THE STUDY OF OUTCOME HYPONATREMIA AMONG STEMI WITH REDUCED LEFT VENTRICULAR FRACTION

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ABSTRACT

BACKGROUND
Hyponatremia is the most common electrolyte disorder in hospitalized patients in diverse clinical settings. In fact, the neurohormonal activation that accompanies acute myocardial infarction is similar to that which accompanies heart failure. Hence, we aimed to investigate the prognostic importance of hyponatremia in the setting of acute ST elevation MI with reduced ejection fraction and to determine its usefulness in predicting short term survival.

METHODS
We used a prospective database consisting of all admissions of patients with acute ST-elevation. Qualifying patients underwent detailed history and clinical examination. The primary end point was all cause mortality within 30 days following myocardial infarction.

RESULTS
The odd's ratio for 30-day mortality was found to be high in hyponatraemic groups with reduced ejection fraction compared to normonatraemic group as well normal ejection fraction with hyponatraemia. Multivariate analysis was done which identified hyponatremia on admission or early development of hyponatremia in STEMI with reduced ejection fraction as a significant independent predictor of 30-day mortality.

CONCLUSION
In our study we concluded that hyponatremia on admission or early development of hyponatremia in patients with acute ST elevation myocardial infarction with reduced ejection fraction is an independent predictor of 30-day mortality.

BACKGROUND
Hyponatremia is frequently a marker of significant underlying disease and is therefore associated with poor short-term prognosis, even when the serum sodium level is only mildly reduced. Myocardial infarction is a well-known clinical entity. It is one of the most fatal diseases which is worldwide in distribution, affecting all races and nationalities. Because myocardial infarction may strike any individual during the most productive years, it can have profound deleterious, psychological and economic ramifications. Heart failure is a disabling and growing disease associated, associated with high morbidity and mortality rates and with annually increasing costs. Many factors are implicated in the pathogenesis of hyponatremia in patients with heart. Heart failure reduces cardiac output and results in arterial under filling, which induces the activation of the sympathetic nervous system (SNS). This

leads to peripheral and renal vasoconstriction and decreases glomerular filtration rate, effects that combined with arterial under filling result in increased reabsorption of sodium and water and induce the activation of the renin-angiotensin-aldosterone system (RAAS).

Myocardial infarction causes more deaths and disability and incurs greater economic costs than any other illness in the world. It is the most common, serious chronic, life threatening illness.

Despite impressive studies in the diagnosis and management over the past 4 decades, acute MI continues to be major public health problem in the industrialized world and is becoming an increasing important problem in developing countries. With a decline in infectious disease related death accompanied by accelerated economic development and life style change promoting atherosclerosis, developing countries especially India are expected to experience a sharp increase in ischemic Heart disease and AMI. Given the wide disparity of available resources to treat AMI in developing countries, major efforts are needed to strengthen primary prevention programmes at community level.[1]

Obesity, insulin resistance, and type 2 diabetes mellitus are increasing and are powerful risk factors for IHD and AMI. These trends are occurring in the general context of population growth and as a result of the increase in the average age of the world's population. With urbanization in

countries with emerging economies and a growing middle class, elements of the energy-rich Western diet are being adopted. As a result, the prevalence of risk factors for IHD and AMI and the prevalence of IHD and AMI itself are both increasing rapidly, so that in analyses of the global burden of disease, there is a shift from communicable to noncommunicable diseases. Population subgroups that appear to be particularly affected are men in South Asian countries, especially India and the Middle East. In light of the projection of large increases in IHD and AMI throughout the world, IHD and AMI are likely to become the most common cause of death worldwide by 2020.

Hyponatremia is a common electrolyte disorder amongst hospitalized patients,[2] especially in postoperative period[3] and in patients with heart failure, nephrotic syndrome or cirrhosis.[4,5] Hyponatremia has been shown to be a predictor of cardiovascular mortality among patients with heart failure.[6,7] In fact, the neurohormonal activation that accompanies acute myocardial infarction is similar to that which accompanies heart failure[8] Hyponatremia is common after MI, and clinical improvement is accompanied rise in plasma sodium concentration.[9]

However, while the prognostic value in hyponatremia in chronic heart failure is well established,[10] data on the prognostic importance of hyponatremia in the setting of acute myocardial infarction are lacking.

