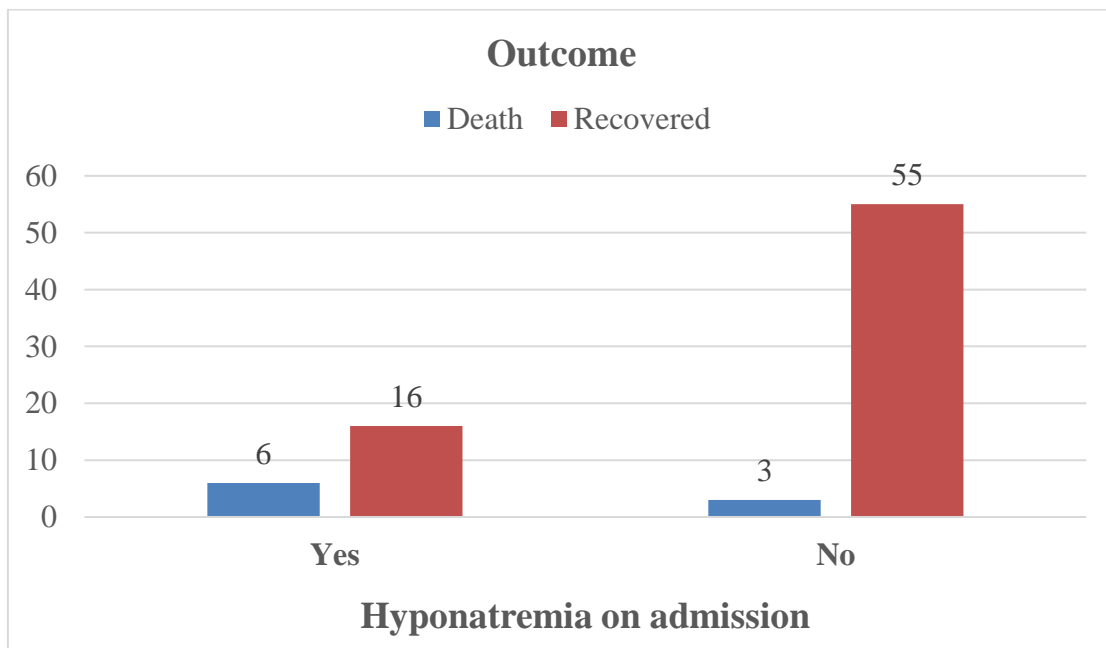
XXV. Comparison of Hyponatremia on admission with the Outcome

27.27% of the subjects with Hyponatremia on admission had Death which is higher compared to those without Hyponatremia on admission of whom 5.17% had Death and the difference was statistically significant ($p < 0.05$)

Table 32. Comparison of Hyponatremia on admission with the Outcome

Hyponatremia on admission	Outcome		Total	Fisher exact p value
	Death	Recovered		
Yes	6 (27.27%)	16 (72.72%)	22 (100%)	0.01
No	3 (5.17%)	55 (94.82%)	58 (100%)	
Total	9 (11.25%)	71 (88.75%)	80 (100%)	

Figure 38. Comparison of Hyponatremia on admission with the Outcome



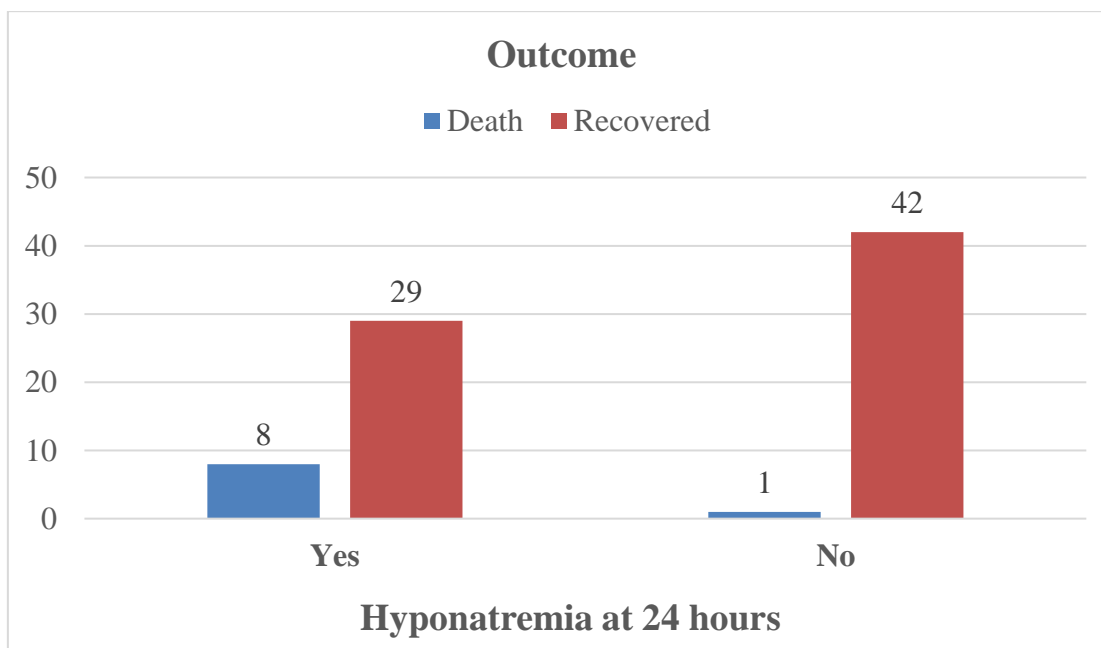
XXVI. Comparison of Hyponatremia at 24 hours with the Outcome

21.6% of the subjects with Hyponatremia at 24 hours had Death which is higher compared to those without Hyponatremia at 24 hours of whom 2.3% had Death and the difference was statistically significant ($p < 0.05$)

Table 33. Comparison of Hyponatremia at 24 hours with the Outcome

Hyponatremia at 24 hours	Outcome		Total	Fisher exact p value
	Death	Recovered		
Yes	8 (21.62%)	29 (78.37%)	37 (100%)	0.008
No	1 (2.32%)	42 (97.67%)	43 (100%)	
Total	9 (11.25%)	71 (88.75%)	80 (100%)	

