"A STUDY ON INCIDENCE OF HYPONATREMIA IN PATIENTS WITH ACUTE ST ELEVATION MYOCARDIAL INFARCTION ADMITTED IN INTENSIVE CARDIAC CARE UNIT OF DR. PRABHAKAR KORE HOSPITAL; KLE UNIVERSITY BELAGAVI"

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With reference to the above, we wish to inform you that your proposed research project titled **"A STUDY ON INCIDENCE OF HYPONATREMIA IN PATIENTS WITH ACUTE ST ELEVATION MYOCARDIAL INFARCTION ADMITTED IN INTENSIVE CARDIAC CARE UNIT OF DR. PRABHAKAR KORE HOSPITAL;KLE UNIVERSITY BELAGAVI ",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

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ABBREVIATIONS

AF	ATRIAL FIBRILLATION
AMI	ACUTE MYOCARDIAL INFARCTION
AWMI	ANTERIOR WALL MYOCARDIAL INFARCTION
ASMI	ANTERO SEPTAL MYOCARDIAL INFARCTIO
AVP	ARGININE VASOPRESSIN
BPM	BEATS PER MINUTE
CAD	CORONARY ARTERY DISEASE
CCF	CONGESTIVE CARDIAC FAILURE
CHB	COMPLETE HEART BLOCK
CK-MB	CREATININE KINASE-MB
CVD	CARDIOVASCULAR DISEASES
DBP	DIASTOLIC BLOOD PRESSURE
DM	DIABETES MELLITUS
ECG	ELECTROCARDIOGRAM
EF	EJECTION FRACTION
FVR	FAST VENTRICULAR RHYTHM
IHD	ISCHEMIC HEART DISEASE
IWMI	INFERIOR WALL MYOCARDIAL INFARCTION
LWMI	LATERAL WALL MYOCARDIAL INFARCTION
LVF	LEFT VENTRICULAR FAILURE
MI	MYOCARDIAL INFARCTION
MR	MITRAL REGURGITATION
PWMI	POSTERIOR WALL MYOCARDIAL INFARCTION
QRBBB	RIGHT BUNDLE BRANCH BLOCK
RVMI	RIGHT VENTRICULAR MYOCARDIAL INFARCTION
SBP	SYSTOLIC BLOOD PRESSURE
TR	TRICUSPID REGURGITATION

ABSTRACT

Background: Acute myocardial infarctions are one of the leading causes of death in the developed world, with prevalence approaching three million people worldwide. Occurrence of hyponatremia in these patients may influence the morbidity and mortality. Present study aimed to assess the Incidence of hyponatremia in patients with acute ST elevation myocardial infarction admitted in intensive cardiac care unit of Dr. Prabhakar Kore hospital.

Material and Method: This cross sectional type of study was conducted among the patients admitted in Intensive Cardiac Care Unit of KLE's Dr. Prabhakar Kore Hospital and MRC, Belagavi fulfilling inclusion criteria. Patients with acute myocardial infarction presenting to ICCU, having chest pain lasting more than 20 minutes with diagnostic ECG changes with characteristic ECG alterations consisting of; 1) ST elevation ≥ 1 mm in ≥ two contiguous limb leads. 2) ST elevation ≥ 2mm in ≥ two contiguous precordial leads of both genders aged more than 18yrs were included. Patients lower than 18yrs, case of Non-ST elevation MI and not willing to be part of study were excluded. Venous blood was drawn and sent for basic laboratory tests; complete blood counts including Serum Sodium at admission, 72 hours post admission and at the time of discharge. Data were collected and stored in Microsoft Excel and analysed using SPSS v21.

Results: In present study total of 80 patients fulfilling inclusion criteria are included, with mean age of 59.17±12.09yrs of age. Among the included patients 28.8% were female and 71.3% were male patients, with male preponderance. Prevalence of hyponatremia at the admission was seen in 38.8% of the patients. Ejection fraction

among the patients with hyponatremia at admission had the lower mean levels compared to the patients with normal sodium levels.

Conclusion: The present study reports Prevalence of hyponatremia at the admission was seen in 38.8% of the patients, at 72hr it was 57.5% with hyponatremia and at discharge it was 57.5% of the patients. Also hyponatremia was associated with the presence of lower ejection fraction documented on 2D-ECHO.

Keyword: Myocardial infarction, Prevalence, Hyponatremia, Sodium, ECHO.

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INTRODUCTION

Acute Coronary Syndrome consists of Myocardial Ischemia or Infarction and is further categorised into ST Segment Elevation Myocardial Infarction (STEMI) and Non ST Segment Elevation Myocardial Infarction (NSTEMI) & Unstable Angina. STEMI is one of the leading causes of mortality and morbidity worldwide. Identification of patients with severity of the disease and prognosis associated with it makes an important part of plan of care to be provided to the patients.[1]

Hyponatremia is the most prevalent dyselectrolytemia among hospitalized infirms, in particular with the immediate aftermath of surgery, individuals suffering from congestive cardiac failure (CCF), liver cirrhosis, and nephrotic syndrome. [2–4] It has been exhibited to be an indicator of cardiovascular (CVS) mortality in people suffering from cardiac failure. Actually, acute myocardial infarction and heart failure both have neurohormonal activity associated with them. Hyponatremia well known [9–11] is typical following a myocardial infarction, and clinical recovery is accompanied by an increase in plasma sodium content.[5–8] Clinical relevance of hyponatremia in chronic cardiac failure is well established, but its prognostic relevance in the circumstances of acute myocardial infarction is not clearly appreciated.[9–11]

The most prevalent electrolyte condition in inpatients across a variety of medical settings is hyponatremia ,substantial hyponatremia.

The emergence of hyponatremia in acute MI is a signifier that likely integrates different prognostic value, among which are the degree of dysfunction noted in the Left ventricle, shifts in the hemodynamical equilibrium, and extensiveness of the neurohormonal axis excitation affecting Left ventricular remodelling, pinning patients

at a greater risk for Heart Failure and mortality. Severe hyponatremia is foreseeably significant and life-threatening and can potentially cause neurological complications.[12,13 14,15]

The objective of this study is to ascertain the incidence of hyponatremia in the context of acute ST elevation MI and to assess its applicability in evaluating the prognosis of patients with acute ST elevation MI. Therefore, the objective of this study has been to assess the prevalence of hyponatremia in patients with ST elevation MI and to establish how it influenced the patients' outcome.

AIMS & OBJECTIVES

AIM:

The current study sought to determine the prevalence of hyponatremia among patients with acute ST-elevation myocardial infarction admitted to the Dr. Prabhakar Kore hospital's Intensive cardiac care unit.

OBJECTIVE:

- Primary objective is to study the incidence of hyponatremia in the patients with acute ST elevation myocardial infarction (STEMI).
- Secondary objective is to study the effect of hyponatremia on outcome of patients with acute ST elevation myocardial infarction.

REVIEW OF LITERATURE

History

The Ebers papyrus from 2600 BC has the first recorded account of a patient with acute coronary syndrome, which reads, "If you discover a guy with heart trouble, with agony in his arms, in the side of his heart, death is nigh." Although the description is still accurate, the prognosis has evolved throughout time.

The 1700s saw the beginning of industrialization, which is when the present epidemic of cardiovascular illness began. Rise in tobacco use, a decline in physical activity, and the adoption of a diet heavy in fat, calories, and cholesterol were the three main factors that contributed to this. Although the clinical syndrome of angina was first described in the 1770s, James B. Herrick did not describe acute myocardial infarction until 1912.

Burden of disease:[16] CVDs, such as ischemic heart disease and stroke, are the major causes of mortality, accounting for 17.7 million fatalities.[17] According to the World Health Organization, India accounts for one-fifth of these fatalities globally, particularly among the young. According to the findings of the Global Burden of Disease Research, India has an age-specific Cardiovascular Diseases mortality rates of 272 per 1 lakh people, which amounts to higher than the global average of 235. CVDs strike Indians a decade before the rest of the world.[18] India in 2016, CVDs contributed to 28·1% of total deaths and 14·1% of total disability-adjusted life years (DALYs) compared with 15·2% and 6·9%, respectively in 1990.[19]

Acute myocardial infarction

Nearly three million people worldwide experience acute myocardial infarctions (MI),one of the components of Acute coronary syndrome (ACS), to be ranked as one of the foremost causes of mortality in the industrialised countries, resulting in more than a million deaths in the US each year. The two kinds of acute myocardial infarction are ST-segment elevation myocardial infarction (STEMI) and Non-ST segment elevation myocardial infarction (NSTEMI) . Unstable angina and NSTEMI are identical. Nonetheless, cardiac indications do not increase. [20–22]

Because of a scarcity of oxygen during a MI, the heart muscle is irreversibly damaged. A MI would compromise diastolic and systolic function, thereby increasing the likelihood of arrhythmias in the patient. A MI may also lead to a number of grave consequences. The restoration of blood flow and reperfusion of the heart are essential. The earlier the therapy is initiated (within six hours), better the outcome.

When two of the subsequent conditions are satisfied, a MI is diagnosed.

- **New onset ST-segment alterations or block in the left bundle branch (LBBB)**
- **The symptoms of ischemia**
- **Evidence of intracoronary thrombus during an angiography or necropsy**
- **The appearance of a pathological Q wave on the strip of ECG**
- **Evidence of New regional wall motion (RWM) abnormalities on imaging**

Etiology[23]

Acute myocardial infarction is primarily caused by a reduction in coronary blood flow. When the body's requirements for oxygen are not fulfilled, heart ischemia ensues. There are a number of causes for decreased coronary blood flow. An atherosclerotic plaque rupture and thrombosis typically results in a significant reduction in coronary artery blood flow. Additional causes of reduced oxygenation and myocardial ischemia comprises coronary artery embolism (2.9%), ischemia induced secondary to cocaine consumption, coronary artery dissection and vasospasm of the coronary arteries. [24,25]

Non-modifiable risk factors

- Gender
- Age
- Pattern of male baldness
- Family history

Risk factors that are Modifiable

- Cigarette smoking
- Diabetes mellitus
- Dyslipidemia
- Obesity
- Hypertension
- Sedentary lifestyle
- Elevated homocysteine levels
- Poor oral hygiene
- Peripheral vascular disease

Other causes of MI

- Vasculitis
- Trauma
- Dissection of Aorta
- Coronary artery anomalies
- Hyperthyroidism
- Anaemia

Epidemiology

An acute myocardial infarction is brought on by a reduction in coronary blood flow. Cardiac ischemia happens when there is not enough oxygen available to meet the demand. Reduced coronary blood flow can be triggered by a variety of factors. Thrombosis is frequently caused by the rupture of atherosclerotic plaques, which drastically reduces blood supply to the coronary arteries. Variable causes of reduced oxygenation/myocardial ischemia comprise coronary artery embolism (2.9 percent), ischemia induced secondary to cocaine consumption, coronary artery dissection and vasospasm of the coronary arteries.[26,27]

Pathophysiology[23]

Atherosclerotic vessel rupture initiates a cascade of inflammatory events that comprises of macrophages and monocytes induced cellular epithelial disruption with eventual platelet aggregation and plaque formation resulting in a thrombus development. The myocardial tissue receives less oxygen as a consequence of the coronary artery's decreased oxygen delivery. The inability of the mitochondria to

generate ATP initiates the ischemic cascade and, as a result, endocardial apoptosis (cell lysis) or myocardial infarction ensues.

Geographical distributions of coronary arteries are distinct and diagnostic, with significant differences caused by genetic diversity. For instance, the ventricular apex, anterior wall, and left anterior descending coronary artery all receive oxygenated blood supply from this artery, and interventricular septum, the inferolateral wall receives blood from the left circumflex artery. Right coronary artery (RCA) is concerned with supply of oxygenated blood to right ventricle. Inferior wall vascularisation is taken care of by Right coronary artery (RCA) or the (LCx) Left Circumflex artery.[28]

Histopathology.

Myocardial infarction's histology evolves as the situation deteriorates. There are no microscopic histological developments at the beginning of time. Between 0.5 and 4 hours, fibre waviness at the tissue's edge can be seen under light microscopy. Glycogen levels are low. Before the cardiac myocardial tissue experiences oedema and coagulative necrosis, four to twelve hours would have passed. The histological specimen darkens and mottles after 12 to 24 hours. Histopathology exhibits neutrophil predominance and contraction band necrosis. Nuclei are destroyed after 1 to 3 days, but it takes 3 to 7 days for macrophages to begin obliterating apoptotic cells. Granulation tissue develops after 7 to 10 days. Ten days later, collagen one starts to build up. There has been myocardial damage after two months.

Cardiac biomarkers

Particularly for Non-ST-elevation MI, cardiac biomarkers can aid in identification of acute myocardial infarction. The most precise laboratory test is a thrombin assay, which has two isoforms, I and T Troponins, which peaks after a duration of 12 hours and continue up to 7 days. Additionally unique to the myocardium is creatinine kinase MB. Within two to three days, it returns to normal after reaching a high at ten hours. LDH increases after 72 hours and returns to normal after 10 to 14 hours. LDH is not the preferred cardiac biomarker for use in clinical practise to identify acute Myocardial Infarction. Finally, MB is not utilised therapeutically and has a very poor myocardium selectivity; it increases fast and recovers to normal too soon. Recently, high-sensitivity troponin received a licence for use in the United States following considerable study and use in Europe. Compared to conventional troponin, it is more sensitive but less focused. As a result, a variety of false-positive interpretations are one of the potential problems.