Hyponatraemia is found more frequently in the early period of ST elevation myocardial infarction, and influences short as well as long term outcomes.[11] In STEMI, like congestive heart failure, arterial under filling causes stimulation of high pressure baroreceptors present in the left ventricle, arch of aorta and carotid sinus, causing stimulation of cardio regulatory centre in the brain, causing stimulation of efferent pathway of the sympathetic nervous system. Activation of this sympathetic nervous system stimulates the non-osmotic release of AVP, renin as well as angiotensin II, leading to activation of renin-angiotensin-aldosterone system.

Hormones thus released by baroreceptor stimulation reflects the severity of heart failure[2,12] & also worsens cardiac remodelling (AVP plays role in regulation of vascular tone and cardiac contractility and negatively influences cardiac haemodynamics and myocardial remodelling).[1,13]

In the early period of STEMI, release of AVP, also retards water excretion, leading to increased blood volume and thus leading to dilutional hyponatraemia. So, hypernatremia actually reflects the baroreceptor-mediated hormonal activation in an exaggerated manner and thus serves as a marker of underlying worsening haemodynamics.

Hyponatraemia, though a marker, can also contribute to the worsening haemodynamics by impairing contraction and relaxation of myocardial cells, decreasing the diastolic membrane potential and abolishing electrical coupling between myocytes.[14,15] Hence, it is worth to evaluate the incidence of hyponatraemia in patients with acute ST elevation myocardial infarction in Intensive Coronary Care Unit & to find out whether hyponatraemia serves as a poor prognostic indicator in these patients.[16,17,18]

This study was done to determine the prognostic importance of hyponatremia in the setting of acute ST elevation M with reduced ejection fraction and to determine its usefulness in predicting short term survival.

Aim of the Study
To determine the prognostic importance of hyponatremia in acute ST elevation myocardial infarction with reduced ejection fraction.

Review of Literature
Acute myocardial infarction (AMI) is a clinical syndrome that results from sudden occlusion of a coronary artery with resultant infarction and death of cardiac myocytes in the region supplied by that artery. When these patients present in emergency with ST elevation in ECG, they are referred as ST elevation myocardial infarction (STEMI). The manifestation of AMI can be varied and depends upon the distribution of the affected coronary artery, it may lead to clinically silent AMI without any manifestation to a massive infarction leading to fatal cardiogenic shock and death.[19]

The AMI has become the commonest cause of death in the developed and developing countries including India. The true incidence of AMI is difficult to judge because varied reporting pattern. Moreover, up to one-third cases die at home before they reach hospital or are examined by qualified practitioner.

The pathological identification of myocardial necrosis is made without reference to morphological changes in the coronary arterial tree or to the clinical history. [20]

Criteria for Previous Myocardial Infarction
Type 1: Spontaneous Myocardial Infarction- Spontaneous MI related to atherosclerotic plaque rupture, ulceration, fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the coronary arteries that leads to decreased myocardial blood flow or distal platelet emboli with ensuing myocyte necrosis. The patient may have underlying severe CAD but on occasion non-obstructive or no CAD.

Type 2: Myocardial Infarction Secondary to Ischemic Imbalance -In instances of myocardial injury with necrosis in which a condition other than CAD contributes to an imbalance between myocardial oxygen supply and demand, e.g., coronary endothelial dysfunction, coronary artery spasm, coronary embolism, tachyarrhythmia/brad arrhythmias, anaemia, respiratory failure, hypotension, and hypertension with or without LV hypertrophy.

Type 3: Myocardial Infarction Resulting in Death When Biomarker Values Are Unavailable Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemic changes on the ECG or new LBBB but death occurring before blood samples could be obtained, before

cardiac biomarkers could rise, or in rare cases, when cardiac biomarkers were not collected.

Type 4a: Myocardial Infarction Related to Percutaneous Coronary Intervention -MI associated with PCI is arbitrarily defined by elevation of cTn values to >5 × the 99th percentile of the URL inpatients with normal baseline values (≤99th percentile of the URL) or a rise in cTn values >20% if the baseline values are elevated and are stable or falling.

In addition, either
(1) Symptoms suggestive of myocardial ischemia,
(2) New ischemic changes on the ECG or new LBBB,
(3) Angiographic loss of patency of a major coronary artery or a side branch or .persistent slow flow or no flow or embolization, or
(4) Imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality is required.

Type 4b: Myocardial Infarction Related to Stent Thrombosis- MI associated with stent thrombosis is detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and/or fall in cardiac biomarkers values with at least one value above the 99th percentile of the URL.

