HYPONATRAEMIA AS A PROGNOSTIC INDICATOR IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION

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ABSTRACT

OBJECTIVES

To assess the incidence of hyponatraemia in patients with acute ST elevation myocardial infarction in Intensive Coronary Care Unit and to find out whether hyponatraemia serves as a bad prognostic indicator in acute ST elevation myocardial infarction patients.

METHODS

60 patients diagnosed with Acute ST elevation Myocardial Infarction within 48 hrs. of onset of symptoms were included. Serum sodium was recorded on the day of admission. Echo with LVEF at discharge was also recorded. Outcome of the patients was recorded as either improved (with mild, moderate or severe LV dysfunction) or expired.

RESULTS

The average serum sodium of the 60 patients was 131.3 mEq/L with values ranging from 122–141 mEq/L. About 78.3% of patients with STEMI had hyponatraemia and patients with Anterior Wall MI had greater incidence of hyponatraemia than Inferior Wall MI. Mortality among study group was 30%. There was significant relationship between the respective serum sodium level and outcome of our patients ($p = 0.0434$), where hyponatraemia was found significantly among those who expired due to MI. Significant correlation existed between hyponatraemia and LV dysfunction which implied that the lower the serum sodium more severe was the LV dysfunction.

CONCLUSIONS

Hyponatraemia is frequently found in ST elevation myocardial infarction, particularly involving the anterior wall than inferior wall. It is strongly associated with higher Killip class of patients, more severe LV dysfunction and even more number of deaths. Hence, hyponatraemia serves as a poor prognostic indicator in Acute ST elevation myocardial infarction in short-term outlook.

KEYWORDS

Hyponatraemia, Myocardial Infarction, Prognosis.

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INTRODUCTION: Hyponatraemia is found more frequently in the early period of ST elevation myocardial infarction, and influences short as well as long term outcomes[1]. In STEMI, like congestive heart failure, arterial underfilling causes stimulation of high pressure baroreceptors present in the left ventricle, arch of aorta and carotid sinus, causing stimulation of cardioregulatory 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[2,3] & also worsens cardiac remodelling (AVP plays role in regulation of vascular tone and cardiac contractility and negatively influences cardiac haemodynamics and myocardial remodelling).[1,4]

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[5,6].

Hence, it is worth to evaluate the incidence of hyponatraemia in patients with acute ST elevation myocardial infarction in Intensive Coronary Care Unit & to

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find out whether hyponatraemia serves as a poor prognostic indicator in these patients.[7,8,9,10]

MATERIAL AND METHODS: This study was done among 60 admitted patients (males and females aged above 12 yrs.) diagnosed with Acute ST elevation Myocardial Infarction (Thrombolysed as well as not thrombolysed patients) at Intensive Coronary Care Unit, Government Rajaji Hospital, Madurai within 48 hrs. of onset of symptoms. Serum sodium on the day of admission was recorded.

Patients with coexisting head trauma, postoperative patients, self-poisoning patients, snake bites patients, patients who had PCI (either Primary PCI, Rescue PCI or Facilitated PCI) during the course of stay in hospital, patients with Non-STEMI and Unstable angina, patients with systemic malignancy, patients with hypernatremia (Serum sodium > 145 mg%), patients who developed dysnatraemia during the hospital stay, patients with endocrine disorders and patients who are on drugs that can cause hyponatraemia were excluded from the study.

With reference from standard literatures, patients were grouped according to their serum sodium concentration as follows:

Normonatraemia		- 135 to 145 mEq/L
Hyponatraemia	Mild	- 130 to 134 mEq/L
	Moderate	- 115 to 129 mEq/L
	Severe[4]	< 115 mEq/L

Serum proteins and serum cholesterol were done in all patients and patients with hyperlipidaemia and hyperproteinaemia were excluded to avoid Pseudohyponatraemia. Also patients with Random Blood Sugar >200 mg% were excluded to avoid correction to sodium levels. The patients were also divided according to their volume status.

Every patient was followed up till their discharge. Those patients who recovered from the acute STEMI were included in the survived group, while the patients who expired in their stay were added in the mortality group. Patients belonging to the survival people group were further grouped according to LVEF into Mild (EF = 41-50%), Moderate (EF = 31-40%) & Severe (EF ≤ 30%) LV dysfunction patients.