Assessment

All patients who present with chest pain should have rapid and early ECG testing. Women may report strange symptoms like stomach ache or vertigo and may not even experience any chest pain. Breathlessness is a typical early symptom of myocardial infarction in older people. Each of these symptoms should prompt an ECG test.[29–31]

ECG sensitivity is low, although it is fairly sensitive for MI (95 to 97%) (approximately 30 percent). ECG sensitivity can be increased with additional ECG testing and right-sided, posterior lead placement. For instance, in an ECG, "hyperacute T waves," or peaked T-waves, quite often signifies the early onset of ischemia and its progression to an ST elevation. An ST-elevation myocardial

infarction is noticed when there is an ST-elevation of more than 2 mm in two consecutive ECG leads (lateral: I, aVL, V5, V6; septal equal V1, V2; the inferior limb leads II, III, aVF ; anterior chest leads: V3, V4). ST depressions are generally seen in the opposite myoca rdium anatomical regions (reciprocal changes).

It can be difficult to identify STEMI with an ECG, especially in patients who also have pacemakers and a block in the left bundle branch. Application of Sgarbosa criteria offers measure that helps a medical expert identify a STEMI in these patients. In the proper clinical scenario, a blockage in the left main coronary artery results in isolated ST-elevations in lead aVR . According to Wellens, strongly biphasic T waves in the chest leadsV2, V3 are often an indication suggesting an impending occlusion of the proximal left anterior descending artery. These T waves could result in a significant infarction of the anterior wall of the myocardium. Individuals suffering with myocardial infarction may or may not show ECG abnormalities diagnostic or characteristic of ST-Elevation. Patients presenting with typical chest discomfort must be evaluated for NSTEMI if an ECG shows modest abnormalities inclusive of ST-depressions and alterations in the T wave .

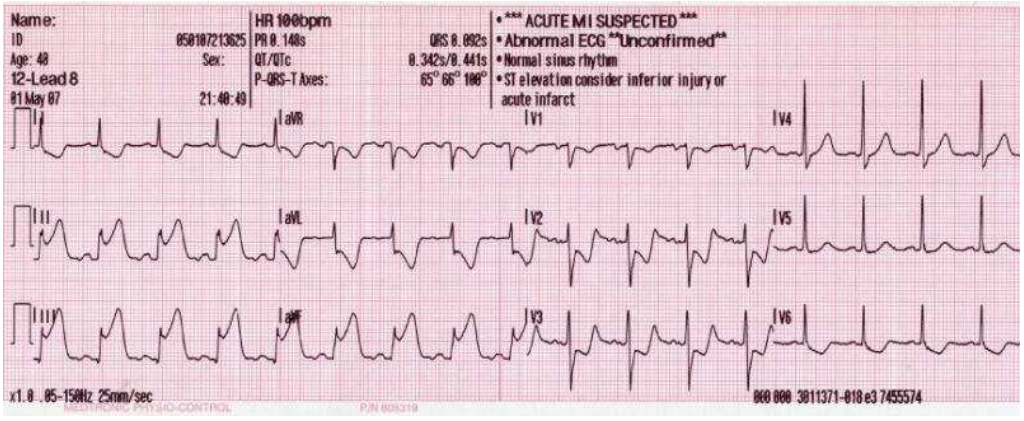

Figure 1: ECG indicating acute myocardial infarction[23]

An ECG with pardee waves in inferior limb leads II, III, and aVF denoting acute myocardial infarction and reciprocal changes in anterolateral chest leads.

Practitioners can use diagnostic criteria to evaluate whether further testing is required to identify people with NSTEMI. In patients with a dubious clinical history, troponins are almost always used since ECG has a limited sensitivity for STEMI. Hence, the most extensively used and validated HEART score for the same. Based on the care giver's suspicion, the individual's clinical profile and risk factors, the diagnostics of ECG, and the serum troponin level, it establishes the "risk level".

Laboratory features

- Cardiac troponins I or T
- CBC
- Renal profile
- Lipid profile
- The best way to stratify risk is to use the metabolic parameters
- Brain-type natriuretic peptide (BNP), particularly in MI patients who go on to develop heart failure. BNP shouldn't be recommended as a MI marker.
- Cardiovascular imaging tests: Cardiac coronary angiography is utilized for both percutaneous coronary intervention (PCI) and the diagnosis of coronary channel blockages. An transthoracic 2D echocardiography is recommended to ascertain the wall motion(RWM) abnormalities, ischemic mitral regurgitation (MR), extent of abnormalities in the valve, existence of pericardiac tamponade.

Management and Treatment

Individuals experiencing STEMI and NSTEMI chest pain must take aspirin that must be immediately chewed and ranges from 160 milligrams to 325 milligrams. Additionally, if their oxygen saturation is lower than 91 percent, intravenous access to be secured and oxygen supplementation should be provided. In addition to sublingual nitroglycerin for pain treatment, opioids may be provided if blood pressure is adequate.

Rapid reperfusion is a component of treatment for STEMI. An immediate percutaneous coronary intervention is preferred (PCI). Patients should get two antiplatelet medications—intravenous heparin and a P2Y12 RECEPTOR INHIBITOR,i.e., adenosine diphosphate receptor antagonist , preferably Ticagrelor—as a combination before having PCI. Direct thrombin inhibitors or glycoprotein IIb/IIIa inhibitors may also be used during the procedure of percutaneous intervention. If the coronary percutaneous intervention is necessary, reperfusion with an intravenous thrombolytic drug should be administered.

A stable asymptotic patient with NSTEMI might not benefit from the procedure of urgent percutaneous coronary intervention; instead, they must be given antiplatelet (Aspirin 325mg) medication. A Percutaneous coronary intervention to be performed less than 48 hours of admission, potentially lowering the in-hospital cardiovascular mortality and prolonged in-hospital stay. PCI should be performed right away on NSTEMI patients who have refractory ischemia or ischemia with hemodynamic or electrical instability.

Before being released from the hospital after an acute MI, patients commonly receive aspirin, a statin therapy of high-dose, a beta-blocker, and an angiotensin converting enzyme (ACE)inhibitor.

Percutaneous coronary intervention (PCI) must be finished within 12 hours if it is being taken into consideration. If at all possible, fibrinolytic therapy must be started less than 120 minutes. In addition to the antiplatelet therapy, all patients in hospital should receive parenteral anticoagulation.

Differential diagnosis of chest pain

- Pericarditis
- Aortic dissection
- Acute gastritis (APD)
- Esophagitis
- Pneumothorax
- Acute cholecystitis (Calculous / Acalculous)
- Pulmonary embolism

Prognosis[23]

The factors which will negatively influence the prognosis;

- DM
- Elderly individuals
- Delayed reperfusion
- Peripheral vascular disease or previous MI
- Stroke (CVA)

- Reduced cardiac ejection fraction
- Congestive cardiac failure (CCF)
- Depression
- Elevated C-reactive protein (CRP)and Brain natriuretic peptide (BNP) levels.

Noted Complications (possible adverse outcomes)

- Mitral regurgitations (MR) of new onset
- Aneurysm of the Left ventricular
- Emboli
- Rupture of the Ventricular septum
- Malignant Arrhythmias

Outcome

Outside of hospitals, acute myocardial infarction has a significant fatality rate. According to data, 40% to 50% of patients die once they arrive at the hospital, while about 1/3 rd of the mortality is in-hospital. Another 5% to 10% of people will pass away in the first 12 months after a myocardial infarction. Approximately 50% of individuals experience a readmission during the initial one year following their primary event of MI. The ejection fraction, age, and other relevant comorbidities affect the prognosis. The outcome for patients who do not have any revascularization will be worse than it is for those who do. The greatest outcome is seen in patients with intact left ventricular function and early, successful reperfusion..[32–34]

Body fluid balance

Humans are primarily an aquatic species. It is the basic building block of existence of life and aqueous base solution where the vital metabolic activities that give origin to life take place. Humans roughly comprises of 75% of water by weight as new-borns, and as adults comprises of 50% to 60% water by mass. Several processes which are regulatory in keeping the optimal concentrations in the various compartments of the body also cause fluid to change continuously. Although the flow of fluid between areas is routinely monitored by passive diffusion of osmotically active solutes along the concentration gradient, hydrostatic pressures do have a governing impact.[35]

Cellular distribution

Distribution of fluids in the body encompasses two different types of fluid: Extracellular fluid and Intracellular fluid. The Intracellular fluid (ICF) that makes up the body makes up about 40% of its overall weight. It encompasses the entirety of what is commonly referred to as the cytoplasm of a cell. Cellular fluids normally are stable and doesn't respond quickly to variations. Numerous chemical reactions take place in this region, making it essential in maintaining an optimal serum osmolality. Extracellular fluid (ECF), which sums up to about 20% of total body weight (TBW), further is broken down into plasma, which makes up about 5% of the total weight of the body, and the interstitial space, makes up about twelve percent (12%) of the weight. Furthermore, Additional compartments of fluid is conceivable in pathological conditions and is categorised into transudate and exudate.

Body fluids' precise chemical composition varies widely. Which organ and which body component the fluid is housed in will determine this. Extracellular fluid (ECF) and interstitial fluid share a identical chemical make-up. Proteins, sodium, chloride, and bicarbonate are abundant in extracellular spaces, whereas potassium, magnesium, and phosphate are few. Interstitial fluids contain little protein physiologically. Proteins, phosphate, magnesium, and potassium are abundant in intracellular fluids, but sodium, chloride, and bicarbonate are insignificantly present.[36–38]

Mechanism[35]

Fluid travels across the body's cellular surroundings by passively crossing membranes which are semipermeable. The number of particles of solute per litre of solution characterises Serum Osmolarity. The Serum Osmolarity in the physiological range is around 285-295 mOsmol/L. Hypoosmotic or hypotonic solution is considered when the serum osmolarity is lesser than the normal physiological range, in comparison Hyperosmotic or hypertonic solution is considered when the serum osmolarity is greater than that of the physiological range. Osmotic concentration gradients at the cellular level is conserved by constant monitoring of the Active pumping of ionic proteins of the transmembrane. On the other hand, those components dilate or concentrate when fluid volume changes rapidly. By Secretion of the solutes into the gastrointestinal system (GIT) or by the excretion as the ultrafiltrate of the plasma (urine) or solute absorption by the gastrointestinal system (GIT), blood plasma osmotic gradients are kept constant.

Osmolarity is influenced by proteins such as albumin and as well as ionic components, found in the human serum. Glucose is another chief substance which is osmotically active to be taken into account. There will be a shift of fluid away from hypoosmotic compartments and toward hyperosmotic ones. To demonstrate that cations and anions are in balance, all the bodily fluids must possess an ionic net electrical charge that is closest to zero(0). Preferentially the diffusion of ionic components occurs through fluids based on the presence of permeability of the membranes. A gradient with a somewhat higher concentration osmolarity occurs from a membrane that is impermeable to an ion.

Energy expansion in the form of generation of ATP is brought about by Membrane pumping proteins to move constituents from hypo osmolar concentration places to areas of higher concentration (hyperosmolar) areas against the diffusion gradient, generates solute gradients physiologically.

As a result of these processes, water is osmotically "drawn" into fluid compartments within the cell. Additionally to osmotic pull of the fluids, produced and maintained hydrostatic pressures are necessary for fluid mobility throughout the body. The capillary membrane is particularly useful for transferring the fluids in the extracellular space from plasma into the tissue interstitium. Hydrostatic pressure, which happens when rising pressure forces the fluid out of a spot, has a "push" effect on fluid movement.

The net flow of fluid is caused by the "push" of the hydrostatic pressures mixed with "pull" in the osmotic pressure gradient. The Starling equation is used to describe this mathematically:

$$Jv = Kfc ([Pc - Pi] - n [Op-Oi])$$

Kfc represents filtration fluid coefficient in the capillaries, while Jv stands for net rate of the capillary fluid flow. The hydrostatic pressure of the Capillaries (Pc), hydrostatic pressure of the interstitium (Pi), osmotic reflection coefficient (n), oncotic pressure of plasma (Op), and the oncotic pressure of interstitium (Oi) are all abbreviations for pressure.[36]

Clinical relevance of fluid balance[36]

Disorders of fluid balance can either have an excess of fluids or decrease in the amount of the fluid that is actually in circulation. The medical term for fluid overflow and overload is oedema. Although it can happen in any tissue, oedema is commonly found in the soft tissues of the extremities. A reduction in the fluid load is termed as dehydration.

The first manifestation of oedema presents as swollen face and soft tissues of the limbs, then as an increase in skin size and stiffness. By exerting a certain force of pressure using a finger to compress for about 30 seconds into the tissue and quickly creates a dimple (depression) in the oedematous skin, one can relieve peripheral oedema by increasing the hydrostatic pressure in interstitial space. On the other hand, by the rise of the interstitial hydrostatic pressure, which drives to seep back into the capillaries, usage of compression stockings can lessen the symptoms of peripheral oedema.