Type 5: Myocardial Infarction Related to Coronary Artery Bypass Grafting- MI associated with CABG is arbitrarily defined by elevation of cardiac biomarker values to >10 × the 99th percentile of the URL in patients with normal baseline cTn values (<99th percentile of the URL).

Hyponatremia:
Definition
Hyponatraemia is defined as a serum sodium less than 135 mEq/L and is the most frequent electrolyte abnormality in clinical medicine, but is rarely seen in ambulatory patient (if present, reflects a chronic disease status).

Pseudohyponatraemia is a rare situation in which serum Na+ is low but the ECF osmolality and tonicity are normal. It is an artefact due to accumulation of other plasma constituents, viz. triglycerides or plasma proteins.

Hyponatremia is a common electrolyte disorder amongst hospitalized patients and in volume overloaded cases and its homeostasis play a major role in the cellular function in diverse clinical settings, especially in postoperative period and in patients with heart failure. Severe hyponatremia is a potentially serious and life-threatening disorder, which can lead to grave neurological complications.

A plasma Na+ concentration less than 135 mmol/L usually reflects a hypotonic state. Isotonic or slightly hypotonic hyponatremia may complicate transurethral resection of the prostate or bladder because large volumes of isosmotic (mannitol) or hypoosmotic (sorbitol or glycine) bladder irrigation solution can be absorbed and result in a dilutional hyponatremia.

The metabolism of sorbitol and glycine to CO_2 and water may lead to hypotonicity if the accumulated fluid and solutes are not rapidly excreted. Hypertonic hyponatremia usually due to hyperglycaemia or, occasionally, intravenous administration of mannitol. Relative insulin deficiency causes myocytes to become impermeable to glucose. Plasma Na+ concentration falls by 1.4 mmol/L for every 100 mg/dL rise in the plasma glucose concentration.

In general, hypotonic hyponatremia is due either to a primary water gain (and p secondary Na+ loss) or a primary Na+ loss (and secondary water gain). In the absence of water intake or hypotonic fluid replacement, hyponatremia is usually associated with hypovolemic shock due to a profound sodium deficit and transcellular water shift. The increased water ingestion and impaired renal excretion result in hyponatremia.

In contrast, thiazide diuretics lead to Na+ and K+ depletion and AVP- mediated water retention.

Hyponatremia can also occur by a process of desalination. This occurs when the urine tonicity (the sum of the concentrations of Na+ and K+) exceeds that of administered intravenous fluids (including isotonic saline). This accounts for some cases of acute postoperative hyponatremia and cerebral salt wasting after neurosurgery.

I. Hypo-osmolal Hyponatremia
A. Primary NA+ Loss (Secondary Water Gain)
1. Integumentary loss: sweating, burns.
2. Gastro intestinal loss: vomiting, tube drainage, fistula, obstruction, diarrhoea.

B. Primary Water Gain (Secondary NA+ Loss)
  1. Primary polydipsia
  2. Decreased solute intake (e.g., beer potomania)
  3. AVP release due to pain, nausea, drugs
  4. Syndrome of inappropriate AVP secretion
  5. Glucocorticoid deficiency
  6. Hypothyroidism
  7. Chronic renal insufficiency