Figure 39. Comparison of Hyponatremia at 24 hours with the Outcome



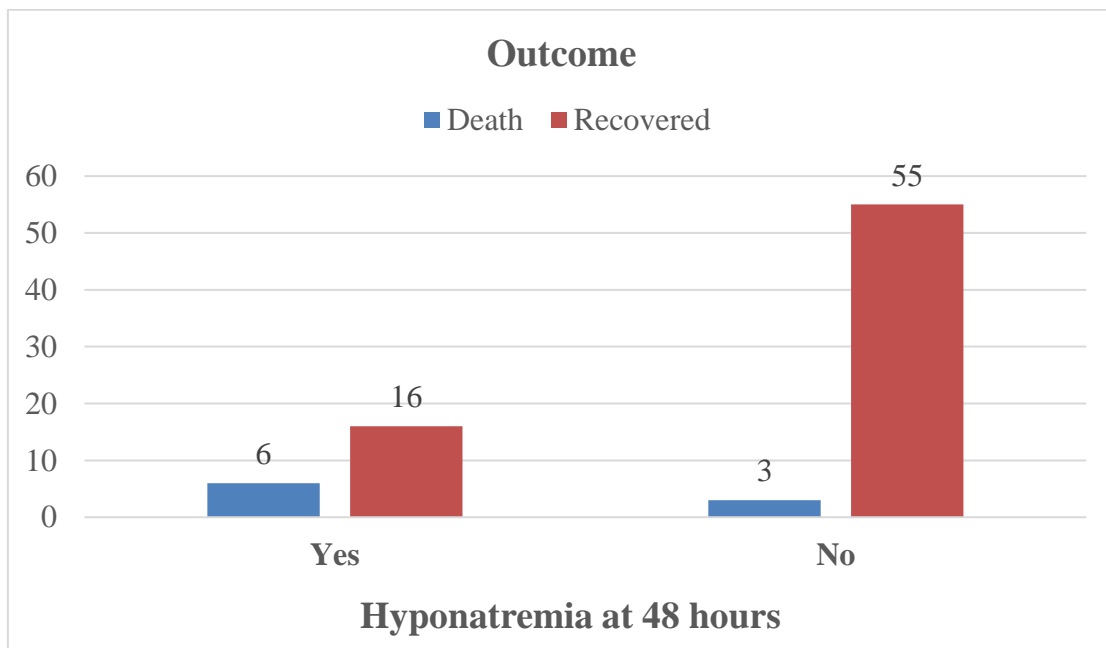
XXVII. Comparison of Hyponatremia at 48 hours with the Outcome

27.27% of the subjects with Hyponatremia at 48 hours had Death which is higher compared to those without Hyponatremia at 48 hours of whom 5.17% had Death and the difference was statistically significant ($p < 0.05$)

Table 34. Comparison of Hyponatremia at 48 hours with the Outcome

Hyponatremia at 48 hours	Outcome		Total	Fisher exact p value
	Death	Recovered		
Yes	6 (27.27%)	16 (72.72%)	22 (100%)	0.01
No	3 (5.17%)	55 (94.82%)	58 (100%)	
Total	9 (11.25%)	71 (88.75%)	80 (100%)	

Figure 40. Comparison of Hyponatremia at 48 hours with the Outcome



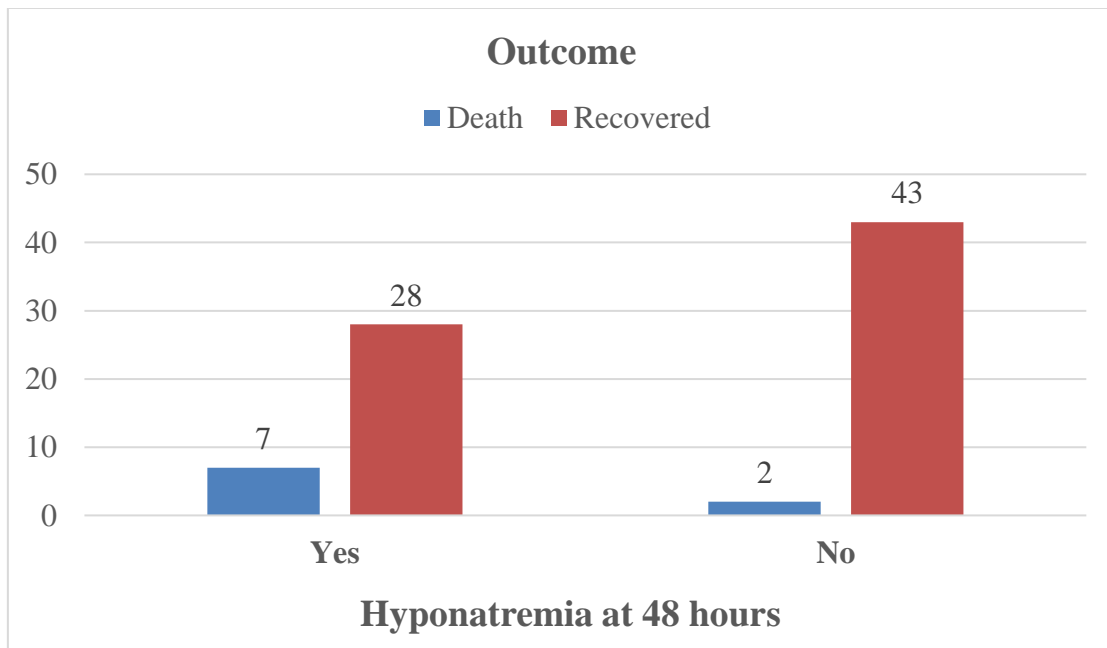
XXVIII. Comparison of Hyponatremia at 72 hours with the Outcome

20% of the subjects with Hyponatremia at 72 hours had Death which is higher compared to those without Hyponatremia at 72 hours of whom 4.44% had Death and the difference was statistically significant ($p < 0.05$)

Table 35. Comparison of Hyponatremia at 72 hours with the Outcome

Hyponatremia at 72 hours	Outcome		Total	Fisher exact p value
	Death	Recovered		
Yes	7 (20%)	28 (80%)	35 (100%)	0.029
No	2 (4.44%)	43 (95.55%)	45 (100%)	
Total	9 (11.25%)	71 (88.75%)	80 (100%)	