Pulmonary oedema is brought on by extra fluid expanding into the interstitial tissues present in the lung. Chest pain and shortness of the breath are typical signs. Orthopnoea, i.e., breathing difficulty on lying flat on a surface, may happen as extra

fluid spreads throughout the entire lung. Due to the impairment of gas exchange in the lungs caused by pulmonary oedema, the situation might quickly worsen. Cardiac and renal dysfunction are both associated with pulmonary oedema. According to conventional wisdom, heart failure causes pulmonary oedema by lessening the capacity and efficacy of the left atrial and left ventricular pumps. The pulmonary veins experience back pressure as a result, increasing vessel pressure. Then, in accordance with the Starling equation, the pulmonary capillaries hydrostatic pressures increase, "pushing" fluids into the interstitial lung space. Renal failure prevents body from properly removing fluids and osmotic components, which results in oedema. As a result, there is an increase in the hydrostatic push out of the capillaries and increased osmotic draw into tissues.[39]

The malfunction of the liver can also result in oedema. Lack of osmotically active proteins is the cause of this. specifically, the inability to produce albumin. In extracellular blood, albumin is primarily found in the plasma. In the interstitial space, it is unusual to notice it. The "pull" of the osmotic pressure into capillaries is consequently quickly reduced when body albumin is decreased. According to the Starling forces, causing the fluid to migrate into interstitial gaps.[40]

SODIUM

Electrolytes are necessary for maintaining the electrical neutrality of cells as well as the generation and transmission of action potentials in neurons and muscles. Along with magnesium, calcium, phosphate, and bicarbonates, sodium, potassium, and chloride are the primary electrolytes. Electrolytes are obtained from diet and bodily fluids.

These electrolytes may not be in equilibrium, which could result in either high or low concentrations. By interfering with normal biological processes, high or low electrolyte levels can have potentially disastrous repercussions.[41]

Sodium is one of the most crucial electrolytes in extracellular fluid because it is an osmotically active cation. It is in charge of controlling both the volume of extracellular fluid and the potential of cell membranes. Active transport describes the movement of sodium and potassium through cell membranes.

The kidneys are in charge of regulating salt levels. The majority of sodium reabsorption occurs in the proximal tubule. Where sodium reabsorption takes place is in the distal convoluted tubule. A hormone called aldosterone causes sodium-chloride symporters, which move salt, to become active.[41]

The most prevalent dyselectrolytemia is hyponatremia. A diagnosis is made when the serum sodium level is less than 135 mmol/L. There are neurological effects of hyponatremia. Headaches, disorientation, nausea, and delirium are possible side effects for patients. When serum sodium levels surpass 145 mmol/L, hypernatremia develops. Hypernatremia symptoms include tachypnoea, insomnia, and irritability. Significant problems including cerebral oedema and osmotic demyelination syndrome might arise with rapid sodium changes.

The sodium level in the body is closely related to the water balance. Sodium controls the volume of extracellular fluid. The body has about 4000 mEq of sodium in total. Extracellular fluid contains 40% of it, bones contain 50%, and soft tissues include 10%. The most prevalent cation in extracellular fluid is sodium. All cells have sodium pumps that are working to keep the sodium extracellular. This process

depends on ATP. Sodium is essential for maintaining the acid-base balance as sodium bicarbonate.[42]

Normal Na+ levels in plasma are 136–145 mEq/L and 12 mEq/L in cells.

The average diet comprises 5–10 g of sodium, mostly in the form of sodium chloride. Every day, the same quantity of salt is expelled in the urine. However, the body may preserve salt to the point that urine does not contain sodium on a sodium-free diet. Sodium consumption should ideally be lower than potassium intake, yet processed meals increase sodium intake.

The kidneys are designed to excrete potassium while maintaining salt balance. When urine is produced, the first 175 litres of glomerular filtrate each day contain 800 g of sodium, of which 99 percent is reabsorbed. The proximal convoluted tubules reabsorb the majority of this (80%). This endeavour is ongoing. Like salt, water is capable of being reabsorbed. The sodium excretion is regulated by the distal tubules. Aldosterone improves sodium reabsorption in the distal tubules. Water reabsorption from tubules is stimulated by antidiuretic hormone (ADH).

The numerous mechanisms include sodium channels in the collecting duct, sodium hydrogen exchangers in the proximal convoluted tubules and ascending limb, sodium chloride co-transporters in the distal tubules (ascending limb), and sodium potassium exchanger in the distal tubule. The rate of sodium filtration, which is affected by renal plasma flow and blood pressure, directly affects the rate of sodium excretion (acting through atrial natriuretic peptide). Aldosterone controls how much is reabsorbed.

Hyponatremia

Low sodium levels in the blood are referred to medically as hyponatremia. Dehydration, a reduction in blood pressure, sleepiness, lethargy, confusion, stomach aches, oliguria, tremors, and coma are some of the clinical signs and symptoms of hyponatremia. On the other hand, hyponatremia is frequently asymptotic.

Causes of hyponatremia

Vomiting

Diarrhoea

Burns

Renal tubular acidosis

Addison's disease

Congestive cardiac failure

Excess non-electrolyte IV infusion

SIADH and defective ADH secretion

Drugs (ACE inhibitors, Lithium, NSAIDS, Chlorpropamide, Vasopressin and Oxytocin)

Causes of Hypernatremia

Hypernatremia is defined as an increase in blood sodium levels. Hypernatremia symptoms include dry mucous membranes, fever, thirst, and agitation.

Cushing's illness, extended cortisol treatment, and pregnancy, when steroid hormones increase salt accumulation in the body, are all causes of hypernatremia.

Prolonged cortisone therapy

Cushing’s disease

Pregnancy

Sodium retention due to steroid hormones

Dehydration

Exchange transfusion with stored blood

Primary hyperaldosteronism

Excessive intake of salt

Elderly patients with poor water intake

Drugs (Ampicillin, tetracycline, oral contraceptives, anabolic steroids, osmotic diuretics)

Various article discussing the hyponatremia in ST elevated MI.

Klopotowski et al. found that hyponatremia had an independent connection with in-hospital mortality after studying 1858 ST-elevation MI patients. Hyponatremics had higher overall death rates from cardiac failure represented (27.8% vs. 18.4%, p=0.022) and in-hospital death rate (13.5% vs. 3.8%, p0.001) as well. Hyponatremia was discovered to be independently linked with in-hospital mortality and the composite endpoint after adjusting for variables. Patients representing the

lowest and highest quintiles of sodium had 3.27 times and 2.65 times the likelihood of dying in the hospital, respectively, compared to those in the second quartile (best survival). Patients representing the lowest quintile demonstrated a significantly elevated risks of in-hospital mortality in the adjusted model. Thus concluding that Hyponatremia is a simple laboratory parameter independently linked to a higher mortality risk in STEMI infirms receiving primary coronary arteries angioplasty.[43]

Presence of untimely hyponatremia is straightforward a measure of the neurohormonal axis activation during initial stages of myocardial infarction (MI), and it foresees the continuing progression into cardiac morbidity and mortalities, according to Goldberg A et al's research of 978 patients. 36.2 percent of study participants with the least serum sodium decile (135mEq/L) and 9.7% of study participants with the maximum serum sodium decile (>=143mEq/L) had death or heart failure. An independent predictor of 30-day mortality in patients with acute ST-elevation myocardial infarction is the presence of hyponatremia in the first presentation to the hospital or untimely development of hyponatremia, and the degree of hyponatremia worsens the prognosis. If serum sodium levels be utilised as a straightforward laboratory tool to recognise people at greater risk, more investigation is required.[44]

In a study by Singla I et al., to assess the effect of hyponatremia on outcome in patients with non-ST elevation acute coronary syndrome. On presentation, 341 (23.1%) of the 1,478 patients were hyponatremic (sodium 135 mEq/L). In the next 30 days, patients who had hyponatremia at admission had a substantially higher risk of dying or experiencing a recurrent myocardial infarction (odds ratio (OR) 1.98, 95% confidence range (CI) 1.35 to 2.89, p0.001). Left ventricular ejection fraction

(EF),Age, diuretic usage prior to in-hospital admission, hypotension(low blood pressure) at initial presentation, anaemia, chronic renal failure (CKD), pulmonary emphysema, and high Cardiac troponin levels were all taken into account, but this relationship still existed (95% confidence interval (CI) 1.1 to 2.5, odds ratio(OR) 1.7, p = 0.01). Finally, incidence of hyponatremia on admission is linked to a 30-day worse consequence in individuals who present with suspected acute coronary syndrome (ACS) or non-STEMI.[45]

Ahmad Sajadieh et al. found that adverse outcomes defining it as mortality i.e., myocardial infarction (MI) a component of acute coronary syndrome (ACS) occurred in 43% of individuals with blood levels of sodium lesser than 134mEq/L in the trial comprising of six hundred and seventy one women and men aged between 55-75 years who had nil prior records of cancer, cardiac co-morbidities (CVD, MI), stroke (CVA). A total of 62 participants (9.2%, group B) and 14 subjects (2.1%, group A) exhibited s-Na levels of 137 mEq/L and 134 mEq/L, respectively. No individual exhibited sodium concentrations lower than 129 mEq/L. 43% of group A participants, 27% of group B subjects, and 14% of subjects with serum sodium > 137 mEq/L (controls) experienced an unfavourable result (P .002) They came to the conclusion that in middle-aged and elderly individuals, as an independent predictor of mortality and myocardial infarction (MI) is attributable to hyponatremia.[46]

In the analysis of two hundred and thirty five (235) consecutive infirms registered to the Intensive coronary care unit (ICCU), Fleat CT and Hilton P found that hypochloraemia, hyponatremia, uraemia were frequent in patients with confirmed myocardial infarctions (MI), and that severity of the infarctions (MI) was closely correlated with all of aforementioned indices. Additionally, they discovered greater

rates of in-hospital death in individuals with plasma sodium levels below 130mmol/L. Plasma salt, chloride, and potassium values varied significantly from day to day, frequently over the normal range. Patients who were given diuretics experienced more disruptions. It is concluded that after an infraction, plasma sodium concentrations fall, and the extent and duration of the fall are indices of the severity of the infarction.[47]

In a study conducted by Goldberg A et al., to assess the serum hyponatremia and long term mortality benefits in survivors of acute STEMI. A lower than 136 mEq/L mean serum sodium level, defined as hyponatremia, documented in hundred and eight (108)study participants comprising of about 11.0% of the study population during admission. An independent predictor of post discharge death in multivariable Cox proportional hazards model, attributable to presence of the hyponatremia at the time of presentation to the hospital, correcting for left ventricular ejection fraction (LVEF) and other possible clinical predictors of death (hazard ratio(HR) 2.0; 95% confidence interval(CI) 1.3-3.2; P =.002). Incidence of Hyponatremia during admission was also associated with post-discharge heart failure readmission (HR, 1.6; 95% CI, 1.1-2.6; P =.04). For each 1-mEq/L fall in serum sodium level, adjusted hazard ratio for cardiac mortality or heart failure represented 1.12 (95%) confidence interval (CI)1.07-1.18; P.001). Presence of Hyponatremia in initial stages of STEMI is a predicts of continued mortality and re-admission into the hospital for heart failure after being discharged from the hospital, independent of the other clinical parameters predicting of unfavourable outcomes includes the reduced left ventricular ejection(LVEF) percent.[14]

In a study by Tada Y et al., to assess the untimely development of hyponatremia in the implications of short lived outcome in STEMI. Hyponatremia

defined as serum sodium concentration of 136mmol/L 72 hours post admission. Initially, researchers looked at the brief (in-hospital mortalities or the development of cardiac failure) & prolonged (delayed) effects (cardiac death, re-admission for congestive heart failure) prognoses in individuals having suffered from STEMI. Then Secondly the study looked into establishing the causal relationship between the plasma arginine vasopressin and serum sodium levels. The incidence of heart failure during the in-hospital admission was significantly higher in hyponatremic patients (P=0.0018), long-term cardiovascular mortality was higher (17.2% vs. 6.3%, P=0.19), with re-hospitalisation secondary to Cardiovascular system failure being notably higher (20.7% vs. 4.5%, P=0.0024). The hyponatremia group had higher plasma arginine vasopressin (AVP) levels (4.5 vs. 2.7pg/ml, P=0.003), and it represented an unfavourable association with serum sodium level (r=-0.28, P=0.02). Hyponatremia (low serum sodium) being usual in initial stages of STEMI, was linked to cardiac failure in both short-lived and prolonged deleterious outcomes. Hyponatremia in STEMI patients may be caused by non-osmotic AVP secretion. [48]

In a study by Tang Q et al., to assess the relation betwixt hyponatremia and in-hospital infirms with STEMI. On presentation, 212 (13.1%) of the 1,620 patients had hyponatremia (sodium 135 mmol/L). In-hospital mortality was higher in hyponatremia patients (13.7% vs. 7.3%, p=0.002), as was cardiac failure (30.2% vs. 18%, p<0.001). Infirms having serum sodium levels of 130 mmol/L experienced a 22.9% rate of adverse events compared to 11.0% in in-hospital admitted infirms with serum sodium level of 130 to 135 mmol/L (p=0.034).Hence, Hyponatremia was solitarily associated with in-hospital cardiovascular morbidities and mortalities (OR: 1.77, 95% CI: 1.02-3.06, p=0.042) and heart failure (OR: 1.61, 95% CI: 1.06-2.43, p=0.025) in multivariate logistic regression. In Chinese patients with acute ST-

elevation myocardial infarction, hyponatremia is independently associated with adverse in-hospital outcomes, and risk of in-hospital death rate increases with increasing hyponatremia severity.[49]

In a study by Lazzeri C et al., to assess the usefulness of hyponatremia in acute phase of ST elevation myocardial infarction as a marker of severity. The following are the investigation's principal conclusions: (1) A common finding is hyponatremia, which is predominantly linked to advanced Killip classes, older age groups, and diabetes mellitus; (2) hyponatremia was associated with increased rates of in-hospital and continued death rates; and (3) hyponatremia was not solitarily associated with greater risk of death in the short and long-term using the propensity score model for evaluation. As a result, the presence of hyponatremia in the acute phase of STEMI should be considered a marker of more sick patients, according to these findings. [50]

In a study by Shah V et al. to evaluate the impact of hyponatremia on patients with ST-elevation myocardial infarction or heart failure's prognosis. Hyponatremia is the most existent dyselectrolytemia encountered in the routine clinical practise, and its suggestive of a poor prognostication in both cardiac failure and STEMI infirms. It significantly increases the risk of both brief and prolonged morbidity and mortalities, readmission rates to the hospital, and prolongation of the average duration of hospital admissions. Though it is not obvious as to hyponatremia is the primary reason for the guarded outcomes in study participants with STEMI and HF (heart failure), it can be ascertained that prompt identification of infirms at greater risk of hyponatremia development, aids in the initiation of early treatment. [51]

In a cross-sectional study by Sanchez AC et al., to evaluate the relationship between hyponatremia and clinical outcome in people who have recently suffered a myocardial infarction. Hyponatremia was present in 50.2% of patients up to seven days after in hospital registration. Patients with hyponatremia lasting up to 7 days had a higher rate of intensive care admission (69.7% vs. 54.3%, P=0.019, OR=1.9), unfavourable 30-day mortalities (12.7% vs. 2.2%, P=0.004, OR=6.5), hospital mortality (9.9% vs. 1.1%, P=0.006, OR=9.9), a higher rate of newly diagnosed heart failure (31.5% vs. 17.9%, P=0.043, OR= upon in-hospital registration), hyponatremia was linked to higher rates of mortality in the hospital (16.3% vs. 3.8%, P 0.004, OR 4.9) and 30-day mortality (18.4% vs. 5.9%, P 0.017, OR 3.5). In-hospital and 30-day mortality are linked to hyponatremia upon admission and at any time during the first seven days of hospitalisation. [52]

MATERIAL & METHOD

Source of data: Patients that meet the inclusion requirements and are admitted to KLE's Dr. Prabhakar Kore Hospital's ICCU and MRC, Belagavi.