C. Primary NA+ Gain (Exceeded by Secondary Water Gain)
  1. Heart failure
  2. Hepatic cirrhosis
  3. Nephrotic syndrome although the differential diagnosis is quite broad, most hyponatremia can be divided into hypertonic, normotonic, or hypotonic in origin. Miscellaneous causes account for the remainder of cases.
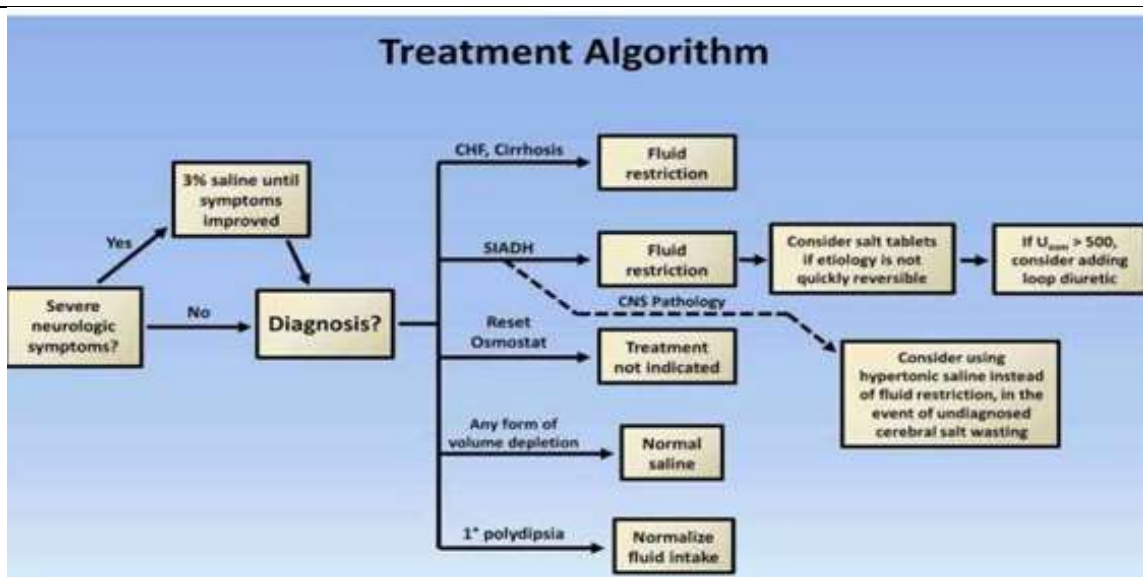
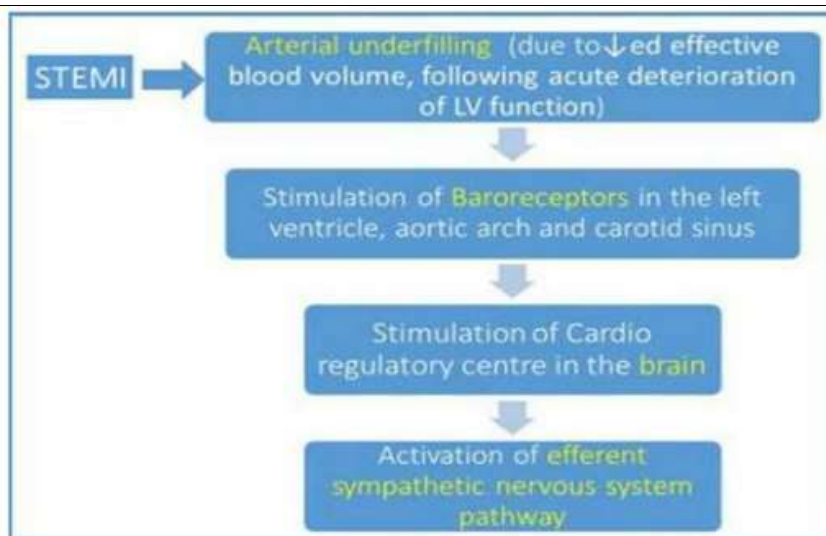**Figure 1. Flowchart Depicting Treatment Algorithm of Hypernatremia**

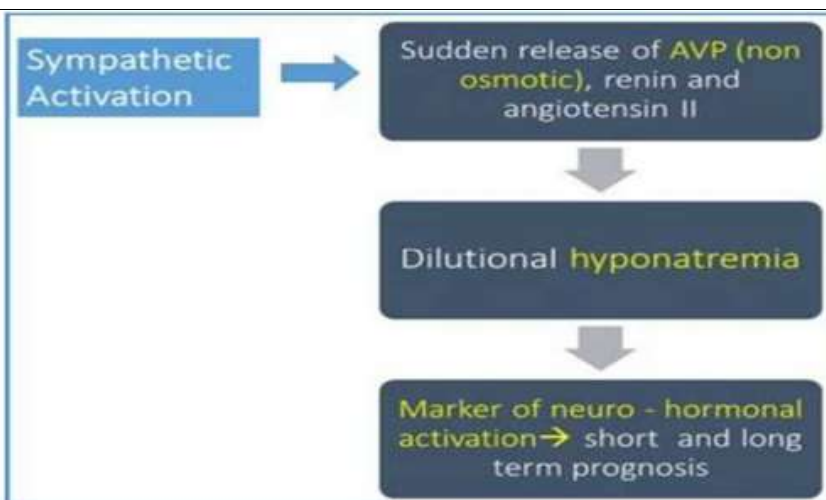**Figure 2. Neurohormonal Activation Following Acute Myocardial Infarction**

