

Evaluation of Serum Sodium Levels in Acute Myocardial Infarction Patients at a Tertiary Care Teaching Centre: A Clinical Study

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ABSTRACT

Background: More than 80% of acute myocardial infarction cases (AMI) are the result of coronary atherosclerosis with superimposed luminal thrombus. Alteration in the serum electrolyte balance has been to occur in AMI patients. Hence; present study was conducted to assess the role of serum sodium levels in AMI patients.

Materials & Methods: The present study was planned to investigate the serum sodium profile in patients with AMI. A total of 50 AMI patients and a total of 50 age and gender matched healthy controls were included in the present study. Capillary blood was withdrawn from the all the subjects and was sent to laboratory, where an Autoanalyzer was used for evaluation of serum sodium levels.

Results: Mean sodium levels among the AMI patients and the healthy controls were found to be 130.1 and 138.4 mEq/L respectively. Significant results were obtained while comparing the mean sodium levels among the AMI group and the control group respectively.

Conclusion: Serum sodium levels are significantly altered in AMI patients.

Key words: Acute Myocardial Infarction, Electrolyte, Sodium.

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INTRODUCTION

More than 80% of acute myocardial infarction cases (AMI) are the result of coronary atherosclerosis with superimposed luminal thrombus. Uncommon causes of myocardial infarction include coronary spasm, coronary embolism, and thrombosis in nonatherosclerotic normal vessels. Non-invasive imaging provides an alternative diagnostic approach in acute myocardial infarction, although the need for expensive equipment limits its application.¹⁻³ Echocardiography reliably identifies abnormalities of wall motion soon after coronary occlusion but fails to distinguish between acute infarction, old infarction, and unstable angina. Myocardial scintigraphy with infarct avid agents (usually technetium-99 pyrophosphate) is also of limited value because the uptake of the isotope is rarely adequate for imaging until 12-24 hours after the onset of symptoms. The role of immunoscintigraphy with antimyosin antibody fragments is being investigated. Alteration in the serum electrolyte balance has been to occur in AMI patients.⁴⁻⁶ Hence; present study was planned to assess the role of serum sodium levels in AMI patients.

MATERIALS & METHODS

The present study was planned in the Department of Medicine, SMS Medical College, Jaipur, Rajasthan, India. It included assessment of serum sodium profile in patients with AMI. Written consent from all the participants of the present study was

obtained. A total of 50 AMI patients and a total of 50 age and gender matched healthy controls were included in the present study. Detailed demographic and clinical details of all the subjects was taken.

Any subject with underlying metabolic disorder was excluded from the present study. Capillary blood was withdrawn from the all the subjects and was sent to laboratory, where an Autoanalyzer was used for evaluation of serum sodium levels. All the results were compiled and analyzed by SPSS software. Student t test was used for assessment of level of significance. P- value of less than 0.05 was taken as significant.

RESULTS

A total of 100 subjects were included in the present study. Among these 100 subjects, 50 were AMI patients, while the remaining 50 were healthy controls. Mean age of the subjects of the AMI group and the control group were 42.5 and 44.6 years respectively. 30 subjects among the AMI group and 28 subjects among the control group were males while the remaining were females respectively. Mean sodium levels among the AMI patients and the healthy controls were found to be 130.1 and 138.4 mEq/L respectively. Significant results were obtained while comparing the mean sodium levels among the AMI group and the control group respectively.

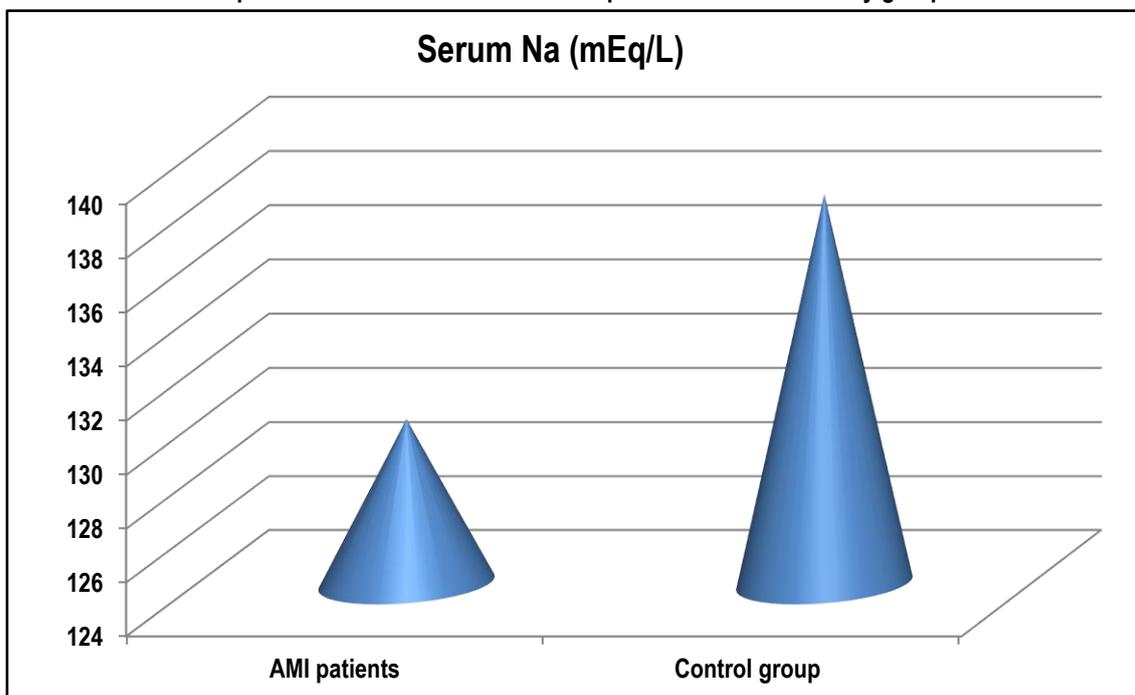
Table 1: Demographic details of the subjects

