

Hyponatremia–A Predictor of Short Term Mortality in Acute ST Segment Elevation Myocardial Infarction (STEMI)

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ABSTRACT

To study the prevalence of hyponatremia in a case of acute ST segment elevation myocardial infarction. To study the relationship between severity of hyponatremia and short term mortality. To determine the prognostic importance of hyponatremia in a case of acute ST segment elevation myocardial infarction. 50 patients admitted in the Intensive care unit of Sree Balaji Medical College and Hospital between November 2017 to July 2019, with acute ST segment elevation myocardial infarction (STEMI) were included in this prospective study. Along with other risk factors, hyponatremia on admission or early development of hyponatremia appeared to be a significant independent risk factor in predicting short term mortality in acute myocardial infarction.

Key words: Hyponatremia, Cardiac failure, Cirrhosis, Diabetes

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INTRODUCTION

The leading cause of death worldwide is coronary artery disease [1]. In 2015 coronary artery disease accounted for 72 million deaths worldwide [2,3]. 80% of which were in low income countries like India [4]. It has been estimated that by 2020, 2.6 million Indians are predicted to die because to coronary artery disease [5]. Indians are prone to get coronary artery disease at an earlier age compared to people in developed countries because of the high prevalence of risk factors like diabetes and hypertension [6,7]. ST segment elevation myocardial infarction is most common type of acute coronary event contributing 60.6% of overall incidence of acute coronary syndrome in Indian population [8]. The overall mortality in STEMI is approximately 4 to 7 % in the published clinical trials [8].

However this is not the case in the real world situation [9,10]. This is because the patients enrolled in the randomized trials are selected ones and represented low-risk subgroup. Therefore the results of these trials are not applicable to 50% of patients in clinical practice [11]. A realistic view can be obtained from registry data.

In India, CREATE registry data recorded an in-hospital mortality rate of 7.9% and 30 day mortality rate of about 8.6%, which included both patients with unstable angina and AMI. Jacob et al. from Vellore (Tamilnadu), observed 16.9% in hospital mortality amongst the South Indian

population following STEMI [12]. Hyponatremia is commonly seen amongst hospitalized patients [13-16] especially with comorbid conditions like cardiac failure, cirrhosis or nephrotic syndrome. Hyponatremia plays a major role in prediction of cardiovascular mortality amongst patients with cardiac failure [17-19]. The neurohormonal activation accompanying an acute myocardial infarction is similar to the one which accompanies a cardiac failure [20].

Hyponatremia is common after Myocardial infarction [21], and a rise in plasma sodium concentration accompanies clinical improvement in patients [22]. The prognostic importance of hyponatremia in a case of chronic heart failure is very well established whereas its importance acute myocardial infarction is lacking [23-30]. This study was conducted to determine the prognostic importance and usefulness of hyponatremia for predicting short term survival in a case of acute ST segment elevation MI.

MATERIALS AND METHODS

50 patients admitted in the Intensive care unit of Sree Balaji Medical College and Hospital between November 2017 to July 2019, with acute ST segment elevation myocardial infarction (STEMI) was included in this prospective study.

Study design

Single centre and prospective.

Acute STEMI was diagnosed according to the following criteria:

Diagnosis of STEMI

- Presence of chest pain of >20min duration.
- ST segment elevation of >1mm in atleast two standard limb leads or >2mm in atleast two contiguous precordial leads or new onset of Left bundle Branch block.
- Elevated cardiac biomarkers.

Two out of Three criteria qualified for diagnosis of MI.

Inclusion criteria

Patients who presented within 12 hrs. of onset of symptoms STEMI included in the study.

Exclusion criteria

- Patients with Non STEMI or Unstable angina.
- Patients with previous history of coronary artery disease.
- Patients with previous history of arrhythmias.
- Patients with previous history of cardiomyopathy or heart failure.
- Patients with previous diuretic use.
- Patients with cirrhosis of liver, renal disease, hypothyroidism.
- Patients with Serum Creatinine >2mg%, Blood urea >60mg/dl.

Patients who fulfilled the above inclusion criteria and not having any of the above said exclusion criteria were included in the study as a participant.

Methods

12 lead ECG was taken for all patients, Leads V3R, V4R

was taken in patients with inferior wall MI. Location of the infarct were defined as follow Anteroseptal MI: ST-segment elevation in V1-V4 Apical MI: ST-segment elevation in V5-V6 Anterolateral MI: ST-segment elevation in LI, aVL, V4-V6 Extensive Anterior wall MI: ST-segment elevation in LI, aVL, V1-V6 Inferior wall MI: ST-segment elevation in LII, LIII, aVF Right ventricular MI: ST-segment elevation in V3R, V4R Posterior wall MI: Tall and wide R wave, depressed and concave upwards ST, upright and widened T wave in V2.