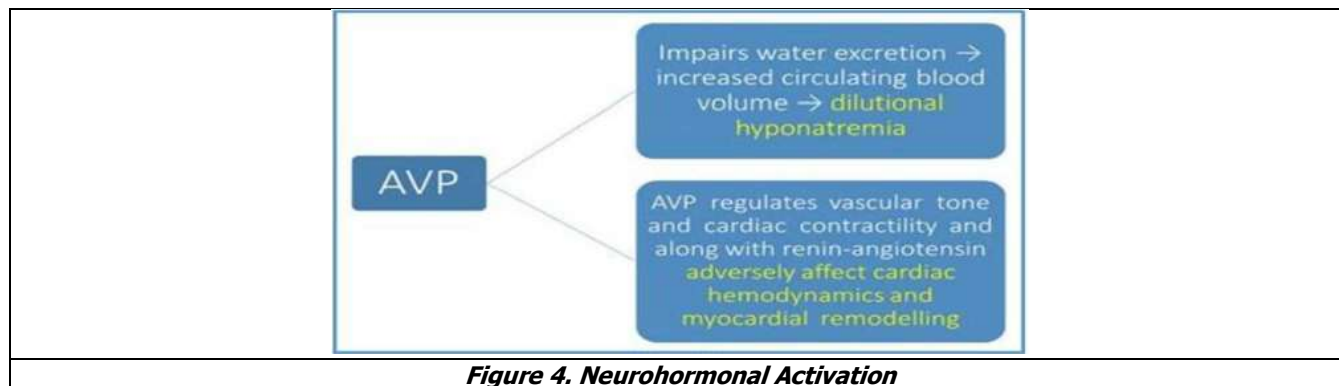**Figure 3. Neurohormonal Activation**
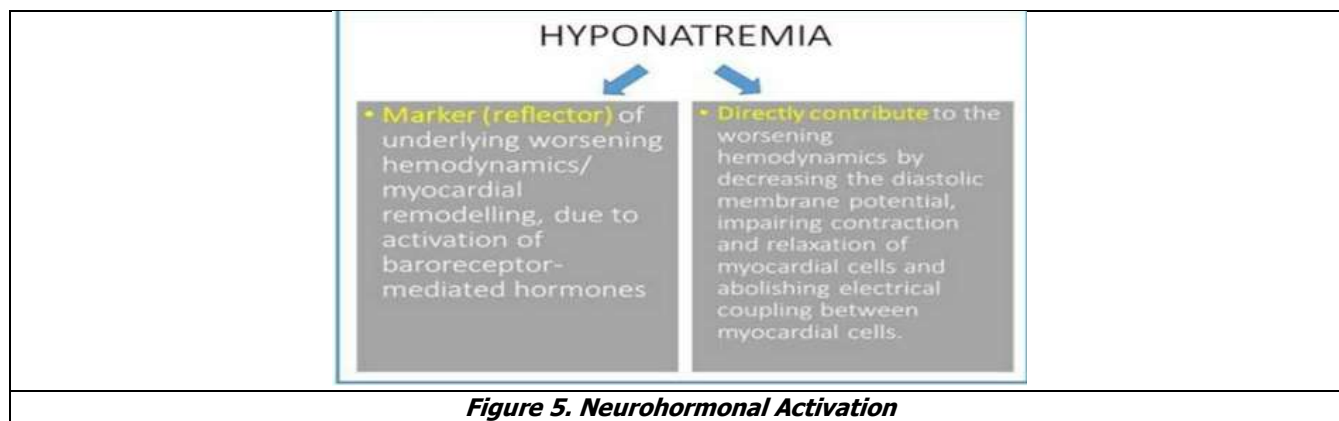
Figure 4. Neurohormonal Activation