STATISTICAL ANALYSIS: All the details obtained from the patients were noted and results were analysed statistically. Student's 't' test was used to test the association between quantitative variables and its significance. For qualitative variables, chi square test was used. The 'p' value which was lesser than 0.05 denoted significant one to one relationship.

RESULTS: The mean age of the patients in our study was 49.4 years with age group between 30-77 yrs. Majority of male patients were smokers signifying the importance of smoking as a risk factor in STEMI. Average Serum sodium was 131.3 mEq/L with values ranging from 122–141 mEq/L. About 78.3% of patients with STEMI had hyponatraemia and majority of myocardial infarction patients (65%) had involvement of anterior wall. Patients with Anterior Wall MI had greater incidence of hyponatraemia than Inferior Wall MI. Mortality among study group was 30%. Significant correlation was found between the respective serum sodium and Killip classification (p = 0.0126), where hyponatraemia was associated with (inversely proportional to) higher Killip class of patients. There was significant relationship between the respective serum sodium level and outcome of our patients (p = 0.0434), where hyponatraemia was found significantly among those who expired due to MI. Significant correlation existed between hyponatraemia and LV dysfunction which implied that the lower the serum sodium more severe was the LV dysfunction.

Killip Class	Hyponatraemia			
	Yes		No	
	No.	%	No.	%
1 (38)	26	68.4	12	31.6
2 (22)	21	95.5	1	4.5
'p'	0.0126 Significant			

Table 1: Killip Class and Hyponatraemia

Hyponatraemia	Diagnosis			
	AWMI		IWMI	
	No.	%	No.	%
Yes (47)	35	74.5	12	25.5
No (13)	4	30.8	9	69.2
'p'	0.0054 Significant			

Table 2: Hyponatraemia and Diagnosis

Hyponatraemia	Outcome			
	Survived		Expired	
	No.	%	No.	%
Yes (47)	30	63.8	17	36.2
No (13)	12	92.3	1	7.7
'p'	0.0434 Significant			

Table 3: Hyponatraemia and Outcome

LV Dysfunction	Serum Sodium (MEq/L)	
	Mean	S.D.
Mild LV Dysfunction	133.9	4.2
Moderate LV Dysfunction	131.2	4.2
Severe LV Dysfunction	127.0	1.7
Expired cases	129.1	3.3
'p'	< 0.0001 Significant	

Table 4: LV Dysfunction and Serum Sodium

DISCUSSION: Hyponatraemia is the most common electrolyte disorder in hospitalised patients and it generally predicts the prognosis[5]. In longstanding congestive heart failure (CHF), hyponatraemia causes activation of baroreceptor hormones like AVP, catecholamines and the

well-known renin angiotensin aldosterone system. Dilutional hyponatraemia caused by osmolality independent secretion of AVP[11].

Severity of heart failure and worsening of remodelling is attributed to the baroreceptor-mediated hormones[2]. In STEMI, role of baroreceptor-mediated hormones and their activation is similar to that of CHF and has a role in prognosis. This hyponatraemia, found in the early STEMI, serves as an important prognostic factor according to several studies[12].

Same thing holds for the neurohormonal activation in STEMI and is same to that occurring in heart failure[13]. Hyponatraemia caused by neurohormonal mechanisms are seen during the initial 24 hours after STEMI. They come to normal during first few days if there is no heart failure. If there is persistent neurohormonal activation, it means there is clinical heart failure[5,6].

In STEMI, arterial underfilling 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[1,11]. The sympathetic activation causes release of non-osmotic AVP, renin and angiotensin II, which leads to dilutional hyponatraemia. Thus, hyponatraemia signifies underlying neurohormonal activation.