Figure 41. Comparison of Hyponatremia at 72 hours with the Outcome



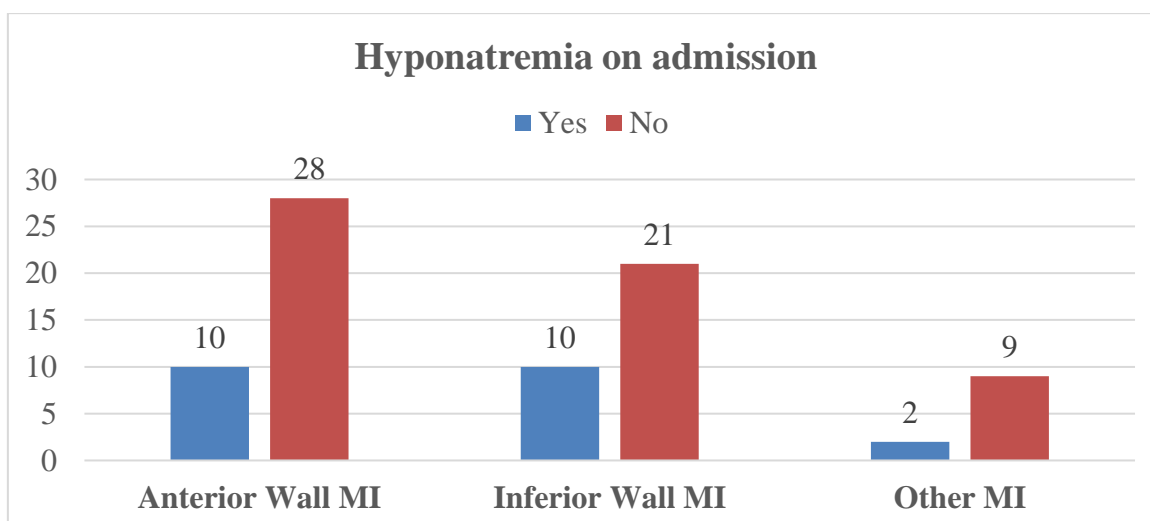
XXIX. Comparison of Myocardial Infarction Type with the Hyponatremia on admission

Comparing the Myocardial Infarction Type with Hyponatremia on admission distribution, Inferior Wall MI had higher proportion of Hyponatremia on admission with 32.25% followed by Anterior Wall MI with 26.31% and in Other MI with 18.18%. The difference in Hyponatremia on admission distribution between different Myocardial Infarction Type was not statistically significant ($p > 0.05$).

Table 36. Comparison of Myocardial Infarction Type with the Hyponatremia on admission

Myocardial Infarction Type	Hyponatremia on admission		Total	Fisher exact p value
	Yes	No		
Anterior Wall MI	10 (26.31%)	28 (73.68%)	38 (100%)	0.137
Inferior Wall MI	10 (32.25%)	21 (67.74%)	31 (100%)	
Other MI	2 (18.18%)	9 (81.81%)	11 (100%)	
Total	22 (27.5%)	58 (72.5%)	80 (100%)	

Figure 42. Comparison of Myocardial Infarction Type with the Hyponatremia on admission



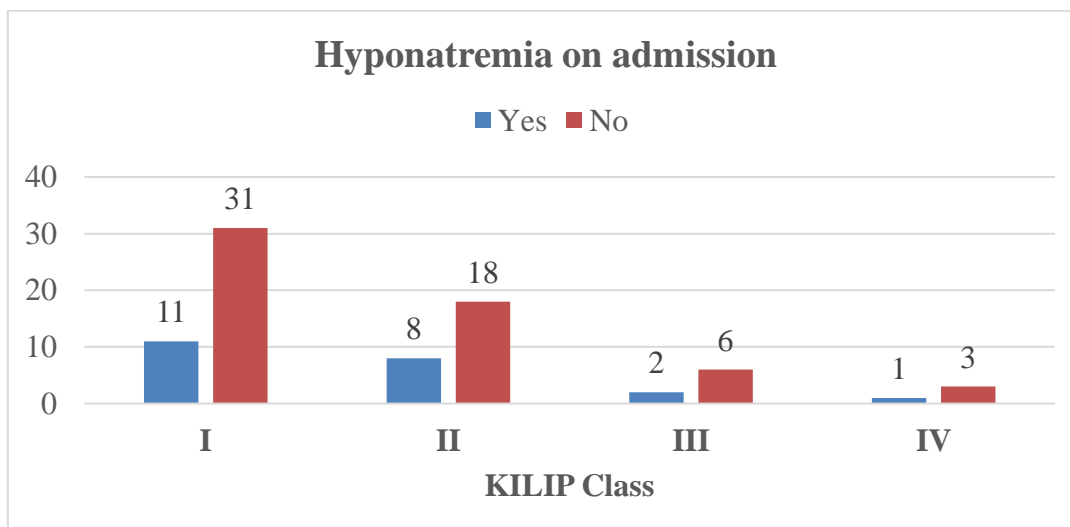
XXX. Comparison of KILIP Class with the Hyponatremia on admission

Comparing the KILIP Class with Hyponatremia on admission distribution, Class II had higher proportion of Hyponatremia on admission with 30.76% followed by Class I with 26.19% and in Class III with 25%. The difference in Hyponatremia on admission between different KILIP Class was not statistically significant ($p > 0.05$).

Table 37. Comparison of KILIP Class with the Hyponatremia on admission

KILIP Class	Hyponatremia on admission		Total	Fisher exact p value
	Yes	No		
I	11 (26.19%)	31 (73.8%)	42 (100%)	0.115
II	8 (30.76%)	18 (69.23%)	26 (100%)	
III	2 (25%)	6 (75%)	8 (100%)	
IV	1 (25%)	3 (75%)	4 (100%)	
Total	22 (27.5%)	58 (72.5%)	80 (100%)	

Figure 43. Comparison of KILIP Class with the Hyponatremia on admission



XXXI. Binomial Logistic Regression for predicting Death in STEMI

Hyponatremia on admission had an odds of 8.26 times of getting death in STEMI compared to normal sodium level on admission. For each increase in KILIP class, the odds of getting death among STEMI increases by 2.33 times.

Table 38. Binomial Logistic Regression for predicting Death in STEMI

Variables	B	Std. Error	Adjusted Odds Ratio (95% C.I.)	p value
Hyponatremia	2.112	0.818	8.26 (1.66 - 41.1)	0.010
KILIP Class	0.846	0.415	2.33 (1.03 - 5.26)	0.042
Constant	-4.602	1.153	0.01 (0 - 0)	0.000

6 DISCUSSION

Following a Myocardial Infarction, hyponatremia is frequent, and a rise in plasma sodium contents coincides with clinical recovery.(13,14) Hyponatremia was present in about one third of the congestive heart failure patients.(15) It has been demonstrated that in patients with heart failure and STEMI, it is a predictor of cardiovascular death.

Acute STEMI patients who presented with hyponatremia at the time of admission and within 72 hrs of admission had high mortality and so serum sodium level (hyponatremia) is a prognostic indicator. Patient at risk can be identified by a simple marker of plasma sodium level.

The main objective of the study is to study the prognostic importance of serum sodium level (hyponatremia). This is a Cross sectional analytical study, among 80 patients admitted with acute STEMI under the Department of General Medicine, Thanjavur Medical College and hospital, Thanjavur. Relevant history, clinical findings, biochemical measurements especially the serum sodium levels were compared with the outcomes. Prognostic value of the serum sodium levels were analyzed.