Figure 5. Neurohormonal Activation

Several systemic metabolic changes have been reported following acute myocardial infarction in man. These are increased plasma concentration of catecholamine, cortisol, glucose, glycerol, and cyclic adenosine phosphate, decreased triglyceride concentrations, and initial fall in plasma insulin concentration followed by early return to normal values. Flear CT, Hilton P in their study of 235 consecutive patients admitted to a coronary care unit, have concluded that hyponatremia, hypochloraemia, and uraemia were common in patients with confirmed myocardial infarctions, the degree of infarctions correlating well with all the above indices of severity. They also found higher in hospital mortality rates among patients with minimal plasma sodium levels. <130 mmol/L.[9] In acute myocardial infarction, non-osmotic release of vasopressin may occur due to the acute development of left ventricular dysfunction; in response to pain, nausea, and major stress, the most common mechanisms of hyponatremia in adults; or in response to the administration of analgesics and diuretics. In this setting, vasopressin levels increase concomitantly with the activation of other neurohormones such as rennin and nor epinephrine. However, vasopressin level does not correlate with serum osmolarity in myocardial infarction, suggesting that non-osmotic mechanisms are involved. Activation of carotid baroreceptors has been implicated in the non-osmotic release of vasopressin due to arterial underfilling. In addition; increased expression of messenger RNA for vasopressin in the hypothalamus has been described. Moreover, the renal effect of vasopressin is enhanced in heart failure, as the vasopressin-regulated water in the collecting duct is up regulated.

In patients with myocardial infarction, hyponatremia may be aggravated further by the concomitant activation of the renin-angiotensin system and increased catecholamine production. These factors decrease the glomerular filtration rate and subsequent delivery of tubular fluid to the diluting segment of the nephron, further contributing to decreased renal water exertion Hormones thus released by baroreceptor stimulation reflects the severity of heart failure & also worsens cardiac remodelling (AVP plays role in regulation of vascular tone and cardiac contractility and negatively influences cardiac haemodynamics and myocardial remodelling).

In the early period of STEMI, release of AVP, also retards water excretion, leading to increased blood volume and thus leading to dilutional hyponatraemia. So hyponatraemia actually reflects the baroreceptor-mediated hormonal activation in an exaggerated manner and thus serves as a marker of underlying worsening haemodynamics.

Hyponatraemia, though a marker, can also contribute to the worsening haemodynamics by impairing contraction and relaxation of myocardial cells, decreasing the diastolic membrane potential and abolishing electrical coupling between myocytes. Hence, it is worth to evaluate the incidence of hyponatraemia in patients with acute ST elevation myocardial infarction in Intensive Coronary Care Unit & to find out whether hyponatraemia serves as a poor prognostic indicator in these patients.

Szatalowicz VL, Arnold PE, Chaimotivz C, Bichet D, Berl T, Schrier RW. In their study have shown that Vasopressin is essential for the development of hyponatremia and

arginine vasopressin levels were detectable in 30 of 37 patients with congestive heart failure. They also found that the degree of neurohormonal activation correlates with the severity of hyponatremia in patients with chronic heart failure.

Sigurdsson A, Held P, Swedberg K in their study of 55 patients with acute myocardial infarction concluded that sustained neurohormonal activation after myocardial infarction mainly occurs in patients with clinical heart failure and is related to the magnitude of myocardial damage, even in patients without heart failure.

Aziz M[14] et al in their study of 978 patients have concluded that early hyponatremia is a simple marker of neurohormonal activation during the acute phase of myocardial infarction and predicts the long-term development of heart failure and death.

Rouleau JL[15] et al in their study of 534 patients have concluded that neurohormonal activation at the time of hospital discharge in post-infarction patients is an independent sign of poor prognosis Klopotowski et al, in their study of 1858 ST-elevation MI patients concluded that hyponatremia independently correlated with in-hospital mortality. Hyponatremics had higher rates of in-hospital mortality (13.5% vs. 3.8%, p<0.001) composite of death and heart failure (27.8% vs. 18.4% p=0.022).

Their study of 671 men and women aged 55 to 75 years with no history of cardiovascular disease, stroke, or cancer, found an adverse outcome defined as death or Myocardial Infarction (MI) occurred in 43% of patients whose serum sodium level was less than 134 mEq/L. They concluded that Hyponatremia is an independent predictor of death and myocardial infarction in middle-aged and elderly patients were reported that in hospitalized survivors of acute myocardial infarction, the presence of hyponatremia at discharge was an independent predictor of 12-month mortality. The study involved 1290 patients.

METHODS
The study was carried out on patients presenting with acute ST-elevation Myocardial infarction.

Inclusion Criteria
All acute myocardial infarction patients having-
  a. Chest pain lasting more than 20 minutes.
  b. Diagnostic ECG changes with characteristic ECG alterations consisting of new pathological Q waves or ST segment and T wave changes.
  c. Elevated creatinine kinase MB levels or elevated cardiac troponin T levels.