Study design: a cross sectional type of study design.

Sample size:

The formula for calculating sample size is

$n = p (100- p) Z^{2} / \mathbf{E^{2}}$

The required sample size is n, the percentage occurrence of a state or condition is p, the percentage maximum error is E, and the value corresponding to the requisite level of confidence is Z.

Prevalence of CHF in patients with Hyponatremia is 27.5%.[53]

1. With percentage of maximum error as 10% at 95% confidence level sample size is given by,

$$= 27.5 \times (100- 27.5) \times (1.96)^{2} / {}^{10}2$$

$$n = 76.59 = 77$$

Early onset of hyponatremia suggests Yuko Tada et al. [48] looked into the results of ST-Elevation Acute Myocardial Infarction in the short- and long-term.

Inclusion Criteria for present study:

Individuals suffering from acute myocardial infarction presenting to ICCU, with

- 18 years or more age group of both sexes.
- 20 minutes or more of chest pain.

  Diagnostic ECG variations include recognizable ECG alterations including; 1) ST elevation ≥ 1 mm in ≥ two contiguous limb leads. 2) ST elevation ≥ 2mm in ≥ two contiguous precordial leads.

  Hyponatremia: when serum sodium levels < 136mmol/ litre.

Exclusion Criteria:

- Age less than 18 years of both sexes.
- All patients diagnosed as a case of Non ST Elevation MI.

Methodology

Patients admitted at KLES Dr Prabhakar Kore hospital & MRC with Acute ST ELEVATION MI Consenting for the study were enrolled and Informed consent was taken. A detailed history was documented and clinical features assessed. 15cc of venous blood was drawn and sent for basic laboratory tests, complete blood counts including Serum Sodium at admission, 72 hours post admission and at the time of discharge. Based on the clinical picture and test results patients was diagnosed to have hyponatremia at specified intervals.

The correlation between the time of detection and presentation of hyponatremia and the outcome due to the same was extrapolated and tabulated.

STATISTICAL ANALYSIS

Data were collected and stored in Microsoft Excel. Data was analysed using statistical software R and Microsoft Excel. Continuous variables were given in mean ± sd/median (range). Categorical variables were represented by frequency. To check the dependency between categorical variables Chi-square test was used. To compare mean/distribution over groups t-test/ANOVA/Mann-Whitney test/Kruskal-Wallis test was used. To compare the survival curves between hyponatremia group and another group Log-rank test was used. Cox proportional hazard model is used to find the risk factors of mortality. Kaplan-Meier Curve used to visualize the survival probabilities. To check the normality of variables Quantile-Quantile (QQ) plot/Shapiro-Wilk's test was used. P-value less than or equal to 0.05 considered statistically significant.

RESULTS

80 patients in total who met the inclusion criteria for this study were included, and their average age was 59.17±12.09yrs. Among the included patients 28.8% were female and 71.3% were male patients, with male preponderance.

Table 1: Average age of the patients in the study

	N	Minimum	Maximum	Mean	SD
Age	80	24.0	86.0	59.175	12.09

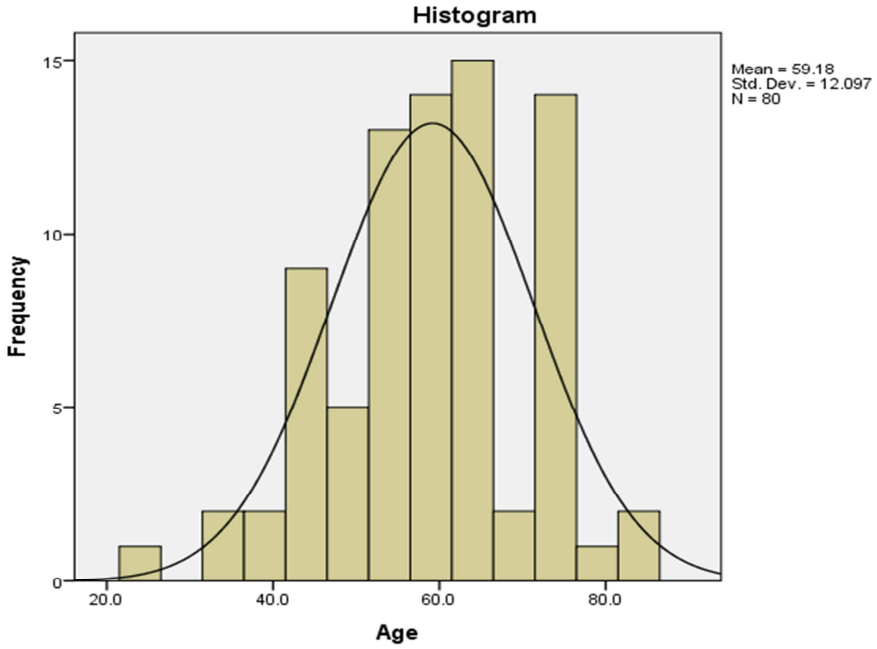

Figure 1: Histogram showing the mean distribution of the patients

Table 2: Gender distribution of the patients

		Frequency	Percent
Gender	Female	23	28.8
	Male	57	71.3
	Total	80	100.0

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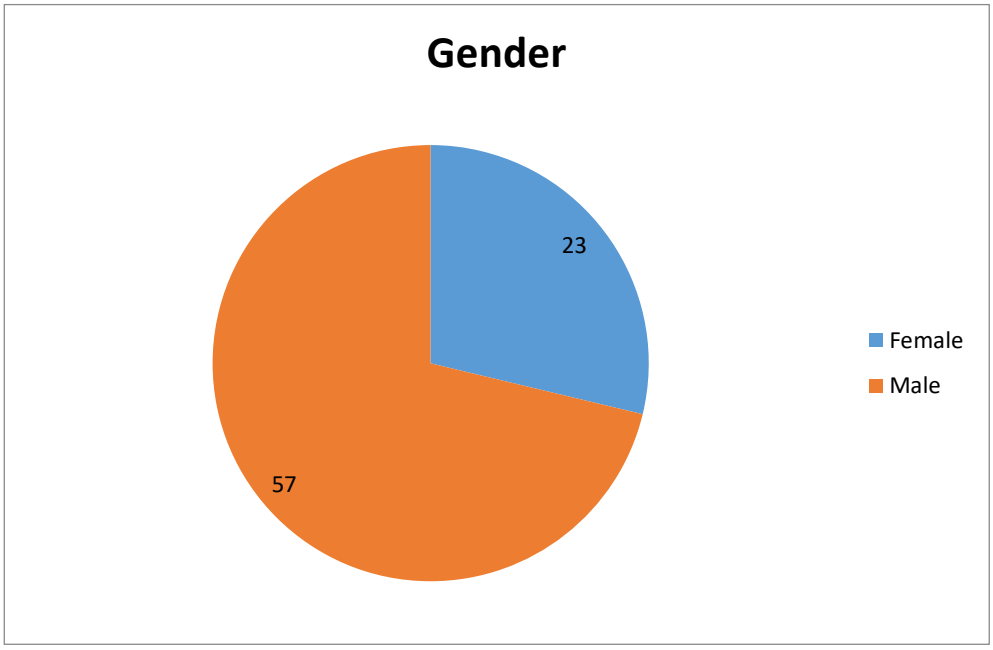

Figure 2: Gender distribution of the patients

Table 3: Distribution of patients based on occupation

		Frequency	Percent
Occupation	Business	10	12.5
	Carpenter	1	1.3
	Cook	3	3.8
	Coolie	5	6.3
	Driver	3	3.8
	Factory worker	1	1.3
	Farmer	29	36.3
	Housewife	21	26.3
	Retired Teacher	1	1.3
	Shopkeeper	2	2.5
	Software Engineer	2	2.5
	Teacher	2	2.5
	Total	80	100.0

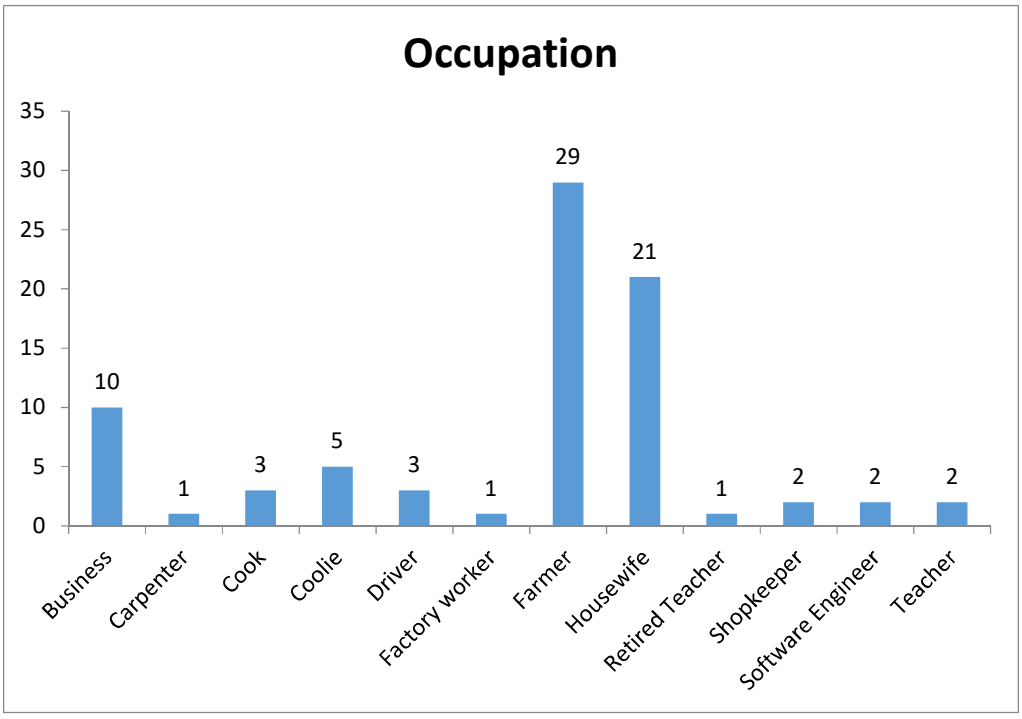

Figure 3: Distribution of patients based on occupation

Table 4: Showing the frequency of patients with comorbidities

		Frequency	Percent
Comorbidities	NIL	39	48.8
	Present	41	51.3
	Total	80	100.0

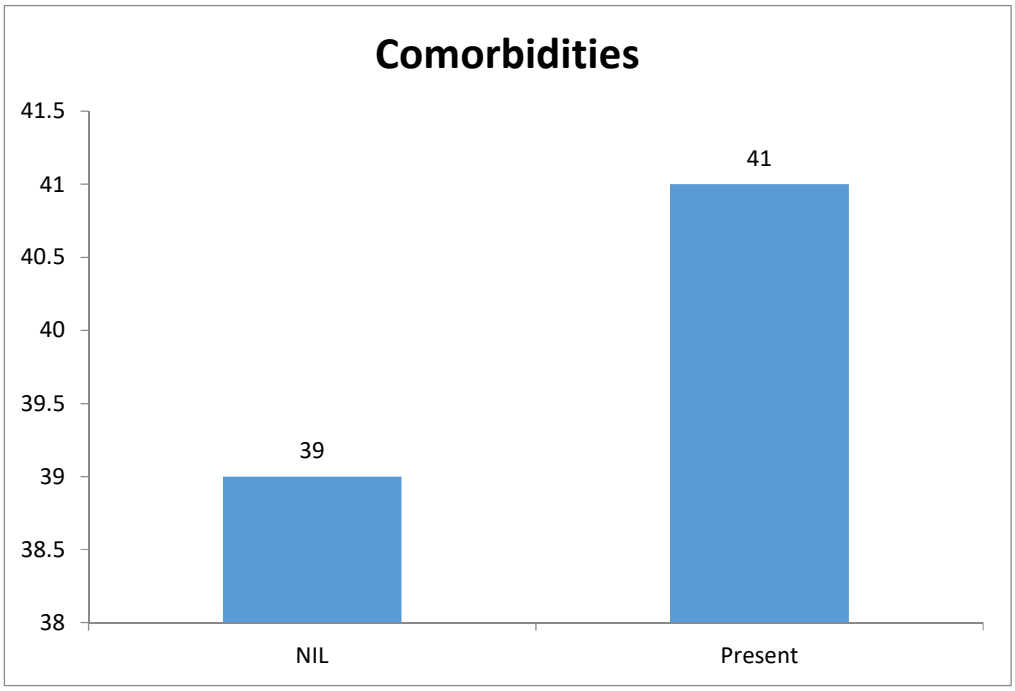

Figure 4: Showing the frequency of patients with comorbidities

Table 5: Showing the frequency of patients with hyponatremia at admission

		Frequency	Percent
Hyponatremia admission	Absent	49	61.3
	Present	31	38.8
	Total	80	100.0