Age: Hyponatremia was found to be significantly correlated with age and gender by **RC Hawkins et al.**(56) In our study, the mean Age (years) among the subjects was 55.33 (\pm 12.62) ranging from 25 to 82 years. Among the subjects, 46 (57.5%) were in < 60 years and 34 (42.5%) were in > 60 years. Among the subjects, 62 (77.5%) were Males and 18 (22.5%) were Females

Smoker: Among the subjects, 50 (62.5%) were Smoker and 30 (37.5%) were Alcoholic. Smoking may be directly associated with the onset of hyponatremia in patients, but it also significantly affects the likelihood of positive results. (57–59)

Diabetes: In this study, 24 (30%) had Diabetes Mellitus. With uncontrolled diabetes and hyperglycemia, hyponatremia is highly frequent and results in electrolyte loss in the urine.(60–62)

Hypertension: In this study, 25 (31.25%) had Hypertension. According to studies, fatality rates were greater in patients with hypertensive STEMI and were more common in older women. (63,64)

Serum CKMB (IU/L): The mean Serum CKMB (IU/L) among the subjects was 87.36 (\pm 44.76) ranging from 31 to 221 IU/L. Although several studies have noted the coexistence of hyponatremia and CKMB levels, it is unclear how they are directly related.(56)

Killip classification: A prognostic indication of acute myocardial infarction is the Killip classification.(65–67) In this study, 42 (52.5%) were in Class I, 26 (32.5%) were in Class II, 8 (10%) were in Class III and 4 (5%) were in Class IV.

Ejection Fraction (%): While **JJ Park et al.** discovered a relationship between hyponatremia, reduced ejection fraction, and higher mortality rate, **Y Cavusoglu et al.** and **C Bavishi et al.** observed a relationship between hyponatremia, reduced ejection fraction, and a decrease in ejection fraction.(68–70) The mean Ejection Fraction (%) among the subjects was 45.56 (\pm 8.13) ranging from 25 to 65 %.

Myocardial Infarction Type: Among the subjects, 38 (47.5%) had AAMI, 25 (31.25%) had IWMI and 5 (6.25%) had ASMI.

Outcome: Among the subjects, 71 (88.75%) were Recovered and 9 (11.25%) had Death.

Suprabhat Giri et al. found that the prevalence of hyponatremia (less than 135 mmol/L) was 29%. Hyponatremia was found to be significantly correlated with both hospital mortality and 7-day MACE in multivariate analysis.(53)

Alexander Goldberg et al. observed that, Hyponatremia (135 mmol/L) was present in 12.5% of patients at admission and 19.9% of patients after 72 hours. Hyponatremia (135 mmol/L) upon admission was linked to a 2.0 odds ratio for 30-day death; (95 percent CI: 1.0 to 3.9). Hyponatremia (130 mmol/L) upon admission was associated with a 30-day mortality rate with an odds ratio of 3.4 (95 percent confidence interval: 1.5 to 7.8).(46)

Serum Sodium with Outcome: The mean Serum Sodium at admission, at 24 hours, at 48 hours and 72 hours (mEq/L) among Death was lower and statistically significant compared to the Recovered. **Vraj Shah et al.** noted that hyponatremia worsens short-term and long-term mortality, rehospitalization rates, lengthened hospital stays, and decreased ejection fraction rates.(16)

Correlation of Serum Sodium with CKMB: There was no significant correlation between serum sodium and CKMB levels from admission to 72 hours. **Konsam Biona Devi et al.** found that a considerably greater proportion of Killip III and IV, a higher level of CK-MB, and a higher degree of ST-elevation were all related to hyponatremia. Out of six hyponatremia patients, four passed away.(49)

Correlation of Serum Sodium with Ejection Fraction: Serum Sodium on admission, at 24 hours, at 48 hours and 72 hours has a significant positive correlation with Ejection Fraction (%). **Vikas et al.**, discovered that hyponatremia had a strong correlation with both long- and short-term mortality, a greater number of risk factors, a higher Killip class, and a lower ejection percentage.(52) **Sidhnath Singh et al.** found that long- and short-term mortality, as well as risk variables like smoking, diabetes, anterior infarction, higher Killip class, and reduced ejection fraction, were all strongly correlated with hyponatremia.(54)

ROC for predicting Death among STEMI using Serum Sodium: Serum Sodium on admission, at 24 hours, at 48 hours and 72 hours in predicting Death was significant, with the good diagnostic accuracy value.

Binomial Logistic Regression for predicting Death in STEMI: Hyponatremia on admission had an odds of 8.26 times of getting death in STEMI compared to normal sodium level on admission. For each increase in KILIP class, the odds of getting death among STEMI increases by 2.33 times. **Alexander Goldberg et al.** found that for every 1-mEq/L decrease in serum sodium, the adjusted Hazard Ratio for mortality or heart failure was 1.12.(47)

Chiara Lazzeri et al. noted that a considerable proportion of the aged population, diabetics, Killip III and IV, Triple Vein Disease, and increased rates of both long-term and short-term mortality were all associated with hyponatremia..(50) **Makrand B. Mane et al.** found that a considerably larger percentage of elderly people, tobacco use, diabetes, hypertension, ejection fraction, and both long-term and short-term mortality were all linked to hyponatremia.(51)

7 LIMITATIONS

Because of the cross-sectional nature of the study's design, it is unable to clearly determine the temporal link of the causal association between hyponatremia and outcomes.

It is impossible to discount the impact of confounding factors' bias on study results and outcomes.

Although the sample size was appropriate to evaluate the frequency of hyponatremia, it was insufficient to investigate the causes of severe hyponatremia.

Due to the fact that the study was carried out in a hospital and in a setting that provides tertiary care, there is a chance that Berksonian bias will result from the varying rates at which cases are admitted.

The study did not take into account a long-term follow-up of the complications and fatality rates due to limitations in the study period.

8 STRENGTHS

Age, gender, smoking, were not linked to the occurrence of hyponatremia; as a result, their potential to distort the results should be disregarded.

Numerous research have demonstrated the connection between hyponatremia and congestive heart failure; however, this study focuses on the connection between hyponatremia and non-stem MI.

The lead investigator, a general medicine postgraduate, collected the data, therefore there won't be any ascertainment bias or interobserver bias.

The desired sample size was reached despite the COVID-19's limitations and the difficulties in data gathering.

9 RECOMMENDATIONS

Serum sodium can be used as a prognostic parameter for predicting death among the acute STEMI patients.