Exclusion Criteria
  a. Acute coronary syndrome without ST elevation.
  b. Age less than 16 years of both sexes. Pre-existing renal diseases.
  c. Patients with Acute MI and cardiac failure and on Diuretic therapy.

Qualifying patients underwent detailed history and clinical examination. Patients of acute myocardial infarction received thrombolytic therapy (tissue type plasminogen activator or streptokinase).

Study End Points and Definitions
The primary end point was all cause mortality within 30 days following myocardial infarction. Mortality data after discharge but within 30 days of myocardial infarction were obtained by postcard returned by patients or their families. When no postcard was received, follow-up status was determined over telephone or visit to their house whichever possible.

Plasma sodium concentrations were determined by using an ion selective electrode auto analyser (Roche OMNI C) Hyponatremia was defined as sodium level less than 135 mmol/L (<135 mEq/L)

Statistical Method
1. Odd ratio
2. Confidence interval
3. Mean + standard deviation
4. Suitable parametric and non-parametric tests (Chi square test for non-continuous variables, Analysis of variance for continuous variables, Z test etc.)
   Univariate and multivariate logistic regression tests to determine the association between hyponatremia and 30-day mortality.

RESULTS

Range of Sodium Levels in Hyponatremia Patients	No. of Patients	Mortality
<130	7	4 (57.14%)
131-134	25	4 (16.11%)
Table 1. Showing Severity of Hyponatremia and Outcome in Terms of Mortality		

Number of patients with sodium levels less than 130 is 7 and mortality was 4(57.14%).

Number of Patients with sodium levels between 131-134 is 25 and mortality was 4(16.11%).

	Survivors	Non-Survivors	Odds Ratio	p Value
Group 1	56	2		
Group 2	22	7	3.143	0.008
Group 3	12	1	12.0	0.017
Table 2. Odds Ratio for 30 Days Mortality Group 1 Versus Other Groups				

Group 1=Patients with Normal Sodium Levels.
Group 2 = Hyponatremia on Admission.
Group 3= Hyponatremia within 72 hours.

Odds ratio for 30-day mortality was found to be high in hyponatraemic groups. (Group 2=3.143, Group 3=12.0)

	Survivors	Non-Survivors	T or x^2	p Value
n	90	10		
Age (yrs.) (mean±SD)	55.02±12.78	58.9±14.15	-0.901	0.370
Sex M	69 (76.7%)	9(90%)	1.00	0.452
Hyponatremia on admission (mean±SD)	136.96±4.02	131.63±4.24	3.963	<0.0001
Hyponatremia on at 24 hours	136.92±3.422	134±1.19	2.505	0.014
Hyponatremia on at 72 hours	136.01±3.32	132±4.372	3.508	0.001
Smoking	66(72%)	4(50%)	1.65	0.198
Diabetes	27(30%)	5(50%)	1.654	0.19
Hypertension	10(11.1%)	2(20%)	0.673	0.412
Infarct site Anterior Inferior	64(68.9%) 28(31.1%)	6(60%) 4(40%)	0.1323	0.6972
Killip Class 1	89(98.9%)	1(10%)	79.012	<0.0001
EF (%)	49.7±5.59	44.6±6.096	2.711	0.008

Table 3. Survivors and Non-Survivors Were Also Compared for Various Factors

It was seen that serum sodium levels were statistically significant in determining mortality. Mean serum level was 136.96±4.02 in the survivors and 131.63±4.24 in non survivors.

Other factors such as Killip class, hypertension, diabetes, age and sex were found to be statistically significant in determining mortality.

Multivariate analysis using logistic regression analysis was performed including variables that had p valve <0.2 in the univariate analysis to identify the variables that were independently associated with 30-day mortality.

	Mean Square	F	"p" Valve
Age	.007	.384	.537
Sex	.000	.026	.873
Smoking	.004	.210	.648
Diabetes	.015	.849	.359
Hypertension	.055	3.167	.079
Killip Class	4.948	283.242	<0.0001
Sodium on Admission	.140	8.021	.006
Sodium at 72 hours	.009	.488	.487
Ejection Fraction	.002	.125	.725

Table 4. Showing Results of Multivariate Analysis

So, multivariate analysis showed that along with other risk factors, hyponatremia was the significant independent predictor of 30-day mortality.