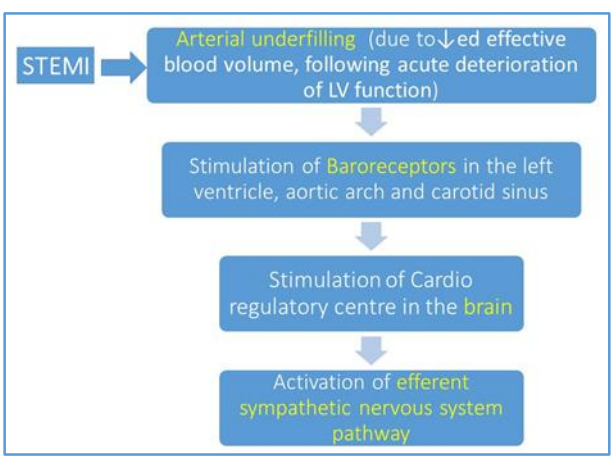

Figure 1

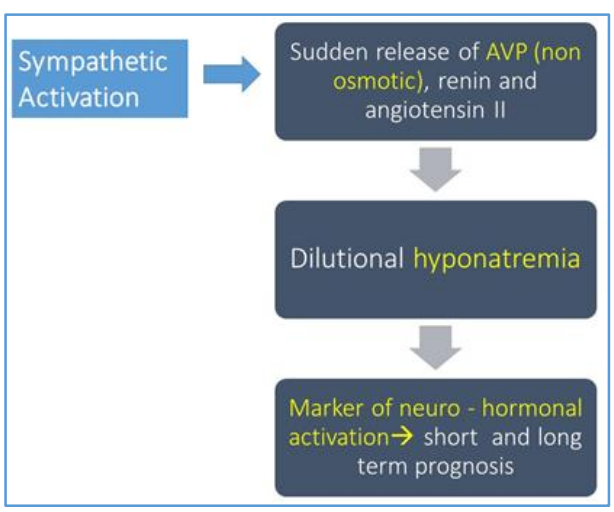

Figure 2

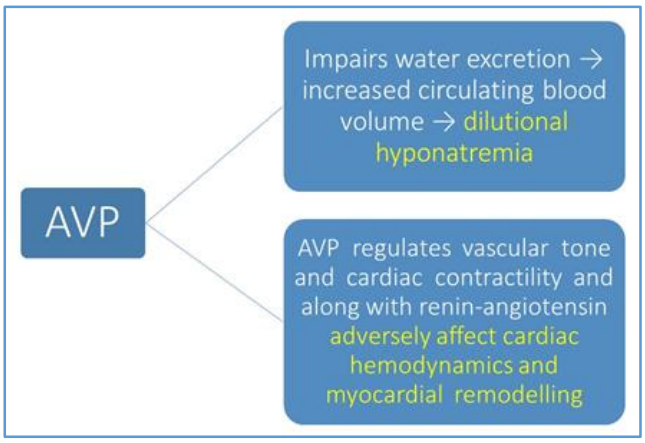

Figure 3

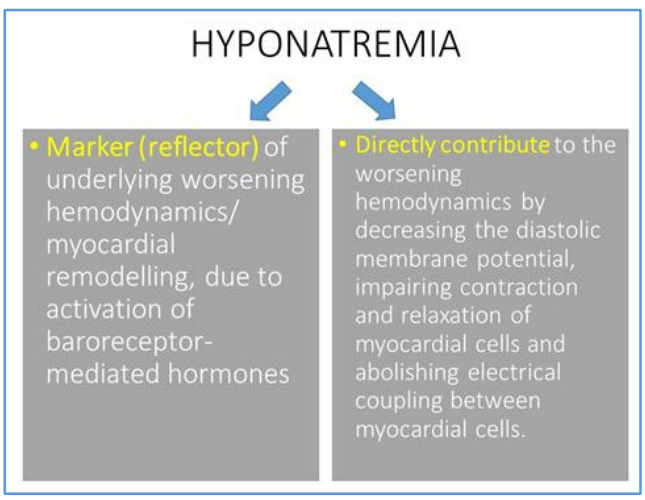

Figure 4

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.

The usefulness of AVP receptor antagonists in heart failure is clearly proved by studies. Chronic administration of Tolvaptan, V2 receptor binding antagonist relieved hyponatraemia and thereby water retention. However, controversial incidence of adverse cardiac events like sudden cardiac death or recurrent hospitalisation for heart failure has been found. In rat model, conivaptan (V1/V2 receptor binding antagonist) and tolvaptan (selective V2 receptor binding antagonist) were found to prevent heart failure. Tolvaptan is found to improve contractility because of increased dP/dt (max)/LV pressure[14,11,13].

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