The role of correction of the hyponatremia in prevention of the mortality rate among the acute STEMI patients' needs to be explored.

Hyponatremia upon admission and after 72 hours will serve as a reliable indicator of severity and prognosis. If hyponatremia is treated or avoided, these values may enable us to deliver a better result.

It is possible to conduct additional research with a follow-up that focuses on the impact of sodium supplementation and level correction on mortality.

A true picture will emerge from further research with slightly larger sample sizes, matched for known confounding variables, and all the different types of instances included.

10 SUMMARY OF RESULTS

- **Age (years):** The mean Age (years) among the subjects was 55.33 (\pm 12.62) ranging from 25 to 82 years. Among the subjects, 46 (57.5%) were in < 60 years and 34 (42.5%) were in > 60 years.
- **Gender:** Among the subjects, 62 (77.5%) were Males and 18 (22.5%) were Females
- **Smoker:** Among the subjects, 50 (62.5%) were Smoker
- **Alcoholic:** Among the subjects, 62 (77.5%) were Alcoholic
- **Diabetes Mellitus:** Among the subjects, 24 (30%) had Diabetes Mellitus.
- **Hypertension:** Among the subjects, 25 (31.25%) had Hypertension.
- **Prior CAD:** Among the subjects, none had Prior CAD.
- **Prior Diuretic Therapy:** Among the subjects, none had Prior Diuretic Therapy.
- **Serum CKMB (IU/L):** The mean Serum CKMB (IU/L) among the subjects was 87.36 (\pm 44.76) ranging from 31 to 221 IU/L.
- **KILIP Class:** Among the subjects, 42 (52.5%) were in Class I, 26 (32.5%) were in Class II, 8 (10%) were in Class III and 4 (5%) were in Class IV.
- **Ejection Fraction (%):** The mean Ejection Fraction (%) among the subjects was 45.56 (\pm 8.13) ranging from 25 to 65 %.
- **Myocardial Infarction Type:** Among the subjects, 38 (47.5%) had AWMI, 25 (31.25%) had IWMI and 5 (6.25%) had ASMI.
- **Outcome:** Among the subjects, 71 (88.75%) were Recovered and 9 (11.25%) had Death.
- **Serum Sodium on admission (mEq/L) with Outcome:** The mean Serum Sodium on admission (mEq/L) among Death was 132.78 (\pm 4.71) which is lower by 4.41 and statistically significant compared to 137.18 (\pm 2.49) in Recovered.

- **Serum Sodium at 24 hours (mEq/L) with Outcome:** The mean Serum Sodium at 24 hours (mEq/L) among Death was 132.56 (\pm 2.3) which is lower by 3.42 and statistically significant compared to 135.97 (\pm 2.31) in Recovered.
- **Serum Sodium at 48 hours (mEq/L) with Outcome:** The mean Serum Sodium at 48 hours (mEq/L) among Death was 133.33 (\pm 2.65) which is lower by 2.24 and statistically significant compared to 135.58 (\pm 2.25) in Recovered.
- **Serum Sodium at 72 hours (mEq/L) with Outcome:** The mean Serum Sodium at 72 hours (mEq/L) among Death was 134 (\pm 2.65) which is lower by 4.01 and statistically significant compared to 138.01 (\pm 3.3) in Recovered.
- **Serum Sodium with Myocardial Infarction Type:** The mean Serum Sodium on admission, at 24 hrs, 48 hrs and 72 hrs among the various type of MI, were not significantly different ($p > 0.05$).
- **Correlation of Serum Sodium with CKMB:** There was no significant correlation between serum sodium and CKMB levels from admission to 72 hours.
- **Correlation of Serum Sodium with Ejection Fraction:** Serum Sodium on admission, at 24 hours, at 48 hours and 72 hours has a significant positive correlation with Ejection Fraction (%).
- **ROC for predicting Death among STEMI using Serum Sodium:** Serum Sodium on admission, at 24 hours, at 48 hours and 72 hours in predicting Death was significant, with the good diagnostic accuracy value.
- **Binomial Logistic Regression for predicting Death in STEMI:** Hyponatremia on admission had an odds of 8.26 times of getting death in STEMI compared to normal sodium level on admission. For each increase in KILIP class, the odds of getting death among STEMI increases by 2.33 times.

11 CONCLUSION

Among the subjects, 71 (88.75%) were Recovered and 9 (11.25%) had Death. The mean Serum Sodium at admission, at 24 hours, at 48 hours and 72 hours (mEq/L) among Death was lower and statistically significant compared to the Recovered. Serum Sodium on admission, at 24 hours, at 48 hours and 72 hours in predicting Death was significant, with the good diagnostic accuracy value. Hence serum sodium can be used as a prognostic parameter for predicting death among the acute STEMI patients. The role of correction of the hyponatremia in prevention of the mortality rate among the acute STEMI patients' needs to be explored.

12 REFERENCES

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13 ANNEXURES

14.1 QUESTIONNAIR

NAME:

IP. NO:

AGE:

SEX:

DOA:

DOD:

OCCUPATION:

RELIGION:

MARITAL STATUS:

ADDRESS:

TELEPHONE NO:

STATUS AT DISCHARGE

PRESENTING COMPLAINTS:

I. HISTORY OF PRESENTING ILLNESS:

A. CHEST PAIN:

- Site: Precordial/ Restrosternal Epigastric/ Shoulder/ Neck
- Time of onset:
- Nature: Squeezing/ Crushing/ Compressive/ Tightness
- Radiation: Arm/ Back/ Epigastric/ Neck
- Frequency:

- Severity
- Aggravating Factor:
- Relieving Factor:
- Associated sweating:

B. BREATHLESSNESS:

- Onset: Sudden/ Gradual
- Grade: I/II/III/IV
- H/O Orthopnea: Yes/ No
- Wheeze: Present/ Absent
- H/O PND: Yes/No Associated symptoms

C. COUGH • Onset: acute insidious

- Productive/ Non Productive
- Sputum: Quantity
- Quality
- Colour
- Postural Variation
- Haemoptysis: Yes/ NO

D. PALPITATION

- Onset: Acute/ Insidious
- Duration
- Nature: intermittent/ continuous
- Aggravating Factors: Exertion/ Excitement
- Relieving Factors

E. PRESYNCOPE/ SYNCOPE

- Related to exertion : Yes
- Postural relation : Erect Supine
- Frequency : Isolated Frequent
- Loss of consciousness : Yes No
- Others :

F.SWELLING OF LEGS/ FACE

- Onset: Acute/ Insidious
- Duration:
- Associated with pain: yes/ No
- Diurnal Variation: Yes /No