Parameter		AMI group	Control group
Age group (years)	Less than 25	5	7
	25- 35	10	8
	36- 45	15	12
	46- 55	15	18
	55 and above	5	5
Gender	Males	30	28
	Females	20	22

Table 2: Comparison of mean serum sodium levels in patients of both the study groups

Parameter	AMI patients	Control group	p- value
Serum Na (mEq/L)	130.1	138.4	0.01*

*: Significant

Graph 1: Mean serum sodium levels in patients of both the study groups**DISCUSSION**

In the present study, mean sodium levels among the AMI patients and the healthy controls were found to be 130.1 and 138.4 mEq/L respectively. Significant results were obtained while comparing the mean sodium levels among the AMI group and the control group respectively. Ouwkerk R et al determined whether the absolute tissue sodium concentration (TSC) increases in myocardial infarctions (MIs) in humans and whether TSC is related to infarct size, infarct age, ventricular dysfunction, and/or electrophysiologic inducibility of ventricular arrhythmias. Delayed contrast material-enhanced 1.5-T hydrogen 1 (1H) magnetic resonance (MR) imaging was used to measure the size and location of nonacute MIs in 20 patients (18 men, two women; mean age, 63 years \pm 9 [standard deviation]; age range, 48–82 years) examined at least 90 days after MI. End-systolic and end-diastolic volumes, ejection fraction, and left ventricle (LV) mass were measured with cine MR

imaging. The TSC in normal, infarcted, and adjacent myocardial tissue was measured on sodium 23 (^{23}Na) MR images coregistered with delayed contrast-enhanced 1H MR images. Programmed electric stimulation to induce monomorphic ventricular tachycardia (MVT) was used to assess arrhythmic potential, and myocardial TSC was compared between the inducible MVT and noninducible MVT patient groups. The mean TSC for MIs (59 $\mu\text{mol/g}$ wet weight \pm 10) was 30% higher than that for noninfarcted (remote) LV regions (45 $\mu\text{mol/g}$ wet weight \pm 5, $P < .001$) and that for healthy control subjects, and TSC did not correlate with infarct age or functional and morphologic indices. The mean TSC for tissue adjacent to the MI (50 $\mu\text{mol/g}$ wet weight \pm 6) was intermediate between that for the MI and that for remote regions. The elevated TSC measured in the MI at ^{23}Na MR imaging lacked sufficient contrast and spatial resolution for routine visualization of MI. Cardiac TSC did not enable differentiation

between patients in whom MVT was inducible and those in whom it was not. Absolute TSC is measurable with ^{23}Na MR imaging and is significantly elevated in human MI; however, TSC increase is not related to infarct age, infarct size, or global ventricular function.⁷

Afridi HI et al compared the levels of micronutrients in biological samples (whole blood, urine, and scalp hair) of myocardial infarction (MI) patients of both genders where ages ranged from 45 to 60 years at first, second and third heart attack ($n = 232$). For comparison purposes, healthy age-matched referent subjects ($n = 103$) and patients with cardiovascular without MI, of both genders were also selected. The elemental concentrations in scalp hair and whole blood were measured by flame atomic absorption spectrophotometer prior to microwave-induced acid digestion. The validity and accuracy was checked by means of certified reference materials. The results of this study showed that the mean values of K^+ and $\text{Mg}(2+)$ were significantly reduced, while the Na^+ level was higher in blood and scalp hair samples of MI patients as compared to control subjects of both genders ($p < 0.05$). The levels of $\text{Ca}(2+)$ in the biological samples of MI patients were found to be higher than in referents, but the difference was not significant ($p > 0.05$). The urinary levels of these elements were found to be higher in MI patients than in the age-matched healthy controls. Although these data do not prove a causal relationship, these results are consistent with the hypothesis that deficiency and efficiency of some essential micronutrients may play a role in the development of heart disease.⁸