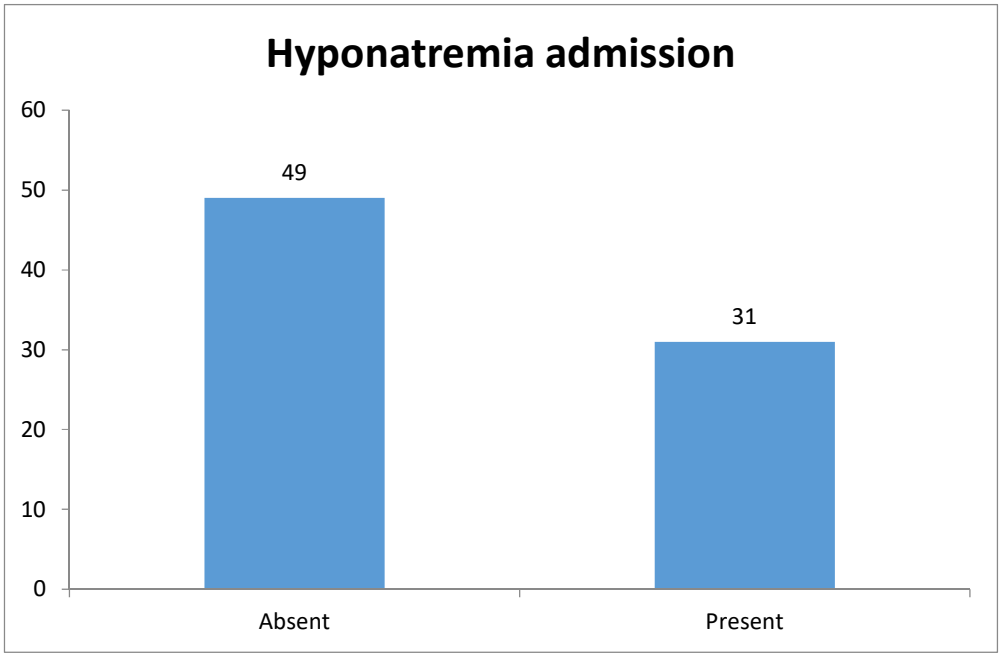

Figure 5: Showing the frequency of patients with hyponatremia at admission

Table 6: Showing the frequency of patients with hyponatremia at 72hr of admission

		Frequency	Percent
Hyponatremia at 72hr of admission	Absent	34	42.5
	Present	46	57.5
	Total	80	100.0

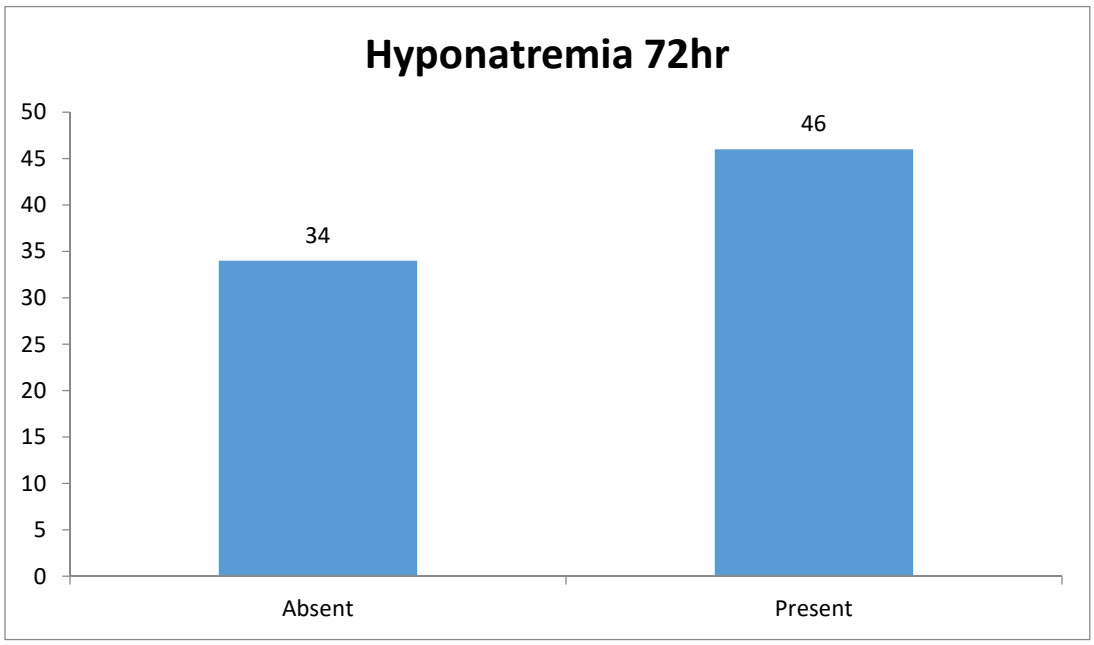

Figure 6: Showing the frequency of patients with hyponatremia at 72hr of admission

Table 7: Showing the frequency of patients with hyponatremia at the time of discharge

		Frequency	Percent
Hyponatremia at discharge	Absent	34	42.5
	Present	46	57.5
	Total	80	100.0

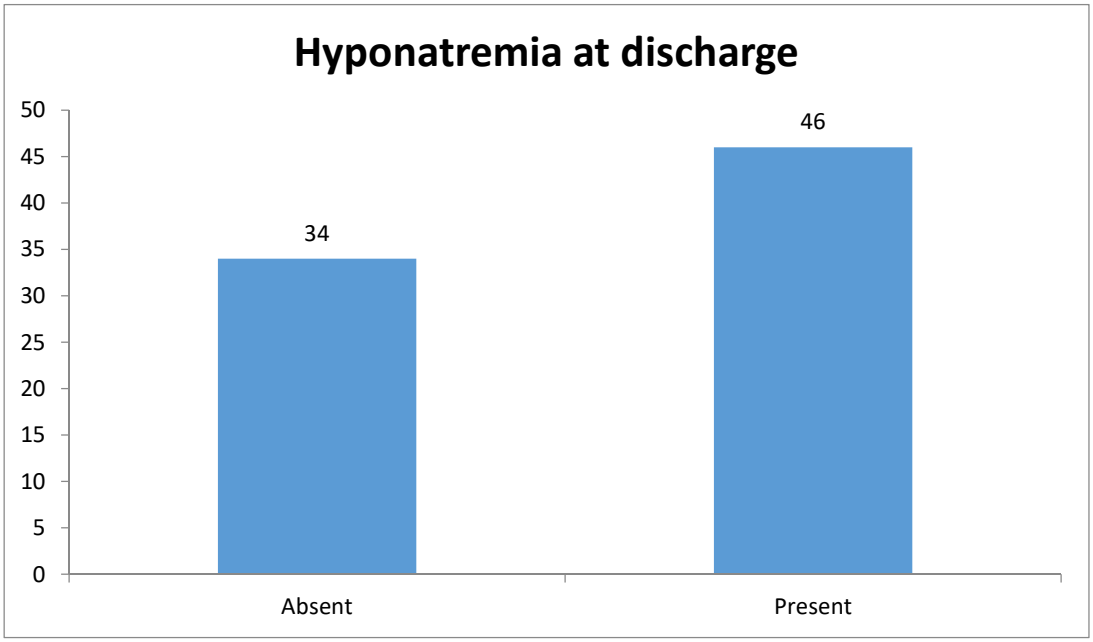

Figure 7: Showing the frequency of patients with hyponatremia at discharge

Table 8: Showing the frequency of ECG changes seen in study participants

		Frequency	Percent
ECG	AWMI	25	31.3
	AWMI, FVR	1	1.3
	AWMI, IWMI	2	2.5
	AWMI, IWMI, QRBBB	1	1.3
	AWMI, LWMI	08	10.1
	AWMI, PWMI	2	2.5
	AWMI, QRBBB	12	15.0
	AWMI, IWMI, AF with FVR(QRBBB)	1	1.3
	IWMI	16	20.0
	IWMI, RVWMI, PWMI	1	1.3
	IWMI, AF	1	1.3
	IWMI, CHB	1	1.3
	IWMI, LWMI	4	5.0
	IWMI, PWMI	1	1.3
	PWMI	4	5.0
	Total	80	100.0

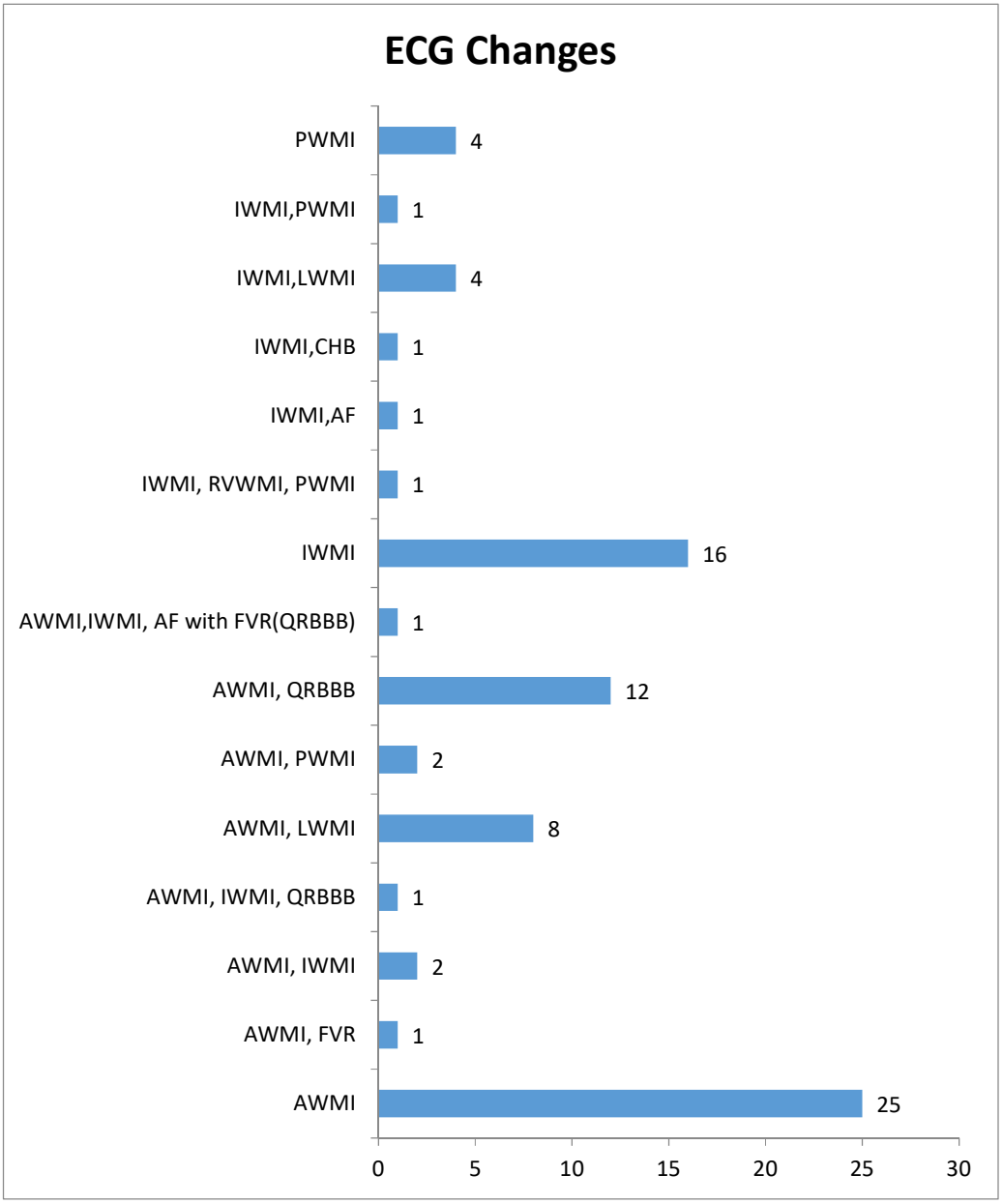

Figure 8: Showing the frequency of ECG changes seen in study participants

Table 9: Showing the mean levels of baseline parameters

	Minimum	Maximum	Mean	SD
Pulse Rate bpm	36.0	150.0	91.363	18.04
Respiratory Rate cpm	14.0	36.0	23.100	5.45
SBP	60.0	200.0	125.475	23.40
DBP	5.0	110.0	78.988	14.82
HB	8.4	19.7	13.223	2.31
Creatinine	.32	5.43	1.3803	.936
Urea	12.0	131.0	39.000	26.07