G. NAUSEA /VOMITING

- Present Absent

H. MISCELLANEOUS

- General weakness/ Fatigue
 - Altered sensorium
 - Oliguria
- Convulsion
- Others

III. PAST HISTORY

- Past history : Present/Absent
- Duration
- Treatment
- IHD –Angina
- Infarction
- Hypertension
- Diabetes
- Rheumatic
- Syphilis
- Vascular heart disease
- TIA/ Stroke
- Any other

III. PERSONAL HISTORY

1. Diet Vegetarian Mixed
2. Sleep Sound Disturbed
3. Appetite Good Decreased
4. Bladder Normal Polyuria/ Anuria/Dysuria
5. Bowel Normal Constipated /Loose stools
6. Menstrual history Normal /Irregular Postmenopausal

7. Habits

- a) Smoking : Duration
- b) Alcohol : Duration
Type
Quantity
- c) Tobacco Chewing: Duration Quantity
- d) History of exposure to STD: Present/ Absen

IV. GENERAL PHYSICAL EXAMINATION

- 1) Built Well/Moderate Poor
- 2) Nourishment Obese/Average Poor
- 3) Emotional state Calm/Anxious Restless
- 4) Pallor Present/ Absent
- 5) Cyanosis Present/ Absent
- 6) Icterus Present/ Absent
- 7) Clubbing Present/ Absent
- 8) Pedal oedema Present/ Absent
- 9) Lymphadenopathy Present/ Absent
- 10) Extremities Warm/ Cold

V. VITAL SIGNS –

Pulse –

Blood pressure –

Respiratory rate –

Temperature

VI. SYSTEMIC EXAMINATION CVS EXAMINATION

1) Pulse

- Rate
- Rhythm
- Volume
- Character
- Condition of Vessel Wall
- Radio Femoral Delay

2) JVP –Normal /Raised

A. INSPECTION

- Precordium Normal/Bulged
- Apical impulse Visible / Non Visible
- Other pulsation

B. PALPATION

- Apical impulse -Location, Character
- Thrills -Apex Parasternal area
- Any other

C. PERCUSSION

- Cardiomegaly
- Pericardial effusion

D. AUSCULTATION

Heart sounds S3/S4 Present/ Absent

Murmur -Timing/Location/Character/Radiation/Grade

Pericardial rub

Basal crepitations

Others

KILLIP CLASS:

RESPIRATORY SYSTEM:

PER ABDOMEN:

CENTRAL NERVOUS SYSTEM

INVESTIGATIONS

I. BLOOD

HAEMOGLOBIN	gm/dl
TC DC	Cells/mm ³
NEUTROPHILS	%
LYMPHOCYTES	%
EOSINOPHILS	%
BASOPHILS	%
MONOCYTES	%
ESR	%
CARDIAC ENZYMES: CPK-MB	IU/L
Or TROPONIN T.	IU/L

II.URINE

ALBUMIN	
SUGAR	
MICROSCOPY	

III.BIOCHEMISTRY

RBS		mg/dl
SODIUM LEVELS	ON ADMISSION	mEq/L
-AFTER 24 HRS		mEq/L
-AFTER 48 HRS		mEq/L
-AFTER 72 HRS		mEq/L
TOTAL CHOLESTEROL		mg/dl
HDL CHOLESTROL		mg/dl
LDL CHOLESTROL		mg/dl
VLDL CHOLESTROL		mg/dl
TRIGLYCERIDE		mg/dl

IV.ELECTROCARDIOGRAPHY

V.ECHOCARDIOGRAPHY

EJECTION FRACTION	
CONCLUSIONS	

OTHER RELEVANT INVESTIGATIONS

DIAGNOSIS

IN HOSPITAL COMPLICATIONS

CCF/LVF

Cardiogenic shock

Arrhythmias

Thromboembolism

Pericarditis

Rupture of Interventricular septum

Rupture of papillary muscle

Aneurysm

Any other

FOLLOW UP UPTO 30 DAYS

13.1.1 CONSENT FORM

13.1.2 PATIENT INFORMATION SHEET

INFORMED CONSENT DEPARTMENT OF GENERAL MEDICINE

Thanjavur Medical College, thanjavur

Principal investigator : Dr. Cauvery N.

Research guide : Dr. S.Vetrivel, M.D.

Organisation : Department of General Medicine

Informed consent :

I have been invited to participate in research Project Titled

“A STUDY OF SERUM SODIUM LEVEL AND ITS PROGNOSTIC SIGNIFICANCE IN PATIENTS WITH ACUTE STEMI IN TERTIARY CARE CENTER”

I understand, it will be answering a set of questionnaire, undergo physical examination, investigations and appropriate treatment.

I also give consent to utilise my personal details for study purpose and can be contacted if necessary.

I am aware that I have the right to withdraw at any time which will not affect my medical care.

Signature of the participant :

Date :

Signature of the witness :

Date :

Signature of the investigator :

Date :

ஒப்புதல் படிவம்

பெயர் :

பாலினம் :

வயது:

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது மருத்துவத் துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவர் மேற்கொள்ளும் "கடுமையான மாரடைப்பும் இரத்தத்தின் சோடியம் அளவு குறைப்பாடினால் ஏற்படும் பின் விளைவுகள்" குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் :

கையொப்பம் / ரேகை

நாள் :

15.3 PATIENT INFORMATION SHEET

I, Dr. Cauvery N (Ph:7373488333) am conducting a “*A STUDY OF SERUM SODIUM LEVEL AND ITS PROGNOSTIC SIGNIFICANCE IN PATIENTS WITH ACUTE STEMI IN TERTIARY CARE CENTER*”

You are being invited to take part in a research study. Before you decide whether to take part, it is important for you to understand why the research is being done. Feel free to discuss the study with others if you wish. Please take time to decide whether or not you wish to take part. The study will follow patients with *ACUTE STEMI* admitted in ,THANJAVUR MEDICAL COLLEGE Hospital over a period of time. The type of treatment patients receive will not be altered by taking part in this study.

If you agree to take part you will be observed from the time you agree to take part until you leave the hospital. No additional tests will be undertaken as part of the study, but you will be asked to give permission for your medical records to be examined in detail, in order to collect information about your health status and any treatments that you have throughout your hospital stay. Your treatment options will not be altered in any way by taking part in this study. Your doctor will decide on the best treatment for you. Participation in the study does not restrict your ability to change from one treatment option to another. There will be no additional visits as part of the study. There are no anticipated disadvantages or risks involved in this study as it is an observational study. All information that is collected about you during the course of this study will be kept strictly confidential.