DISCUSSION

Our study suggests that patients presenting with acute myocardial infarction who had hyponatraemia on admission or developed hyponatremia after admission represent high risk population. Hyponatremia developed in 13 patients (13%) during the first 72 hours of hospitalization. In a similar study conducted by Goldberg et al, hyponatremia was present in 131 patients (12.5%) and hyponatremia developed in 208 (19.9%) during the first 72 hours of hospitalization. In our study a total of 10 deaths (10%) occurred within 30 days of admission. 3.44% (2/58) of patients without hyponatremia, 24.1% (7/29) of patients with hyponatremia on admission, 7.69% (1/13) of patients who developed hyponatremia after admission.

In study done by Goldberg 83 et al, a total of 105 deaths (10%) occurred within 30 days of admission. 6.2% (44/708) of patients without hyponatremia, 19.8% (26/131) of patients with hyponatremia on admission and 16.8% (35/208) of patients who developed hyponatremia after admission. Their study of 1858 ST-elevation MI patients concluded that hyponatremia independently correlated with in-hospital mortality.

Hyponatremics had higher rates of in-hospital mortality (13.5% vs. 3.8%, p<0.001) composite of death and heart failure (27.8% vs 18.4% p=0.022). Ahmad Sajadieh et al in their study of 671 men and women aged 55 to 75 years with no history of cardiovascular disease, stroke, or cancer, found an adverse outcome defined as death or Myocardial Infarction (MI) occurred in 43% of patients whose serum sodium level was less than 134 mEq/L. They concluded that Hyponatremia is an independent predictor of death and myocardial infarction in middle-aged and elderly patients Bae et al reported that in hospitalized survivors of acute myocardial infarction, the presence of hyponatremia at discharge was an independent predictor of 12-month mortality. The study involved 1290 patients.

In comparison with the above study, our study had higher mortality in patients with hyponatremia on admission whereas mortality was almost equal in patients who developed hyponatremia after admission.

The group with sodium level <130 mmol/L had 58% mortality and those with serum sodium in the range of 131-134 mmol/L suffered 17% deaths. This was in concordance with the study conducted by Goldberg et al., who showed increasing mortality with severity of hyponatremia. Hilton et al and Alexander et al, who showed increasing morality with severity of hyponatremia. Hilton, P. showed in their study that mortality of patients with MI is related to plasma sodium level. It had been showed that acute STEMI patients without hyponatremia had a mortality rate 6.2% and the mortality rate was 19.8% in patients with hyponatraemia on admission and 16.8% in patients with hyponatremia developed after admission.

When we compared the various risk factors and outcomes among the survivors and the non survivors, we found, apart from age, sex, diabetes, hypertension, Killip class on admission, ejection fraction, hyponatremia was significant risk factor in determining mortality. All the variables among the survivors and non survivors that were significantly associated with mortality were included in the multivariate logistic regression analysis. Hyponatremia remained a significant independent predictor of mortality. This is in concordance to similar study conducted by

Goldberg et al., they found that hyponatremia was independently associated with 30-day mortality. In a similar study of 235 patients admitted to a coronary care unit, Flear et al., found higher in hospital mortality rates among patients with minimal plasma sodium levels ≤130 mmol/L.

It was observed that the development of hyponatremia is a biochemical marker for prognostic importance i.e. left ventricular dysfunction severity, hemodynamical changes and neurohumoral activation.

Hence in our study, we concluded that hyponatremia on admission or early development of hyponatremia in patients with acute ST elevation MI with or without reduced ejection fraction is an independent predictor of 30-day mortality.

CONCLUSION

In our study substantial proportion of patients who presented with acute ST elevation myocardial infarction with reduced ejection fraction were hyponatraemic on admission or developed hyponatremia shortly after admission. Univariate analysis of the several variables among survivors and non survivors identified hyponatremia as significant risk factor in determining mortality.

Summary

In STEMI, arterial under filling leads to immediate stimulation of baroreceptors in the carotid sinus, LV, and aortic arch, which in turn leads to activation of efferent sympathetic nervous regulatory system. The sympathetic activation causes release of non-osmotic AVP, renin and angiotensin II, which leads to dilutional hyponatraemia.

Thus, hyponatraemia signifies underlying neurohormonal activation.

At cellular level, hyponatraemia is caused due to:

1) Cellular permeability caused by ischaemia which is aggravated by stress hormones.
2) Arginine-vasopressin causing water retention.

Hence, we aimed to investigate the prognostic importance of hyponatremia in the setting of acute ST elevation MI with reduced ejection fraction and to determine its usefulness in predicting short term survival.

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