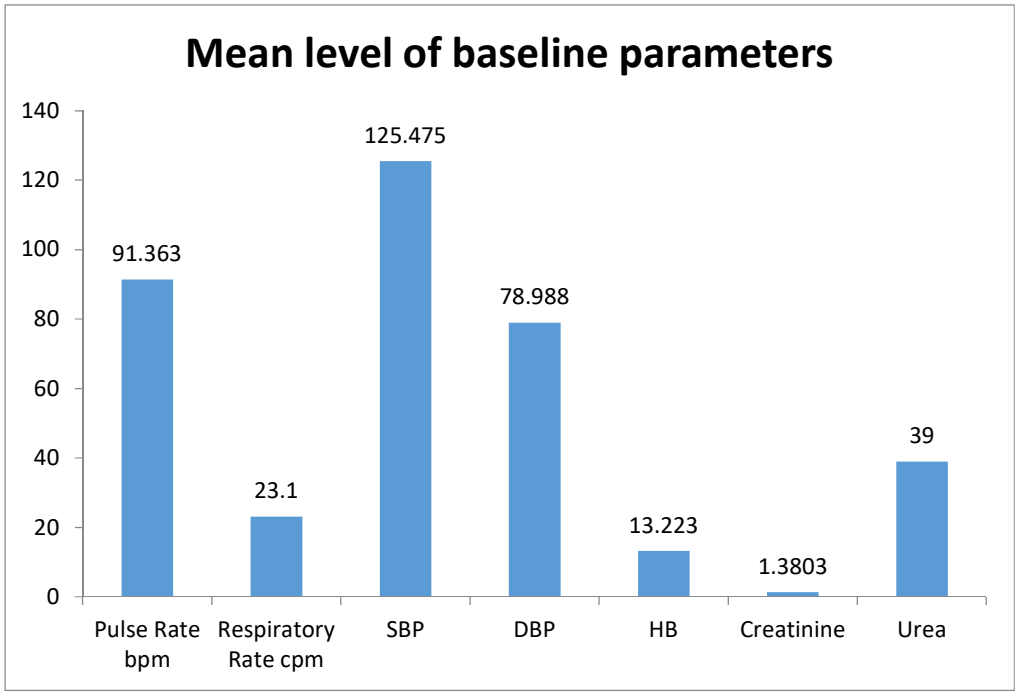

Figure 9: Showing the mean levels of baseline parameters

Table 10: Showing the mean level of sodium, potassium and chloride among study participants

	Minimum	Maximum	Mean	SD
Sodium at admission	118.0	144.0	136.225	4.31
Sodium at 72hrs	124.0	154.0	135.088	5.57
Sodium at discharge	122	142	134.50	4.05
Potassium	2.77	7.46	4.3034	.792
Chlorides	86.0	113.0	100.0	4.50

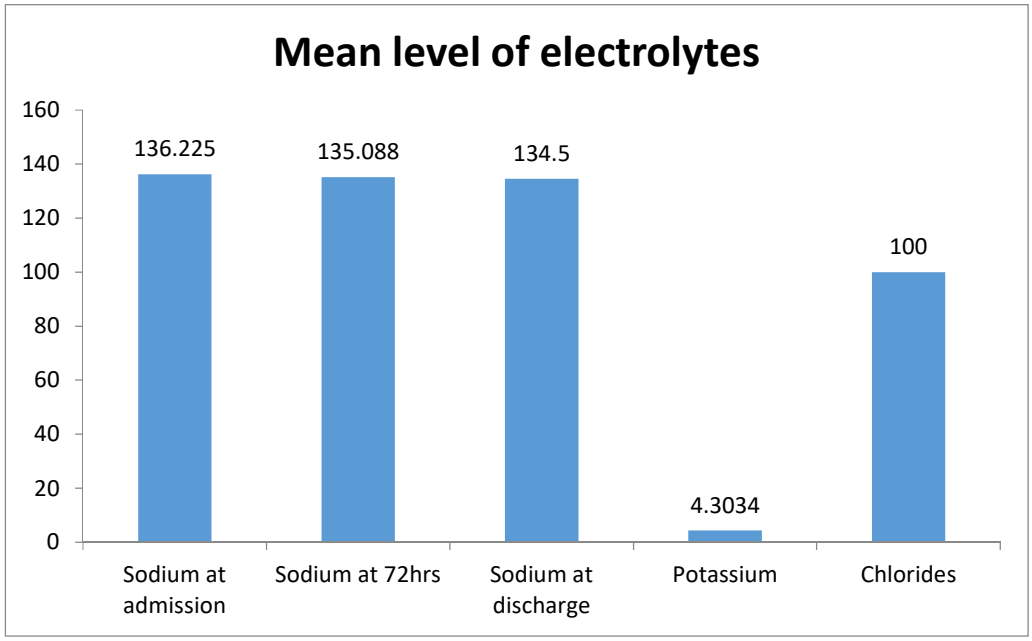

Figure 10: Showing the mean level of sodium, potassium and chloride among study participants

Table 11: Showing the 2D-ECHO findings among study participants

	Minimum	Maximum	Mean	SD
2D-Echo LVEF	25.0	60.0	43.388	7.7631

Table 12: Comparison of the 2D ECHO findings with the presence of hyponatremia

	Hyponatremia at admission				p-value
	Absent		Present		
	Mean	SD	Mean	SD	
2D-Echo LVEF	44.0	8.0	42.4	7.4	0.343

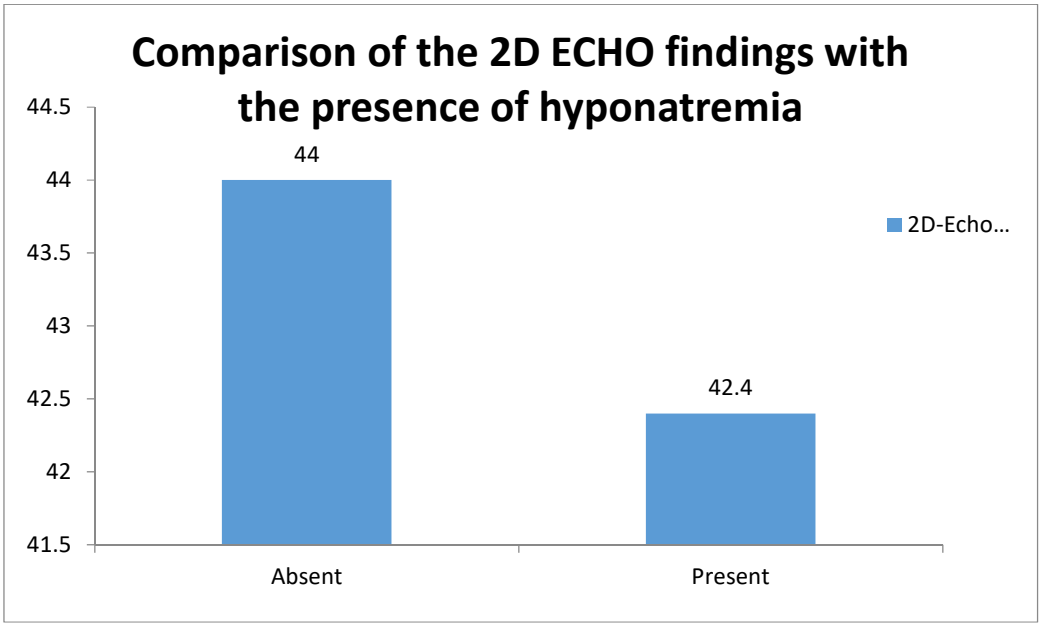

Figure 11: Comparison of the 2D ECHO findings with the presence of hyponatremia

DISCUSSION

With an estimated three million cases yearly, acute myocardial infarctions is one of the leading causes of mortality in the developed nations, accounting to more than a million fatalities in the US per year. Non-ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI) are the two types of acute myocardial infarction. NSTEMI and Unstable angina are comparable. Cardiovascular indicators, however, are not increased. [20–22]

Hyponatremia in these patients can be attributed to a number of factors, including sympathetic overstimulation brought on by decreased stroke volume and consequent arterial underfilling and the neurohormonal axis activation of the (RAAS) renin-angiotensin-aldosterone system. The predictive significance of blood sodium concentrations in STEMI and HF patients upon admission and throughout hospitalisation has been investigated in a number of observational studies and clinical trials. [10,13,15]

In current study total of 80 patients who meet the eligibility requirements are included., with mean age of 59.17±12.09yrs of age. Among the included patients 28.8% were female and 71.3% were male patients, with male preponderance. By occupation 36.3% were farmers, 26.3% were housewives, 12.5% were business persons, 6.3% were coolie, and 3.8% were cook and driver. Other included carpenter, factory worker, shopkeeper, software engineer and retired teacher. Comorbidities were present in 51.3% of the patients included in the study. In similar to present study Goldberg A et al., documented the mean age of patients to be 59.0±12yrs with male preponderance in the study.[14]

Prevalence of hyponatremia at the admission was seen in 38.8% of the patients, at 72hr it was 57.5% with hyponatremia and at discharge it was 57.5% of the patients. The mean sodium level at admission was found to be 136.2±4.31, at 72hrs it was found to be 135.08±5.57 and at discharge it was 134.50±4.05meq/L.

In a study that is identical to the one at hand, 50.2% of infirms experienced hyponatremia within seven days of in-hospital registration, according to Sanchez AC et al. Patients with hyponatremia for up to 7 days had a greater rate of intensive care admission, and they also had a higher 30-day mortality. On-admission hyponatremia was linked to in-hospital mortality. [52] Hyponatremia was shown to be the most frequent electrolyte anomaly seen in routine clinical practise and having a poor prognostication value in both the infirms suffering either from heart failure & or STEMI in a study by Shah V et al. [51]

In concordance to present study Goldberg A et al., documented 11% of the patients with hyponatremia. Also Hyponatremia at the time of in-hospital admission endured to be an solitary predictor of post-discharge mortality.[14]

On assessment of ECG 31.3% had the AWMI, 20% with IWMI followed by 15.0% AWMI+QRBBB, with 10% AWMI+LWMI. The mean 2D-ECHO LVEF was found to be 43.38±7.76%. In study by Tang Q et al., it was found that on presentation, 212 (13.1%) of the 1,620 patients had hyponatremia (sodium 135 mmol/L). In-hospital mortality was higher in hyponatremia patients (13.7% vs. 7.3%, p=0.002), as was cardiac failure of (30.2% vs. 18%, $p < 0.001$). Study participants having serum sodium levels of 130 mmol/L experienced a 22.9% rate of adverse events compared to 11.0% in study participants having serum sodium of 130 to 135 mmol/L (p=0.034). Concluded that Hyponatremia is solely linked to negative in-hospital outcomes in

Chinese patients with acute ST-elevation myocardial infarction (STEMI), and the probability of in-hospital death rises with increasing severity of hyponatremia.[49]

In the study of Sajadieh A et al., 43% of infirms with plasma sodium levels lesser than 134mEq/L experienced myocardial infarction (MI). [46] According to research by Singla I et al. (odds ratio 1.98, 95% confidence range 1.35 to 2.89, $p<0.001$), patients who had hyponatremia at the time of admission had a significantly higher risk of dying or experiencing a repeat myocardial infarction in the next 30 days. [45] According to Goldberg A et al., severe hyponatremia affects the prognosis. An independent predictor of 30-day death is hyponatremia upon in-hospital registrations or the untimely onset of hyponatremia in study participants with acute ST-elevation myocardial infarction (STEMI). Additionally, more research deemed necessary to discover whether serum sodium levels be utilised as straightforward an indicator to identify patients at greater risk. [44]

In the current investigation, patients with hyponatremia upon admission had lower mean values of the ejection fraction than patients with normal sodium levels, but this difference was not statistically significant.

Hyponatremic individuals exhibited significantly higher rates of heart failure during hospitalisation (P=0.0018), long-term cardiac mortality (17.2% vs. 6.3%, P=0.19), and re-admission due to CHF (20.7% vs. 4.5%, P=0.0024) in a study by Tada Y et al. Plasma AVP concentrations were higher (4.5 vs. 2.7pg/ml, P=0.003) in the hyponatremia group and negatively linked with serum sodium concentrations (r=-0.28, P=0.02) in the hyponatremia group. Hyponatremia was common in the early stages of STEMI and was linked to both short and long-term outcomes of heart failure. Hyponatremia in STEMI patients may be brought on by non-osmotic AVP

production.[48]

Hyponatremia during admission was linked to a higher risk of post-discharge heart failure readmission, according to a study by Goldberg A et al. (HR 1.6; 95% CI, 1.1-2.6; P =.04). For every 1-mEq/L fall in serum sodium level, the adjusted HR for mortality or heart failure was 1.12 (95% CI, 1.07-1.18; P<.001). Independent of other clinical predictors of a poor prognosis, such as left ventricular ejection percentage (LVEF), hyponatremia, when present in the early stages of an ST-elevation myocardial infarction (STEMI), is a predictor of continued mortality & hospitalisation for heart failure after being discharged from the hospital. [14]

Although it is unclear whether hyponatremia is merely a sign of illness or the main factor contributing to the poor prognosis in the patients with STEMI & HF, Shah V. et al study made the following statement: "One thing is certain: timely identification of patients at risk of developing hyponatremia could aid in the initiation of early treatment." [51] These results led Lazzeri C et al., to draw the conclusion that One indication that there are more sick people is the presence of hyponatremia during the acute phase of ST-segment elevation myocardial infarction. [50] Hyponatremia is an independent predictor of mortality & myocardial infarction (MI) in the middle-aged & the elderly population, according to Sajadieh A et al. [46]

CONCLUSION

The present study reports Prevalence of hyponatremia at the admission was seen in 38.8% of the patients, at 72hr it was 57.5% with hyponatremia and at discharge it was 57.5% of the patients. Also hyponatremia was associated with the presence of lower ejection fraction documented on 2D-ECHO.