This study was reviewed and approved by the Institutional Review Board and Ethics’

Contact details:

Please contact **Dr.CAUVERY N** at Department of Medicine ,THANJAVUR MEDICAL COLLEGE. PHONE NO: 7373488333

13.1.3 DATA SHEET

A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S
NAME	FATHER NAME	AGE	SEX	SMOKING	ALCOHOL	DM	SHT	PRIOR CAD	PRIOR DIURETIC THERAPY	CKMB	KLIP	Na on Admission	Na at 24hrs	Na at 48hrs	Na at 72 hrs	EF	MI	OUTCOME
1	ANBALAGAN	65	MALE	YES	YES	NO	NO	NO	NO	170	1	136	138	135	138	48	ALMI	+
2	RADHA KRISHNAN	44	MALE	YES	YES	NO	NO	NO	NO	70	1	140	136	134	140	50	AWMI	+
3	MURUGESAN	37	MALE	NO	YES	NO	NO	NO	NO	80	1	136	134	130	132	30	AWMI	-
4	RAJENDRAN	60	MALE	YES	YES	NO	NO	NO	NO	160	2	138	138	136	140	50	AWMI	+
5	RATHNAVEL	60	MALE	YES	YES	YES	NO	NO	NO	140	1	136	140	140	142	48	AWMI	+
6	GOPI	30	MALE	NO	YES	NO	NO	NO	NO	140	2	136	138	135	136	45	IWMI	+
7	VJAYAKUMAR	43	MALE	NO	YES	NO	YES	NO	NO	210	2	138	136	136	134	42	IWMI	+
8	KRISHNAMOORTHY	50	MALE	YES	YES	NO	NO	NO	NO	220	2	135	136	134	138	46	ALMI	+
9	KRISHNAMOORTHY	40	MALE	YES	YES	NO	NO	NO	NO	116	1	135	136	138	136	40	IWMI-PWMI	-
10	RAJENDRAN	60	MALE	YES	YES	YES	NO	NO	NO	110	1	137	135	134	142	45	AWMI	+
11	VINOTH	38	MALE	NO	YES	NO	NO	NO	NO	108	2	132	132	132	138	30	AWMI	+
12	JAYAM	55	FEMALE	NO	NO	NO	NO	NO	NO	96	2	138	135	134	146	40	AWMI	+
13	MALLIKA	53	FEMALE	NO	NO	NO	NO	NO	NO	90	2	140	138	136	132	40	AWMI	+
14	SAMBASIVAMI	62	MALE	YES	YES	NO	YES	NO	NO	80	3	140	139	135	144	40	ASMI	+
15	KUMAR	36	MALE	YES	YES	NO	NO	NO	NO	221	1	142	140	136	142	45	ASMI	+
16	ILAYAPERUMAL	57	MALE	YES	YES	YES	YES	NO	NO	95	1	135	135	133	133	36	IWMI-PWMI	+
17	RAMKI	32	MALE	NO	NO	NO	NO	NO	NO	65	2	135	135	136	140	50	IWMI	+
18	GEETHA	70	FEMALE	NO	NO	YES	YES	NO	NO	48	2	137	138	137	138	60	AWMI	+
19	DHARMALINGAM	50	MALE	NO	YES	NO	YES	NO	NO	108	1	138	134	136	137	50	IWMI-PWMI	+
20	VEERAMANI	56	MALE	NO	YES	NO	NO	NO	NO	126	1	136	135	136	135	54	PWMI	+
21	SEKAR	48	MALE	YES	YES	NO	NO	NO	NO	110	1	138	139	140	140	50	AWMI	+
22	SADHAMHUSAN	25	MALE	YES	YES	NO	NO	NO	NO	96	1	140	137	137	138	50	AWMI	+
23	SIVA SARAVANAN	40	MALE	YES	YES	NO	YES	NO	NO	124	2	137	137	138	136	50	AWMI	+
24	MALLIKA	62	FEMALE	NO	NO	NO	NO	NO	NO	70	2	132	132	134	136	45	IWMI	+
25	KALIAMOORTHY	54	MALE	YES	YES	YES	NO	NO	NO	35	1	135	133	130	132	45	AWMI	+
26	YAGATHALI	70	MALE	YES	YES	NO	NO	NO	NO	80	1	136	134	137	140	48	AWMI	+
27	ANJAMMAL	60	FEMALE	NO	NO	NO	NO	NO	NO	90	2	138	135	135	142	50	IWMI	+
28	KUMAR	55	MALE	YES	YES	NO	NO	NO	NO	78	1	135	136	138	138	54	AWMI	+
29	JAYALAXMI	48	FEMALE	NO	NO	NO	NO	NO	NO	34	1	137	140	136	138	60	AWMI	+
30	KANDHAVEL	62	MALE	YES	YES	NO	NO	NO	NO	65	1	135	132	132	130	45	IWMI	-
31	SRINIVASAN	61	MALE	YES	YES	YES	YES	NO	NO	37	2	140	135	136	142	50	IWMI	+
32	ABDUL RAHIM	46	MALE	YES	YES	NO	NO	NO	NO	137	1	138	135	137	144	50	ASMI	+
33	MALATHI	52	FEMALE	NO	NO	NO	NO	NO	NO	144	2	135	138	136	136	54	AWMI	+
34	MOORTHY	60	MALE	YES	YES	NO	NO	NO	NO	138	4	136	130	130	138	50	IWMI--	+
35	JAYALAXMI	60	FEMALE	NO	NO	YES	YES	NO	NO	31	3	138	130	132	138	45	IWMI--	+
36	XAVIER	55	MALE	YES	YES	NO	NO	NO	NO	45	4	138	134	134	136	50	AWMI--	-
37	NARAYANASAMY	80	MALE	YES	YES	YES	YES	NO	NO	74	2	137	136	135	140	45	AWMI--	+
38	SAMINATHAN	68	MALE	YES	YES	YES	NO	NO	NO	80	1	138	132	132	133	25	AWMI	+
39	RAJAGOPAL	75	MALE	YES	YES	NO	NO	NO	NO	50	3	135	136	136	137	45	IWMI-PWMI	+