Statistical method

The collected data were analysed with IBM.SPSS statistics software 23.0 Version. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups the Unpaired sample t -test was used. To find the significance in categorical data Chi-Square test was used similarly if the expected cell frequency is less than 5 in 2x2 tables then the Fisher's Exact was used. In all the above statistical tools the probability value .05 is considered as significant level.

RESULTS

Our Study population was 50 which included 40 male patients (80% of the study population were male) and 10 were female (20% of the study population were female) (Table 1). Our study population was 50 patients demographic distribution shows 18 patients have diabetes mellitus (36% of study population) and 32 patients were non-diabetic (64% of the study population) (Figure 1).

Table 1: Distribution of patients according to gender.

Gender	Frequency	Percent
Female	10	20
Male	40	80
Total	50	100

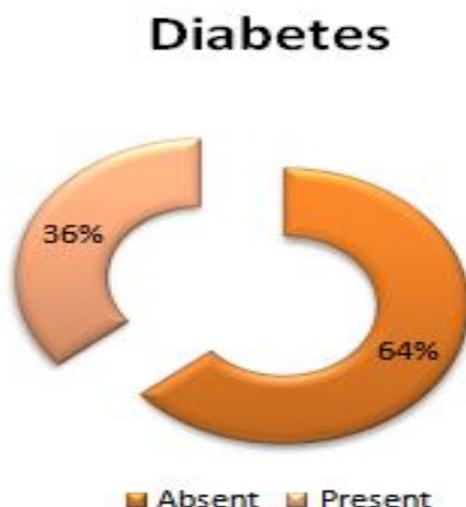


Figure 1: Showing distribution of diabetes mellitus in the study.

Our study population was 50, demographic distribution showed 21 patients as hypertensive (42% of the study population) and 29 patients were non-hypertensive (which includes 58% of the study population) (Table 2).

Our study population was 50 demographic distributions shows 20 patients as smokers (which includes 40% of the study population) and 30 were non-smokers (which includes 60% of the study population) (Figure 2).

Table 2: Showing distribution of hypertension amongst patients.

HTN			
		Frequency	Percent
Valid	Absent	29	58
	Present	21	42
	Total	50	100

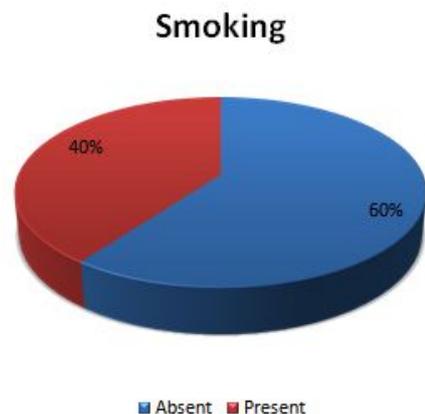


Figure 2: Showing distribution of smoking in the study.

Our study population was 50 patients, 39 patients were Killip class I (which includes 78% of the study population) 9 patients were Killip class II (18% of the study population) and 2 patients were Killip class III (which includes 4% of the study population) (Table 3). Demographic distribution showing AWMI 58% most commonly involved followed by IWMI 16%, IPWMI 10%, ASMI 8%, ALMI 4%, PRVMI 2% and IRWMI 2% (Figure 3).

Table 3: Showing distribution.

Killip classification		
	Frequency	Percent
I	39	78
II	9	18
III	2	4
Total	50	100

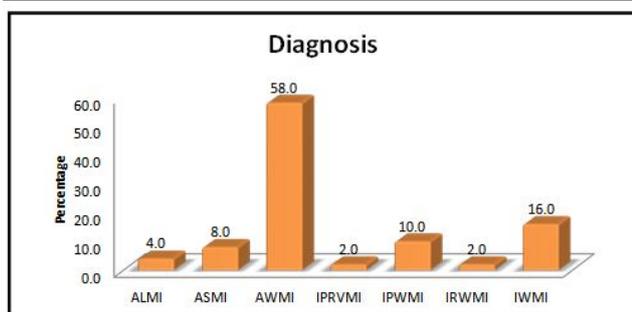


Figure 3: Showing distribution of site of myocardial infarction.

