Hyponatremia and in Hospital Mortality in Acute St Elevation Myocardial Infarction

Authors
Dr M. Ajeeth Fera*1, Dr S. Balasubramaniam2, Dr N. Paari3
*1Post Graduate, Department of Medicine, RMMCH, Chidambaram-608002, Tamilnadu
2Professor & Head, Department of Medicine, RMMCH, Chidambaram-608002, Tamilnadu
3Lecturer, Department of Medicine, RMMCH, Chidambaram-608002, Tamilnadu

Abstract
The present study is done to determine the prevalence and prognostic implications of hyponatremia in the setting of acute ST-elevation myocardial infarction. The study sample consists of 50 patients presenting with acute ST-elevation myocardial infarction. Plasma sodium concentrations were obtained on admission and at 24, 48, and 72 hours thereafter. Patients with ckd stage 3, 4 and 5, CCF patients on diuretics, previous history of head injury and stroke, known hypothyroidism patients and nephrotic syndrome are excluded in this study. Hyponatremia, defined as a plasma sodium level <135 mmol/L (<135 mEq/L), is present in 20 patients within first 72 hours of hospitalization where 25% patients died. In rest of 30 patients, only 3.3% of patients died. Hyponatremia on admission or early development of hyponatremia in patients with acute ST-elevation myocardial infarction is an independent predictor of mortality and prognosis.

Keywords: hyponatremia, myocardial infarction.

Introduction
Among the causes of death worldwide, cardiovascular diseases ranks first.1 Atherosclerotic disease accounts for the major burden of ischemic heart disease. The incidence is on rise in developing nations worldwide. In rural India prevalence has increased from 2% to 4%.2 While comparing with developed countries, in countries like India the disease tends to be more common in the younger population. (mean age-57 years). Neurohormonal activation and cardiac remodelling are being implicated as the common cause for the mortality and morbidity following myocardial infarction. Electrolyte imbalances have a significant role in the neurohormonal activation among which sodium plays a major role.3-10 Hence this study was undertaken to know the impact of hyponatremia on short term mortality following myocardial infarction.

Aims and Objectives
- To determine the prevalence of hyponatremia in acute ST elevation myocardial infarction.
- To determine the significance of hyponatremia on in-hospital mortality in acute ST elevation myocardial infarction.

Materials and Methods
- Prospective observational study. Sample size consists of a total of 50 consecutive cases admitted with acute ST elevation myocardial infarction. The study was conducted among
the patients admitted with Acute ST elevation Myocardial Infarction in the CCU of RMMCH, Chidambaram.

- In this study acute myocardial infarction was diagnosed by
  - Typical anginal pain lasting more than 20 minutes
  - Elevated cardiac biomarkers like Troponin T and creatine kinase –MB.
  - Diagnostic ECG changes consisting of
    - ST elevation ≥ 1mm in two contiguous limb leads.
    - ST elevation ≥ 2mm in two contiguous precordial leads.
- Hyponatremia was diagnosed as serum sodium levels less than 135 mEq/L.

**Study Population**
A total number of 50 patients were included in the study. Patients presenting with acute ST elevation myocardial infarction were included in the study.

**Incidence of hyponatremia**

| Serum sodium levels | Number of patients | Percentage |
|---------------------|--------------------|------------|
| <135mEq/L           | 20                 | 40%        |
| 136-145 mEq/L       | 30                 | 60%        |

**In Hospital Mortality**

|                  | Hyponatremia | Normal sodium levels |
|------------------|--------------|----------------------|
| Number of subjects| 20           | 30                   |
| Mortality        | 25%(n=5)     | 3.3%(n=1)            |
Majority of study subjects had normal sodium levels 60% (n=30) and study subjects with sodium values < 135 were 40% (n=20).

Among patients with hyponatremia mortality was 25% (n=5) while among normonatremia the mortality was 3.3% (n=1).

**Discussion**

In our study the prevalence of hyponatremia was 40%. The overall mortality in our study was 12%. Among patients with hyponatremia mortality was 25% while among those with normal sodium levels it was 3.3%. There are various hypothesis for the cause of hyponatremia following myocardial infarction. In acute myocardial infarction, secretion of vasopressin may be due ventricular dysfunction: as a response to stress, in response to treatment with diuretics. Vasopressin levels didn't correlate with serum osmolarity suggesting that non osmotic mechanisms are involved. The levels of vasopressin increase along with the activation of renin and nor epinephrine. Also hyponatremia was associated with a mean decrease in ejection fraction while compared with patients with normal sodium levels. This shows that there is a significant impact of hyponatremia on mortality in acute myocardial infarction.

**Conclusion**

Hyponatremia on admission or early improvement of hyponatremia in patients with acute ST-elevation myocardial infarction to be considered as a predictor of in hospital mortality. Even though sodium levels are usually taken in instances of STEMI, we "look at the results, but we don't 'see' them." Hyponatremia during the initial phase myocardial infarction must be used as a red flag sign and make sure they are treated with ACE inhibitors and beta blockers. Further research is necessary to decide if plasma sodium levels may serve as a simple marker to identify patients at high risk of morbidity and mortality.

**References**

1. Elliott M. Antman, Joseph Loscalzo. ST-Segment Elevation Myocardial Infarction. In Kasper, Fauci, Hauser, Longo, loscalzo and Jameson editors. Harrison’s Principles of Internal Medicine vol-2.19th edn. Newyork: McGraw Hill; 2015.p. 1602.
2. Gazino JM. Global burden of cardiovascular disease. In: Zipes, Libby, Bonow, Braunwald editors. Brauwnalds Heart disease, a text book of cardiovascular medicine. 8th edn. Philadelphia: Elsevier Saunders; 2008. Part 1 p.1-13
3. Mcalpine HM, Morton JJ, Leckie B, Rumley A, Gillen G, Dargie HJ. Neuroendocrine activation after acute myocardial infarction. Br Heart J 1988;60:117-124
4. Rowe JW, Shelton RL, Hellderman JH. Influence of the emetic reflex on
vasopressin release in man. Kidney Int. 1979; 16:729-735.

5. Schaer GL, Covit AB, Laragh JH, Cody RJ. Association of hyponatremia with increased renin activity in chronic congestive heart failure: impact of diuretic therapy. Am J Cardiol. 1983; 51:1635-1638.

6. 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.

7. Schrier RW, Berl T, Anderson RJ. Osmotic and nonosmotic control of vasopressin release. Am J Physiol 1979; 236:321-332.

8. Kumar S, Berl T. Sodium. Lancet 1998; 352:220-228.

9. Cohn JN, Levine TB, Olivari MT. plasma nor epinephrine as a guide to prognosis in patients with chronic congestive heart failure. N Engl J Med 1984; 311:819-823.

10. Dzau VJ, Colucci WS, Hollenberg NK, Williams GH. Relation of rennin angiotension aldosterone system to clinical state in congestive heart failure. Circulation 1981; 63:645-651.

11. 2006 Apr 10; 166(7):781-6. Hyponatremia and long-term mortality in survivors of acute ST-elevation myocardial infarction. Goldberg A1, Hammerman H, Petcherski S, Nassar M, Zdorovyak A, Yalonetsky S, Kapeliovich M, Agmon Y, Beyar R, Markiewicz W, Aronson D.Department of Cardiology, Rambam Medical Center and Rappaport Medical School, Haifa, Israel.