41	TAMIL SELVAN	JAYARAMAN	38	MALE	NO	NO	NO	NO	NO	NO	NO	127	3	126	130	130	130	135	45	AWMI--	-
42	SELVI	SANKAR	48	FEMALE	NO	NO	NO	NO	NO	NO	NO	152	2	136	137	135	137	135	48	IWMI--	+
43	MASILAMANI	KALIYAMOORTHY	52	FEMALE	NO	NO	NO	NO	NO	NO	NO	179	3	138	135	138	135	135	52	AWMI--	+
44	JAYAKUMAR	RAMASAMY PILLAI	72	MALE	YES	YES	NO	NO	NO	NO	NO	130	1	140	138	135	138	136	50	IWMI--	+
45	BABU	SAMBANTHAMOORHY	42	MALE	YES	NO	NO	NO	NO	NO	NO	48	1	139	136	137	137	137	52	AWMI	+
46	MURUGESAN	MARUTHAMUTHU	56	MALE	YES	NO	NO	NO	NO	NO	NO	80	2	137	138	140	140	140	50	AWMI--	+
47	PALANIVEL	POTHAN	82	MALE	YES	NO	NO	NO	NO	NO	NO	36	2	135	137	136	136	138	48	AWMI--	+
48	BALA KRISHAN	LAKSHMANAN	65	MALE	YES	NO	NO	NO	NO	NO	NO	71	1	136	135	136	142	142	50	AWMI--	+
49	GOVINDARAJ	VEERAVAN	61	MALE	YES	NO	NO	NO	NO	NO	NO	105	1	138	138	140	138	138	54	AWMI--	+
50	RAJENDRAN	MANIKAM	60	MALE	YES	NO	NO	NO	NO	NO	NO	45	4	136	132	134	130	130	38	ORBBMI	-
51	RANGARAJ	THYAGARAJAN	50	MALE	YES	NO	NO	NO	NO	NO	NO	74	1	140	136	137	142	142	50	ALMI	+
52	RAVICHANDARAN	KUMARASAN	54	MALE	YES	YES	NO	NO	NO	NO	NO	50	1	139	136	1385	140	140	60	IWMI	+
53	PALANISAMY	RAMASAMY	81	MALE	YES	YES	NO	NO	NO	NO	NO	36	1	137	138	138	143	143	50	AWMI	+
54	VEERASEKAR	MURUGAN	60	MALE	YES	NO	NO	NO	NO	NO	NO	71	1	128	130	132	135	135	45	AWMI	-
55	PRAKARAN	PARANDHAMAN	40	MALE	YES	NO	NO	NO	NO	NO	NO	44	1	141	137	136	140	140	50	IWMI	+
56	GUNASEKARAN	VENKATACHALAM	51	MALE	YES	YES	NO	NO	NO	NO	NO	85	2	138	136	135	133	133	30	IWMI	+
57	MOHAMAD HUSSAIN	ABDULLAH	62	MALE	YES	NO	NO	NO	NO	NO	NO	77	2	143	140	136	138	138	50	IWMI	+
58	RAJENDARAN	RATNASAMY	58	MALE	YES	YES	NO	NO	NO	NO	NO	68	2	140	134	133	131	131	40	AWMI	+
59	SUDHA	KANDHASAMY	65	FEMALE	NO	NO	NO	NO	NO	NO	NO	55	3	142	136	136	136	136	52	AWMI	+
60	AMULRAJ	KAALI	53	MALE	NO	YES	NO	NO	NO	NO	NO	115	1	140	136	136	135	135	65	IWMI	+
61	SAVITHRI	MARIMUTHU	50	FEMALE	NO	NO	NO	NO	NO	NO	NO	39	1	138	137	139	137	137	50	IWMI	+
62	THYAGARAJAN	RAJARAM	53	MALE	YES	NO	NO	NO	NO	NO	NO	40	3	137	138	136	138	138	40	IWMI-PWMI----	+
63	KAMLA	VAYAPURI	55	FEMALE	NO	NO	NO	NO	NO	NO	NO	67	3	136	138	136	139	139	25	AWMI----	+
64	VETRI SELVI	RAJARATHINAM	47	FEMALE	NO	NO	NO	NO	NO	NO	NO	54	4	126	130	134	135	135	25	IWMI-LWMI--	-
65	RAJENDARAN	NADESAN	69	MALE	YES	YES	NO	NO	NO	NO	NO	88	2	136	136	136	142	142	32	IWMI	+
66	MUTHUKUMAR	THAMARAI SELVAN	35	MALE	NO	YES	NO	NO	NO	NO	NO	36	1	135	135	136	140	140	40	AWMI--	+
67	KUMARAN	KARUPAIYAA	75	MALE	YES	NO	NO	NO	NO	NO	NO	45	1	132	132	130	134	134	30	IWMI--	+
68	CHANDRA SEKAR	NADESAN	75	MALE	YES	YES	NO	NO	NO	NO	NO	39	2	136	136	132	132	132	35	AWMI--	+
69	JAYAKODI	KALIYAN	55	FEMALE	NO	NO	NO	NO	NO	NO	NO	67	2	136	136	135	138	138	50	IWMI--	+
70	DHATCHINAMOORTHY	ARUMUGAM	60	MALE	YES	NO	NO	NO	NO	NO	NO	72	1	138	135	135	136	136	48	IWMI	+
71	THYAGARAJAN	NALLUSAMY	44	MALE	NO	YES	NO	NO	NO	NO	NO	74	1	137	140	137	138	138	50	IWMI	+
72	RAJAKUMAR	NATARAJAN	44	MALE	YES	YES	NO	NO	NO	NO	NO	36	1	139	139	138	137	137	54	IWMI	+
73	MATHI	SELVARJ	50	MALE	YES	NO	NO	NO	NO	NO	NO	54	1	131	132	132	135	135	40	LWMI	+
74	MANONMANI	SAMBANTHI	80	FEMALE	NO	NO	NO	NO	NO	NO	NO	68	1	140	135	134	140	140	48	AWMI	+
75	SEETHARAMAN	RADHAKRISHANAN	73	MALE	YES	YES	NO	NO	NO	NO	NO	77	1	142	135	135	142	142	40	AWMI	+
76	ILAYARAJA	NARAYANASAMY	35	MALE	NO	YES	NO	NO	NO	NO	NO	65	2	132	132	135	135	135	36	IWMI	+
77	TAMILARASU	SOMASUNDARAM	71	MALE	YES	YES	NO	NO	NO	NO	NO	131	2	135	135	132	132	132	40	AWMI	+
78	GOVINDARAJ	KESAVA MOORTHY	59	MALE	YES	YES	NO	NO	NO	NO	NO	145	1	135	135	135	136	137	35	IWMI	-
79	NAGARATHINAM	KALAYAPERUMAL	65	FEMALE	NO	YES	YES	NO	NO	NO	NO	40	1	136	138	136	140	140	48	ASMI	+
80	LAKSHMI	KASINATHAN	60	FEMALE	NO	YES	NO	NO	NO	NO	NO	70	1	136	137	136	140	140	48	ASMI	+
81	DHAVALAN	KAMARAJ	42	MALE	YES	YES	NO	NO	NO	NO	NO	63	1	135	134	138	142	142	52	AWMI	+