DISCUSSION

In acute myocardial infarction the development of hyponatremia is a marker that probably incorporates different prognostic entities, including severe left ventricular dysfunction, hemodynamic alterations, and the extent of neurohormonal activation. Goldberg A et al [31] studied 1047 patients with acute ST elevation MI, without past history of heart failure. It was found that hyponatremia on admission or early development of hyponatremia was independently associated with short term mortality [26]. Our study comprised of 50 patients with acute ST elevation MI. The mean age was 52.7 ± 12.5. Majority of the cases were in the age group of 61 -70. In the study conducted by Aziz M et al, the mean age

was 57.28±6. In Goldberg’s study the mean age was 61±12. When compared to the other studies it is seen that Indians are prone to get MI at an earlier age [31]

In our study, 80% were males and 20% were females. Similar results were seen in a studies conducted by Goldberg et al. [31]. Thus males are more prone to develop MI. In our study 20 patients were smokers, 21 were hypertensive and 18 were diabetic. Which accounts for 40% were smokers, 42% were hypertensive, 36% were diabetic [27]. Framingham heart study said that diabetes and smoking increases the risk of death after myocardial infarction. In GISS-2 trial 69, out of 11483 hypertensive MI patients, 3306 patients expired. Our study reveals that Diabetes, Hypertension and smoking are important risk factors in determining mortality. In our study, hyponatremia was present on admission in 14 patients .In a study conducted by Goldberg et al, [31] hyponatremia was present in 131 patients (12.5%) and hyponatremia developed in 208(19.9%) during the first 72 hours of hospitalisation. Similar results were seen in the studies conducted by Flear CT et al [22] and Schrier et al. [28] our results were also consistent with other studies.

Patients who presented with hyponatremia on admission belonged to a higher age (54 ± 14) when compared to patients with normal sodium levels who belonged to younger age (50 ±12). In the study conducted by Goldberg et al. [31] and Hammerman et al. [32] mean age

among patients with normal sodium levels was 61 ± 13 and hyponatremic individuals was 63 ± 13 . Males constituted majority of the cases [29,30]. There were 30 male (83.3%) with normal sodium levels, 10 male (71.4%) with hyponatremia on admission. There were 6 female with normal sodium levels, 4 with hyponatremia on admission. The higher male ratio was due to the fact that number of females were less in our study. Similar results were found in Goldberg A, Hammerman H et al, Aziz M et al "s study [31].

Among patients with normal sodium levels 27.8% were diabetic, 38.9% were smokers and 38.9% were hypertensive. In patients who presented with hyponatremia on admission 57.1% were diabetic, 42.9% were smokers, 50% were hypertensive. Thus hyponatremia was more common among smokers, diabetic and hypertensive individuals. This is in accordance to the studies conducted by Goldberg et al. [31] and Hammerman H et al [32].

CONCLUSION

Asians are prone to develop STEMI at a younger age than western population. In Indians hypertension, smoking, dyslipidemia, diabetes are predominant risk factors for STEMI. Hyponatremia on admission or early development of hyponatremia within 72 hours was associated with a poor prognostic outcome which had a mortality rate of 63.61%. Hyponatremia on admission with low ejection fraction showed a poor prognostic outcome.

Patients with hyponatremia were males belonging to a higher age group, with lower ejection fraction; anterior wall infarction and a higher proportion of them were smokers, hypertensive, diabetic and had dyslipidemia. Along with other risk factors, hyponatremia on admission or early development of hyponatremia appeared to be a significant independent risk factor in predicting short term mortality in acute myocardial infarction.

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ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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REFERENCES