LIMITATIONS

- One of the limitations of the study is that this being a single centric study conducted among small sample size.

The study also did not consider for a long-term follow-up of patients, due to the reason that this being a time bound study part of post-graduation. Hence to strengthen the present research findings, we recommend the larger study conducted at multiple health centres to determine the hyponatremia in MI patients and effect of timely intervention on the outcome of the patients from intensive care unit and hospital discharge.

STRENGHTS

- The present study highlights the prevalence of hyponatremia among patients with ST elevation Myocardial infarction and also important determinant of outcome from the intensive cardiac care unit.
- The study also strengthens the need for monitoring and also management of the electrolyte derangements among these patients.

SUMMARY

This cross-sectional study was conducted on patients who met the inclusion criteria and were admitted to the intensive cardiac care unit of the KLE's Dr. Prabhakar Kore Hospital and the MRC in Belagavi. Patients with acute myocardial infarction report to the intensive care unit (ICCU) with chest pain that lasts longer than 20 minutes with diagnostic ECG abnormalities, including: 1) ST elevation of less than 1 mm in more than two adjacent limb leads. 2) Two contiguous precordial leads in both sexes older than 18 years that had ST elevation of more than 2 mm were considered.

The current study sought to determine the prevalence of hyponatremia among patients with acute ST-elevation myocardial infarction admitted to the Dr. Prabhakar Kore hospital's intensive cardiac care unit.

- In the current study, a total of 80 patients who met the eligibility requirements were enrolled, with a mean age of 59.17±12.09 years.
- Among the included patients 28.8% were female and 71.3% were male patients, with male preponderance.
- By occupation 36.3% were farmers, 26.3% were housewives, 12.5% were business, 6.3% were coolie, and 3.8% were cook and driver.
- Others included carpenter, factory worker, shopkeeper, software engineer and teacher.
- 51.3% of the study participants' patients had comorbidities.

- Prevalence of hyponatremia at the admission was seen in 38.8% of the patients, at 72hr it was 57.5% with hyponatremia and at discharge it was 57.5% of the patients.
- The sodium mean level at admission was found to be 136.2±4.31, at 72hrs it was found to be 135.08±5.57 and at discharge it was 134.50±4.05meq/L.
- On assessment of ECG 31.3% had the AWMI, 20% with IWMI followed by 15.0% AWMI+QRBBB, with 10% AWMI+LWMI.
- The mean 2D-ECHO LVEF was found to be 43.38±7.76%.
- In the current investigation, patients with hyponatremia upon admission had lower mean values of the ejection fraction than patients with normal levels of sodium, but this difference was not statistically significant.
- The mean 2D ECHO LV EF was found to be 42.4±7.4 in hyponatremia patients compared with normal sodium as 44±8.0%. ($p>0.05$)

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ANNEXURE I – CONSENT FORM

TITLE OF RESEARCH STUDY: "A STUDY ON INCIDENCE OF HYPONATREMIA IN PATIENTS WITH ACUTE ST ELEVATION MYOCARDIAL INFARCTION ADMITTED IN INTENSIVE CARDIAC CARE UNIT OF KLE'S DR. PRABHAKAR KORE HOSPITAL & MRC ; BELAGAVI"

Principal Investigator: -

_____

POST GRADUATE STUDENT,

DEPARTMENT OF GENERAL MEDICINE,

JNMC, BELGAVI.

Guide: -

_____

Professor, Dept. of General Medicine

JNMC-Belagavi.

Introduction and Purpose: - Hyponatremia has been shown to be a predictor of CVS mortality among patients with heart failure. Hyponatremia is common after Myocardial Infarction and clinical outcome is accompanied by rise in plasma sodium concentration. This study is being undertaken to determine the incidence of hyponatremia in setting of Acute ST Elevation MI and determine its effect on the outcome

PROCEDURE:

If you agree to be part of the research study, you will be asked the relevant history and will be subjected to relevant clinical examination and investigations. You will also have to give blood for the necessary investigations.

Risk and Benefits:

The only risk and possible discomfort you might get is while taking blood from your arm for the investigations. It may cause swelling, pain, redness (rarely happens) at the site from where the blood is drawn.

You may not be benefitted by these investigations but you will be part of this study which is going to be useful to others in the future.

Alternatives:

Taking part in this study is voluntary. You may choose not to take part in this study.

If you decide to take part and later change your mind to withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients for your condition.

Privacy and Confidentiality:

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy:

Does not apply to this research

Financial incentives for participation:

No additional costs shall be incurred upon you for the purpose of this study.

Its purely being done with the idea of research and all the cost of the study will be borne by the investigator.

Authorization to publish the results:

The results of the study would be forwarded to the KLE University, Belagavi as part of requirement towards the completion of MD degree, review and publication.

In case of the queries during study or in future you may contact following persons

Dr. HARSHA HEGDE
Head of Ethical Committee
for
Human Research
JNMC, Belagavi.

CONSENT FORM

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has Been read to me, this consent form and have had all the questions answered

Signature / Left Thumb print of the Participant or legally authorized representative

Participant's name: _____

Signature / Left thumb impression: _____

of the participant

Name of the legally authorized _____

representative / guardian

Signature / Left thumb impression: _____

Witness' name: _____

Signature / Left thumb impression: _____

Investigator's name and signature: _____

Date:

Place:

ANNEXURE- II

PROFORMA

A STUDY ON INCIDENCE OF HYPONATREMIA IN PATIENTS WITH ACUTE ST ELEVATION MYOCARDIAL INFARCTION ADMITTED IN INTENSIVE CARDIAC CARE UNIT OF DR. PRABHAKAR KORE HOSPITAL; KLE UNIVERSITY BELAGAVI

CASE NO:

_____

NAME:

_____

AGE/SEX:

_____

IP NO.:

_____

ADDRESS:

_____

OCCUPATION:

_____

COMPLAINTS AT PRESENTATION:

_____

_____

_____

PAST HISTORY:_____

_____

_____

FAMILY

HISTORY:_____

_____

PERSONAL

HISTORY:_____

_____

TREATMENT

HISTORY:_____

_____

_____

PHYSICAL

EXAMINATION:_____

_____

GENERAL

CONDITION:_____

_____

_____

PALLOR-

YES/NO:_____

ICTERUS-

YES/NO:_____

LYMPHADENOPATHY-

YES/NO:_____

CYANOSIS-

YES/NO:_____

CLUBBING-

YES/NO:_____

EDEMA-

YES/NO:_____

VITALS:_____

TEMPERATURE:

_____

PULSE:_____

RESPIRATORY RATE:

_____

BLOOD

PRESSURE:_____

RASH:_____

PURPURA:_____

SYSTEMIC

EXAMINATION:_____

R.

S.: _____

C.V.S.:_____

P.A.: _____

C.N.S.:_____

INVESTIGATIONS:

HB –	**T. BILIRUBIN -**
NEUTROPHILS –	**D.BILIRUBIN-**
LYMPHOCYTES-	**ALBUMIN -**
PLATELET COUNT-	**ALP –**
PCV–	**SGOT -**
RBC-	**SGPT -**
WBC –	**A/ G RATIO**
MCV-	**PT -**
MCHC-	**control-**
MHC-	**test -**
ESR-	**INR –**
Monocytes -	**Creatinine –**
Eosinophils -	**Urea -**
Basophils –	**Sodium at admission**
	Sodium at 72hrs post admission
	Sodium at the time of discharge-
	Potassium-
	Chloride -

ANNEXURE III – MASTERCHART

Case No	Name	Age	Sex	Occupation	Chief Complaints	Co-morbidities	Habits	Pulse Rate - bpm	Respiratory Rate - cpm	SBP	DBP	HB	Creatinine	Urea	Sodium at admission	Sodium at 72 hrs	Sodium at discharge	Pottassium	Chlorides	2D Echo - LVEF %	ECG
1	Yallappa Alavane	55	M	Business	Chest pain - 3 days	NIL	Smoking,Tobacco chewer	150	36	80	80	12.7	4.42	85	137	143		4.6	101	30	AWMI,IWMI, AF with FVR(QRBBB)
2	Allappa Alakanur	62	M	Retired Teacher	Dyspnoea on exertion - 2 days	NIL	NIL	92	26	150	90	14.5	0.81	19	135	138	133	4.18	98	40	AWMI
3	Basappa Honakuppi	60	M	Farmer	(Chest pain, Breathlessness, Sweating) - 6 hours	NIL	Smoking	70	30	110	70	14.2	1.83	74	143	138	135	4.32	106	45	AWMI
4	Devakka Dhere	65	F	Coolie	(Chest pain, Sweating) - 1 day	T2 DM - 6 months, HTN - 20 years	Tobacco chewing	100	28	150	80	10.3	1.1	20	134	128	133	4.24	97	45	IWMI
5	Hakamharam Rathod	60	M	Coolie	(Breathlessness, Sweating) - 1 day	T2 DM - newly detected, HTN - 3 months	Smoking	130	32	150	90	13.8	0.75	33	139	140	142	4.21	103	30	AWMI
6	Nagappa Sangolli	72	M	Farmer	(Chest pain on exertion, Breathlessness) - 20 days	(T2 DM , HTM) - 8 years	NIL	110	30	160	100	9.9	1.47	32	140	142	140	4.15	102	45	AWMI, LWMI
7	Raghavendra Sudheendra	45	M	Business	(Chest pain, Palpitations, Sweating) - 8 hours	T2 DM - 8 hours	Smoking,Tobacco chewer, Alcohol consumption	96	26	200	110	19.5	2.18	75	130	128	132	4.1	101	35	AWMI, IWMI
8	Amarayya Chandalimath	63	M	Farmer	Chest pain - 12 hours, Sweating - 30 minutes	NIL	NIL	87	22	130	90	12.1	1.01	20	136	133	135	3.5	99	50	AWMI
9	Prabhavathi Mahadev Desai	45	F	Housewife	(Breathlessness, Chest Pain) - 12 hours	HTN - 6 years	NIL	102	32	140	90	13.1	1.48	97	128	145	142	4.52	88	45	AWMI, QRBBB
10	Shivaji Kamble	53	M	Farmer	Chest discomfort - 20 hours, Vomiting - 2 episodes	NIL	NIL	100	32	80	80	13.2	2.05	42	135	143	130	2.77	98	30	IWMI
11	Ganapatsa Dani	62	M	Business	Chest pain - 2 days	RVD positive	Alcohol consumption	70	22	140	90	14.9	0.84	26	139	135	140	3.66	103	50	IWMI
12	Husensab Nadaf	71	M	Cook	Chest pain radiating to back - 1 day	NIL	NIL	84	28	130	70	14.5	1.32	27	142	136	137	4.27	104	60	AWMI, QRBBB
13	Manohar Patil	47	M	Business	Chest pain - 3 days, Palpitations - 2 hours	NIL	NIL	89	30	120	80	14.6	1.08	18	139	133	130	3.55	101	45	IWMI, RVWMI, PWMI

14	Sanju Patil	45	M	Shopkeeper	(Chest pain, Sweating) - 3 days	T2 DM - 10 years, Old AWMI with EF 40% - 4 years	NIL	110	30	110	80	12.7	0.94	18	135	132	136	4.33	100	35	IWMI
15	Vithal Bandivaddar	37	M	Driver	Chest pain - 1 day, (Sweating, Vomiting) - 2 hours	NIL	NIL	80	26	140	80	15.1	1.08	19	136	133	142	4.29	99	50	AWMI, PWMI
16	Veerabhadrappa Jadar	86	M	Cook	Chest pain - 3 hours, Sweating, Pain abdomen	NIL	Tobacco chewing	75	29	60	60	10.9	1.47	33	123	126	128	4.68	86	30	AWMI, IWMI, QRBBB
17	Mehboobsubani Gadad	56	M	Farmer	(Chest discomfort, Burning sensation, Sweating) - 3 days	T2 DM - 10 years, HTN - 3 years	Tobacco chewing, Smoking	130	32	130	90	16.5	0.76	30	132	128	133	4.2	94	45	AWMI, QRBBB
18	Vilas Patil	59	M	Farmer	Chest pain - 5 days, Breathlessness - 2 days	T2 DM - 3 years	Alcohol consumption	110	26	110	70	14.1	0.88	19	138	132	140	4.23	101	45	IWMI
19	Veeresh Jhadav	56	M	Driver	Chest pain - 1 day, Breathlessness - 1 day	NIL	NIL	89	24	160	100	10.9	1.3	33	130	126	128	4.3	98	50	AWMI
20	Suman Nagesh Patil	61	F	Housewife	Chest pain - 1 day, Sweating - 4 hours	HTN - 7 years	NIL	84	20	160	100	8.4	1.5	75	137	132	141	4.7	101	45	PWMI
21	Sugandha Shrimanth Pandi	58	F	Housewife	(Chest pain, Sweating) - 8 hours	NIL	NIL	90	20	110	70	10.7	5.43	80	137	130	131	6.91	98	30	AWMI, IWMI
22	Mahantesh Angadi	55	M	Business	Chest pain - 10 hours, Profuse sweating - 3 hours	NIL	NIL	101	28	130	90	11.4	1	18	137	135	136	3.98	100	50	AWMI
23	Gangappa Shindholli	63	M	Farmer	(Chest pain, Sweating) - 1 day	T2 DM - 3 months	Smoking	90	30	130	70	9.9	1.8	13	139	136	140	3.9	106	45	AWMI
24	Laxman Nesrikar	60	M	Cook	(Chest pain, Sweating, Breathlessness) - 6 hours	NIL	NIL	82	26	150	100	12.6	1.01	29	140	136	135	5.45	103	40	AWMI, PWMI
25	Chidanand Dundappa	55	M	Farmer	Breathlessness - 2 days, (Chest pain, Sweating) - 1 days	NIL	NIL	94	22	140	90	15.9	0.73	33	137	132	136	4.06	99	50	IWMI
26	Laxman Hanumath Mareli	63	M	Coolie	Chest pain - 3 days	NIL	NIL	98	30	110	80	14.1	1.63	64	137	133	130	3.46	101	45	AWMI
27	Sharad Hosagouda	46	M	Business	(Chest pain, Breathlessness) - 6 hours	NIL	Smoking, Alcohol consumption	84	30	110	80	16.5	0.68	19	133	135	135	4.5	99	50	IWMI
28	Mallikarjun	45	M	Farmer	Chest pain - 1 hour, Vomitting - 1 episode	HTN - 3 years	NIL	98	22	130	80	16	0.7	28	133	129	131	4	90	50	AWMI, QRBBB
29	Mallappa	85	M	Farmer	Chest pain - 2 hours	NIL	NIL	74	20	100	70	13.8	1.2	40	137	140	130	4.5	103	40	IWMI
30	Sasuraj More	55	M	Business	Chest pain - 2 days, Sweating - 1 hour	NIL	NIL	80	19	130	90	14.9	0.73	15	142	137	133	2.83	106	40	PWMI
31	Kusuma Ranakambi	76	F	Housewife	Chest pain- 1 day,(palpitations, unresponsiveness) - 1hour	T2DM-15Years, HTN-15Years	NIL	110	20	90	5	14.7	0.59	33	130	129	133	3.7	97	45	IWMI,LWMI
32	Srinivas Waddar	35	M	Shopkeeper	Chest discomfort- 1 day, pain abdomen -3bdays	NIL	NIL	80	16	124	94	13.9	0.56	20	138	135	136	3.99	103	50	AWMI

