

Study Of Hyponatremia In Acute St Elevation Myocardial Infarction And It's Prognostic Significance

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ABSTRACT

Hyponatremia is common after acute MI.Theneurohormonal activation that is associated with acute myocardial infarction is identical to that which accompanies heart failure.

However, Hyponatremia's prognostic value in chronic heart failure is well established, while data on hyponatremia's prognostic importance in acute myocardial infarction is lacking.

This study was done to study and determine the prognostic importance of Hyponatremia in the setting of acute ST-elevation MI and to determine its usefulness in predicting short term survival.In acute myocardial infarction, acute development of left ventricular dysfunction causes a nonosmotic release of vasopressin .vasopressin levels increase concomitantly with activation of other neurohormones such as renin and norepinephrine and catecholamine production .These factors reduce the glomerular filtration rate and the subsequent delivery of tubular fluid to the nephrons diluting segment, contributing to decreased renal water excretion

AIMS AND OBJECTIVES

To find out the prognostic importance of Hyponatremia in acute ST- elevation myocardial infarction

INCLUSION CRITERIA

All acute myocardial infarction patients having a) Symptoms of myocardial ischemia

b) Diagnostic ECG changes with characteristic ECG patterns consisting of new

pathological Q waves or ST-segment and T wave changes.

c) Elevated CK – MB levels or elevated cardiac troponin T levels.

Characteristi cs	Normal sodium levels N=71	Hyponat remia on admissio n	Hyponat remia within 72 hrs	
A = -	57.0.1	N=11	56 (1) 1	
Age	57.8±1 1.17	64.9±13. 1	56.61±1 1.54	
Male	57(71)	9(81)	14(77)	

EXCLUSION CRITERIA Acute coronary syndrome without ST – elevation.

Patients using diuretics.

100 consecutive patients presenting with acute STelevation myocardial infarction admitted to GOVERNMENT GENERAL HOSPITAL, KAKINADA, ANDHRA PRADESH, from January 2019 to January 2020.

PATIENT DATA COLLECTION

Qualified patients underwent a detailed history and clinical examination.

Patients with acute myocardial infarction received thrombolytic therapy (tissue- type plasminogen activator or streptokinase).

Plasma sodium concentrations were obtained on admission and at 24, 48, and 72 hours after that.

OBSERVATIONS AND RESULTS AGE DISTRIBUTION:

5 I I III D C	11010	
Age	group	Frequency
(yrs)		cases
21-30		1
31-40		7
41-50		18
51-60		35
61-70		27
71-80		8
81-90		4

SEX DISTRIBUTION

Patients	Cases
Male	80
Female	20
Total	100



Diabetes	9(12.6)	3(27.5)	8(44.45)	
Smoking	50(70)	9(81)	11(61)	
Hypertensio	14(5.6)	2(18.18)	4(22.22)	
n				
Anterior	45(63)	8(72)	15(83)	
infarction				
Killip class	1.06±0.	1.18±0.4	1.06±0.2	F=1.18
	23	0	4	P=0.312
EF	44.63±	40.36±6.	50.11±1	F=2.86
	11.19	14	3.26	P=0.06

In this study, Hyponatremia was present on admission in 11 patients (11%). Hyponatremia developed in 18patients(18%) during the first 72 hours of hospitalization.

Patients who presented or developed Hyponatremia more often had higher Killip class, lower ejection fraction, diabetes, and anterior infarction.

In this study, a total of 8 deaths (8%) occurred within 30 days of admission. 2.8% (2/71) of patients without Hyponatremia, 27.5% (3/11) of patients with Hyponatremia on admission, and 16.67% (3/18) of patients who developed Hyponatremia after admission.

Numbe r of patient s	Norm al sodiu m levels 71	Hypon atremia on admiss ion 11	Hyponat remia within 72hrs 18	Tot al 100
Mortali ty in each group at the end of 30days	2	3	3	8

Range of sodium levels in	Number of	Mortality
hyponatremia	patients	
<130	3	3(100%)
131-134	26	3(11.11%)

When the various risk factors and outcomes among the survivors and the nonsurvivors were compared, it was found, apart from age, sex, diabetes, hypertension, Killip class on admission, ejection fraction, Hyponatremia was a significant risk factor in determining mortality.

All the variables among the survivors and nonsurvivors significantly associated with mortality

were included in the multivariate logistic regression analysis. Hyponatremia remained as a significant independent predictor of mortality.

In this study, it is concluded that Hyponatremia on admission or early development of Hyponatremia after admission in patients with acute

ST-elevation myocardial infarction is an independent predictor of 30-day mortality. Serum sodium levels may serve as a simple marker to identify patients at risk.

Variable	P value
Age	0.025
Sex	0.027
Smoking	0.198
Hypertension	0.027
Diabetes	0.027
Killip class	0.001
Hyponatremia	0.057
Ejection	0.05
fraction	

Multivariate analysis showed that Hyponatremia was the significant independent predictor of 30-day mortality along with other risk factors.

Neurohormonal Activation Following Acute Myocardial Infarction :

In acute myocardial infarction, acute development of left ventricular dysfunction causes a nonosmotic release of vasopressin; in response to pain, nausea, major stress, the most common mechanisms of Hyponatremia in adults; or in response to the administration of analgesics and diuretics.

In this setting, vasopressin(ADH) levels increase concomitantly with the activation of other neurohormones such as renin and norepinephrine.

Activation of carotid baroreceptors has been implicated in the nonosmotic release of vasopressin due to arterial underfilling

The carotid sinus baroreceptors sense a low effective circulating volume, and parasympathetic afferents transfer this signal to the



vasomotor center, which increases the rate of vasopressin secretion by the cells in the paraventricular nuclei

Also, there is increased expression of messenger RNA for vasopressin in the hypothalamus. Moreover, there is an enhanced renal effect of vasopressin in heart failure, as the vasopressin-regulated water in the collecting duct is upregulated

In myocardial Infarction, Hyponatremia may be further aggravated by the concomitant activation of the renin-angiotensin system and increased catecholamine production.

These factors reduce the glomerular filtration rate and the subsequent delivery of tubular fluid to the nephron's diluting segment, further contributing to decreased renal water excretion