1. Lopez AD, Mathers CD, Ezatti M, et al. Global and regional burden of disease and risk factors 2001: Systematic analysis of population health data. *Lancet* 2006; 367:1747-57.
2. Castelli WP. Epidemiology of coronary heart disease: The framingham study. *Am J Med* 1984; 27:4-12.
3. Rogers WJ, Canto JG, Lambrew CT, et al. Temporal trends in the treatment of over 1.5 million patients with myocardial infarction in the US from 1990 through 1999: The national registry of myocardial infarction 1, 2 and 3. *J Am College Cardiol* 2000; 36:2056-63.
4. Reddy KS, Cardiovascular disease in non-western countries. *N Engl J Med* 2004; 350:2438-40.
5. Ghaffer A, Reddy KS, Singhi M. Burden of non-communicable diseases in South Asia. *BMJ* 2004; 328:807-10.
6. Mohan V, Deepa R, Rani SS, et al. prevalence coronary artery disease and its relationship to lipids in selected population in South India. The Chennai urban population study (CUPS No 5). *J Am Coll Cardiol* 2001; 38:682-87.
7. Joshi P, Islam S, Pais P, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. *JAMA* 2007; 297:286-9.
8. Pais P, Xavier D, Gupta R, et al. Treatment and outcome of acute coronary syndrome in India the (CREATE): A prospective analysis of registry data. *Lancet* 2008; 371:1435-42.
9. Brown N, Young T, Gray D, et al. Inpatient deaths from acute myocardial infarction 1982-1992: Analysis of data in Nottingham heart attack register. *BMJ* 1997; 315:159-164.
10. Every NR, Freiderick PD, Robinson M, et al. A comparison of the national registry of myocardial infarction -2 with the co-operative cardiovascular project. *J Am Coll Cardiol* 1999; 33:1886-94.
11. Zeymer U, Senges J. Why do we need prospective registries in patients with myocardial infarction. *Eu Heart J* 2003; 24:1611-12.
12. Jose VJ, Gupta SN. Mortality and morbidity of acute ST segment elevation myocardial infarction in the current era. *Indian Heart J* 2004; 56:210-4.
13. Upadhyay A, Jaber BL, Madias NE. Incidence and prevalence of hyponatremia. *Am J Med* 2006; 119:S30-S35.
14. Clayton JA, Le Jeune IR, Hall IP. Severe hyponatraemia in medical inpatients: aetiology, assessment and outcome. *QJM* 2006; 99:505-511.
15. Zilberberg MD, Exuzides A, Spalding J, et al. Epidemiology, clinical and economic outcomes of admission hyponatremia among hospitalized patients. *Curr Med Res Opin* 2008; 24:1601-1608.
16. Chung HM, Kluge R, Schrier RW, et al. Postoperative hyponatremia. A prospective study. *Arch Intern Med* 1986; 146:333-336.

17. Schrier RW, Abraham WT. Hormones and hemodynamics in heart failure. *N Engl J Med* 1999; 341:577-585.
18. Lee WH, Packer M. Prognostic importance of serum sodium concentration and its modification by converting-enzyme inhibition in patients with severe chronic Hyponatremia. *Circulation* 1986; 73:257-267.
19. Saxon LA, Stevenson WG, Middlekauff HR, et al. Predicting death from progressive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. *Am J Cardiol* 1993; 72:62-65.
20. Sigurdsson A, Held P, Swedberg K. Short- and long-term neurohormonal activation following acute myocardial infarction. *Am Heart J* 1993; 126:1068-1076.
21. Bogdan M, Nartowicz E. Magnesium, potassium and sodium in acute MI. *Kardiologia Pol* 1993; 38:263-266.
22. Fleck CT, Hilton P. Hyponatremia and severity and outcome of myocardial infarction. *BMJ* 1979; 1:1242-1246.
23. Oren RM. Hyponatremia in congestive heart failure. *Am J Cardiol* 2005; 95:2B-7B.
24. Lee DS, Austin PC, Rouleau JL, et al. Predicting mortality among patients hospitalized for heart failure: Derivation and validation of a clinical model. *JAMA* 2003; 290:2581-2587.
25. Klein L, O'Connor CM, Leimberger JD, et al. Lower serum sodium is associated with increased short-term mortality in hospitalized patients with worsening heart failure: Results from the outcomes of a prospective trial of intravenous milrinone for exacerbations of chronic heart failure (OPTIME-CHF) Study. *Circulation* 2005; 111:2454-2460.
26. Gaziano JM. Global burden of cardiovascular disease. In: Zipes, Libby, Bonow, Braunwald. *Braunwald's heart disease, a text book of cardiovascular medicine*. 8th Edn. Philadelphia: Elsevier Saunders; 2008.
27. Schaller MD, Nussberger J, Feihl F. Clinical and hemodynamic correlates of elevated plasma arginine vasopressin after acute myocardial infarction. *Am J Cardiol* 1987; 60:1178-1180.
28. Schrier RW, Berl T, Anderson RJ. Osmotic and nonosmotic control of vasopressin release. *Am J Physiol* 1979; 236:321-332.
29. Kannel WB, McGee DL. Diabetes and cardiovascular risk factors: The Framingham study. *Circulation* 1979; 59:8-13.
30. Fresco C, Avanzini F, Bosi S, et al. Prognostic value of a history of hypertension in 11483 patients with acute myocardial infarction treated with thrombolysis. *J Hypertension* 1996; 14:743-75.
31. Goldberg A, Hammerman H, Petcherski S, et al. Hyponatremia and long term mortality in survivors of acute ST elevation myocardial infarction. *Arch Intern Med* 2006; 166:781-786.
32. Hammerman H, Hillis LD, Forman S, et al. Risk stratification before thrombolytic therapy in patients with acute myocardial infarction. The thrombolysis in myocardial infarction (TIMI) Phase I Co-Investigators. *J Am Coll Cardiol* 1990; 16:313-315.