Prolonged differences in blood pressure of 5 mmHg may result in a one-third reduction in stroke and one-fifth reduction in coronary events. Meta-analysis of randomized controlled trials showed that sodium reduction around 2 g per day could lower blood pressure by 2–3 mmHg, with the effect being twice as large in hypertensives.^{9–11} Geleijnse JM et al examined sodium and potassium intake in relation to cardiovascular disease (CVD) and mortality in an unselected older population. A case-cohort analysis was performed in the Rotterdam Study among subjects aged 55 years and over, who were followed for 5 years. Baseline urinary samples were analyzed for sodium and potassium in 795 subjects who died, 206 with an incident myocardial infarction and 181 subjects with an incident stroke, and in 1,448 randomly selected subjects. For potassium, dietary data were additionally obtained by food-frequency questionnaire for 78% of the cohort. There was no consistent association of urinary sodium, potassium, or sodium/potassium ratio with CVD and all-cause mortality over the range of intakes observed in this population. Dietary potassium estimated by food frequency questionnaire, however, was associated with a lower risk of all-cause mortality in subjects initially free of CVD and hypertension (RR = 0.71 per standard deviation increase; 95% confidence interval: 0.51–1.00). They observed a significant positive association between urinary sodium/potassium ratio and all-cause mortality, but only in overweight subjects who were initially free of CVD and hypertension (RR = 1.19 (1.02–1.39) per unit). The effect of sodium and potassium intake on CVD morbidity and mortality in Western societies remains to be established.¹²

CONCLUSION

Under the light of above mentioned data, it can be concluded that sodium profile is significantly altered in AMI patients.

REFERENCES

1. Esha Mati et al Source of Support: Nil Conflict of Interest: Nil infarction: its correlates and value as a determinant of in hospital outcome. *Chest*. 2000;118:904-913.
2. Kelly RF, Hursy TL, Parillo JE, Schaer GL. Effect of 100% oxygen administration on infarct size and left ventricular dysfunction in a canine model of myocardial infarction and reperfusion. *Am Heart J*. 1995;130:957–965.
3. Nordrehaug J E. Malignant arrhythmia in relation to serum potassium in acute myocardial infarction. *Am J Cardio*. 1985;56:20-23.
4. Grijseels EWM, Bouten MJM, Lenderink T, et al. Pre-hospital thrombolytic therapy with either alteplase or streptokinase. Practical implications, complications and long-term results in 529 patients. *Eur Heart J*. 1995;16:1833–1838.
5. Wilson AT, Channer KS. Hypoxaemia and supplemental oxygen therapy in the first 24 hours after myocardial infarction: the role of pulse oximetry. *J Roy Coll Phys Lond*. 1997;31:657–661.
6. Rawles JM, Kenmure ACF. Controlled trial of oxygen in uncomplicated myocardial infarction. *Br Med J*. 1976;1:1121–1123.
7. Ouwerkerk R, Bottomley PA, Solaiyappan M, et al. Tissue Sodium Concentration in Myocardial Infarction in Humans: A Quantitative ^{23}Na MR Imaging Study. *Radiology*. 2008;248(1):88-96. doi:10.1148/radiol.2481071027.
8. Afridi HI, Kazi TG, Kazi N, Kandhro GA, Baig JA, Shah AQ, Kolachi NF, Wadhwa SK, Khan S, Shah F. Potassium, calcium, magnesium, and sodium levels in biological samples of Pakistani myocardial infarction patients at different stages as related to controls. *Clin Lab*. 2010;56(9-10):427-39.
9. Khaw KT, Barrett-Connor E. Dietary potassium and stroke-associated mortality. *N Engl J Med* 1987;316:235–40.
10. Yamori Y, Nara Y, Mizushima S, et al. Nutritional factors for stroke and major cardiovascular diseases: international epidemiological comparison of dietary prevention. *Health Rep* 1994;6:22–7.
11. Tobian L, Lange J, Ulm K, et al. Potassium reduces cerebral hemorrhage and death rate in hypertensive rats, even when blood pressure is not lowered. *Hypertension* 1985;7(Suppl I):I-110-114.
12. Geleijnse JM, Witteman JCM, Stijnen T, Kloos MW, Hofman A, Grobbee DE. Sodium and potassium intake and risk of cardiovascular events and all-cause mortality: the Rotterdam Study. *European Journal of Epidemiology*. 2007;22(11):763-770. doi:10.1007/s10654-007-9186-2.

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