33	Gurubasu Lakappa	48	M	Carpenter	(Chest discomfort, Breathelessness on exertion)-30 days	NIL	NIL	79	20	140	90	13.4	1.43	30	137	130	133	5.12	100	45	AWMI
34	Sudhir Devappa	40	M	Software Engineer	(Palpitations, Sweating)- 2 hours	NIL	Smoking, Alcohol consumption	86	14	130	90	18.2	0.8	14	139	138	132	4.06	104	40	PWMI
35	Sushila Vittal	68	F	Housewife	Chest pain, Discomfort-2	NIL	NIL	79	16	130	70	13.3	1.04	25	139	142	137	4.53	104	35	AWMI,QRBBB
36	Safurabi Ismail	81	F	Housewife	Chest pain- 2 days, Palpitations, Altered sensorium-1 hour	(T2DM, HTN)- 20 years	NIL	118	26	90	60	11.6	3.43	96	135	128	129	5.81	113	25	IWMI,AF
37	Rajendra M S	45	M	Farmer	(Chest pain, Breathelessness)- 4 days	HTN- 6 Years	Smoking, Alcohol consumption	96	16	140	90	19.7	2.04	46	131	135	136	4.39	93	45	AWMI
38	Siddanna Doddamani	62	M	Farmer	(Chest discomfort, Dyspnoea on exertion) - 3 days	NIL	NIL	82	16	120	90	12.6	0.92	43	132	133	136	4.95	98	50	AWMI
39	Mahantayya	63	M	Teacher	Chest pain - 1 days	NIL	NIL	76	18	130	90	15	0.93	26	133	140	141	4.19	103	45	AWMI, QRBBB
40	Annasab Khot	74	M	Farmer	Chest pain - 14 hours	NIL	Tobacco chewing	88	18	110	70	13.7	1.4	33	141	136	138	4.49	101	60	AWMI, QRBBB
41	Rajendra Gouda Patil	63	M	Farmer	Chest pain- 1 days	HTN-5 years	NIL	78	18	120	70	14.8	1.22	29	138	140	133	4.34	102	50	AWMI,LWMI
42	Laxmi	65	F	Housewife	(Chest pain, breathelessness)- 3 days, Palpitations- 2 hours	NIL	NIL	88	16	128	84	10.1	3.4	27	141	140	132	3.6	108	36	IWMI
43	Yamanavva	60	F	Housewife	(Chest pain, breathelessness on exertion)- 2 days, palpitations-1 day	HTN-10 Years	NIL	86	20	140	80	12.5	0.8	27	141	135	139	4.34	107	45	AWMI,QRBBB
44	Laxman Bagi	60	F	Farmer	Breathelessness -1 day	(T2DM, HTN)-10 years, CKD-3 years	Smoker	88	22	130	80	10.4	4.05	78	142	139	139	7.46	103	30	AWMI
45	Hanumantappa Kamanoor	74	M	Farmer	Breathelessness on exertion- 3days, Profuse sweating- 4 hours	(HTN, COPD)- 10 years	Smoker, Tobacco Chewing	130	28	152	70	14.1	0.76	18	137	135	138	4.66	96	35	AWMI
46	Patrappa Kottershetti	59	M	Teacher	Chest pain - 2 days, Giddiness - on & off	NIL	NIL	116	26	90	60	10.7	1.12	47	135	130	133	3.88	97	40	IWMI
47	Shivasharan	52	M	Driver	Dyspnoea on exertion - 1 month, (Chest pain, Sweating) - 1 day	T2 DM - 12 years, HTN - 8 years	Tobacco chewer, Alcohol consumption	88	24	150	90	15.6	1.1	32	137	135	133	3.2	104	43	AWMI
48	Laxmavva	75	F	Housewife	(Breathlessness,Chest pain) - 1 day	(T2 DM, HTN) - 2 years	Tobacco chewing	80	22	130	90	10.3	0.7	34	134	154	133	3.12	95	50	IWMI
49	Baganna Kurbar	62	M	Farmer	(Chest pain, Vomitting) - 2 days	T2 DM - 6 years	Smoking, Alcohol consumption	86	20	90	60	13.9	1.05	46	140	136	133	3.87	99	35	AWMI
50	Vishnu Gondhakat	60	M	Coolie	Chest pain - 3 days	T2 DM - 8 years, HTN - 5 years	NIL	74	16	110	80	10.2	0.88	14	140	134	130	3.36	97	60	AWMI
51	Khatumbi Shaiwale	51	F	Housewife	(Chest pain, Breathlessness, Profuse sweating) - 8 days	(T2 DM, HTN) - 13 years	NIL	68	20	140	70	12.2	0.96	23	136	133	133	3.76	100	60	AWMI,QRBBB

52	Satyappa Kagavada	55	M	Farmer	Dyspnoea on exertion - 10 days	NIL	NIL	114	24	100	60	12.6	2.64	131	133	130	128	5.22	99	40	AWMI, FVR
53	Meenakshi Patil	74	F	Housewife	Chest pain - 4 hours, Vomitting - 1 episode	NIL	NIL	108	20	140	90	9.6	2.01	66	130	128	133	5.6	99	45	AWMI
54	Virupaxi Samai	64	M	Farmer	Chest pain - 1 day, Sweating - 4 hours	NIL	Smoking	96	20	140	90	14.1	1.01	20	134	133	136	3.8	96	50	IWMI
55	Laxmavva	75	F	Housewife	(Chest pain, Sweatting) - 2 days	(T2 DM, HTM) - 20 years	NIL	80	20	130	80	10.3	0.71	34	134	150	135	5.8	98	50	IWMI
56	Yallappa K	24	M	Factory worker	(Chest pain, Palpitations)- 2 h ours	NIL	NIL	97	28	130	100	15.3	2.11	12	143	143	136	4.02	101	50	AWMI
57	Rafiq Yadawad	45	M	Farmer	(Chest pain- 6hours),Vomiting-1 episode, loose stools- 3 episodes	NIL	Tobacco Chewing	78	18	110	70	13.9	0.75	28	133	140	137	4.55	99	50	AWMI,LWMI
58	Vittal Bairoji	58	M	Farmer	Breathlessness on exertion-10 days, pain abdomen-2 days	T2DM- 6 years	Smoking, Alcohol consumption	80	24	130	70	13.6	0.93	24	134	130	132	3.64	97	45	AWMI,LWMI
59	Shakuntala Hiremath	75	F	Housewife	(Chest pain, breathelessness)- 2 days	T2DM-10 Years, HTN-14 Years	NIL	90	20	100	70	11.8	0.64	24	133	131	122	4.45	91	45	AWMI,QRBBB
60	Sangeeta Walake	48	F	Housewife	Breathelessness- 1 week	NIL	NIL	135	28	90	60	12.4	0.96	30	134	128	129	4.15	95	30	AWMI,LWMI
61	Kavita Mahagaonkar	60	F	Housewife	Chest pain- 5 hours, Sweating ,Vomiting- 4 episodes	T2DM- 5 Years	NIL	82	18	130	80	12.3	0.92	66	136	137	133	4.77	100	45	AWMI
62	Malathi Kapileshwari	61	F	Housewife	Vomiting- 8 episodes, sweating- 4 hours	(T2DM, HTN)- 12 Years	NIL	36	32	90	60	11.9	2.08	67	139	133	136	5.32	105	45	IWMI,CHB
63	Prasanjit Roy	46	M	Software Engineer	(Chest pain, sweating) - 1 day	NIL	Smoking, Tobacco Chewing	65	22	140	80	16	1.3	55	144	142	135	3.9	110	45	PWMI
64	Suvarna Virupaxi	54	F	Housewife	Breathelessness -1 hour	T2DM- 2 Months	NIL	74	29	110	60	9	3.6	70	140	136	137	5.9	100	35	AWMI
65	Shankar Katti	65	M	Farmer	(Chest discomfort, breathelessness)- 1 day	(T2DM, HTN)- 6 Years	Smoking	98	20	158	80	14.5	1.16	26	133	135	133	3.99	95	40	AWMI, LWMI
66	Vidhya Kakade	52	F	Housewife	Dyspnoea on exertion-3 days, Chest pain- 1 day	T2DM-1 Month, HTN- 5 Years	NIL	75	16	140	100	13.9	0.89	30	136	135	133	3.76	100	40	AWMI, LWMI
67	Pampangouda Patil	75	F	Housewife	Breathelessness- 1 day	T2DM-15 Years, HTN-12 Years	Smoking, Alcohol consumption	110	24	150	90	12.4	1.58	31	143	145	136	4.12	105	35	IWMI,LWMI
68	Prabhakar Lohar	74	M	Farmer	Dyspnoea on exertion-1 month, Giddiness- 2 days	HTN-1 Year	NIL	96	17	130	80	11.6	1.32	43	138	133	135	3.88	103	60	AWMI,QRBBB
69	Mohamad Yunus	42	M	Coolie	Chest Pain- 12 hours	NIL	Tobacco Chewer	72	18	120	80	15	0.74	14	138	135	132	4.03	97	50	AWMI
70	Rajashree Chapi	55	F	Housewife	Chest Pain- 4 days	CA BREAST	NIL	76	20	Apr-00	70	10	0.32	16	139	144	140	4.16	100	45	AWMI

71	Nemanna J.A	72	M	Farmer	Dyspnoea on exertion-10 days, Chest pain-5 days	NIL	NIL	104	22	100	60	12.4	1.15	71	136	129	138	4.38	96	42	IWMI,LWMI
72	Jyavanth Kolkar	34	M	Business	(Chest pain, sweating, palpitations) - 5 hours	NIL	NIL	90	20	160	90	18.2	0.85	14	137	130	132	4.02	100	45	IWMI
73	Siddangouda Patil	50	M	Farmer	Chest Tightness- 3 hours	T2DM-8 Years	Smoking, Alcohol consumption	76	18	126	76	12.9	1.02	25	135	138	131	3.9	98	40	IWMI
74	Jagannath Birje	72	M	Business	Chest Pain- 6 hours, sweating-2 hours	T2DM-12 Years, IHD-8 Years	NIL	120	36	80	50	9.9	3.2	98	118	124	125	5.9	103	30	IWMI,PWMI
75	Sumitra Nayakar	59	F	Housewife	(Chest pain, Breathelessness)-3 hours	NIL	NIL	96	16	120	80	11.2	0.56	31	135	138	133	3.84	102	45	AWMI,QRBBB
76	Shivappa Mushannavar	62	M	Farmer	Chest Pain- 6 hours	HTN- 3 Years	Tobacco Chewer	76	16	150	90	14.3	0.86	12	140	133	138	4.6	106	40	AWMI
77	Ningappa Mankale	63	M	Farmer	Giddiness-4 days, Sweating-4 hours	NIL	NIL	76	18	110	70	14.6	1.07	18	133	128	129	3.63	96	50	IWMI
78	Somappa Goravanakoll	75	M	Farmer	Chest pain-1 hour	HTN- 2years	Tobacco Chewer	100	22	140	80	14.6	0.99	44	136	140	139	4.23	102	35	AWMI,LWMI
79	Venkappa Patil	75	M	Farmer	Breathelessness- 8 days, Pallpitations-1hour	T2DM-30 Years, HTN-30 Years, Old CVA-1 Year	Tobacco Chewer	100	26	140	80	13	0.67	27	138	137	138	3.33	96	40	IWMI,LWMI
80	Amar Jayanth Godase	52	M	Business	Chest Pain- 1 day	NIL	Tobacco Chewer	84	16	130	70	13.9	2.02	118	140	131	140	4.17	102	45	AWMI