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Study of Serum Potassium Level in Patients of Acute Coronary Syndrome in Tertiary Care Hospital of MadhyaPradesh

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Abstract

Introduction: Current guidelines recommend a serum potassium (sK) level of 4.0-5.0 mmol/L in acute myocardial infarction patients. Recent trials have demonstrated an increased mortality rate with an sK level of >4.5 mmol/L. The aim of this study was to figure out the relation between admission sK level and in-hospital and long-term mortality and ventricular arrhythmias.

Material&Methods: Retrospectively, 611 patients with ST-elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary inter- vention were recruited. Admission sK levels were categorized accordingly: <3.5, 3.5-<4, 4-<4.5, 4.5-<5, and ≥5 mmol/L.

Results: The lowest in-hospital and long-term mortality occurred in patients with sK levels of 3.5 to <4 mmol/L. The long-term mortality risk increased for admission sK levels of >4.5 mmol/L [odds ratio (OR), 1.58; 95% confidence interval (CI) 0.42–5.9 and OR, 2.27; 95% CI 0.44-11.5 for sK levels of 4.5-<5 mmol/L and ≥5 mmol/L, respectively]. At sK levels <3 mmol/L and ≥5 mmol/L, the incidence of ventricular arrhythmias was higher (p=0.019).

Conclusion: Admission sK level of >4.5 mmol/L was associated with increased long-term mortality in STEMI. A significant relation was found between sK level of <3 mmol/L and ≥5 mmol/L and ventricular arrhythmias. (Anatol J Cardiol 2016; 16: 10-5)

Keywords: potassium, mortality, myocardial infarction, ventricular arrhythmia, hypokalemia

Introduction

Serum K level is critical in cardiovascular diseases for the prevention of adverse events. Most of the body K is intracel- lularly located (98%), and a level of 3.5-5.3 mmol/L is maintained by intra and extracellular shifts and renal excretion (1). Hypokalemia is defined as sK levels of <3.5 mmol/L and plays an important role in cardiovascular disease pathogenesis (2). Studies showed that at the acute phase of myocardial infarction (MI), hypokalemia occurs that as a consequence could lead to ventricular arrhythmia (3-7). Potassium mediates vasodilation by Na-K-ATPase pump and inwardly rectifying K channels (8). Also, K inhibits vasoconstriction associated with angiotensin-II (9). As a consequence, a low level of K further enhances infarction and ischemia. Previous studies showed that hypokalemia is a fairly common finding on admission in acute MI patients (5, 9-11). The mean admission level of sK was approximately 4 mmol/L (9, 10). This level is not defined as hypokalemia. It was reported that after ischemic attack, during the stable phase, the sK level

sig- nificantly increases with a mean value of 4.4 mmol/L (10).

The current guidelines recommend maintaining an sK level of >4-4.5 mmol/L in MI patients (1, 11). On the other hand, very recent clinical trials presented increased mortality with sK level of >4.5 mmol/L (12, 13). The objective of the present study is to figure out the relation between admission sK level and in-hospi- tal mortality, ventricular arrhythmias, long-term (six months) mortality, and hospitalization in acute ST-elevation myocardial infarction (STEMI) patients who underwent primary PCI.

Methods

Patients

The present study was a retrospective observational study. A total of 611 patients who fulfilled the criteria were recruited in the study. The study was conducted from October 2018 to December 2019 in a single center. Patients presenting within 12h of typical chest pain lasting for >30 min and diagnosed with STEMI and treated with primary PCI (angioplasty and/or stent deployment) were enrolled for the study. The specific electro- cardiographic criteria advised by the European Society Cardiology/American College of Cardiology Foundation/ American Heart Association committee were used for the diag- nosis of STEMI (14). These were new ST segment elevation of >0.1 mV in two contiguous leads or those with a true posterior MI or those with definite or probably a new left bundle branch block. Patients treated with coronary artery bypass grafting or who were treated medically or receiving dialysis or who have no follow-up data upon discharge the hospital (patients who were not contacted either by phone or who did not re-admit to hospi- tal) were excluded. The study was undertaken in a high volume tertiary center none? (>3000 PCI/year). The primary PCI proce- dures were performed by an expert interventional cardiologist who performed >75 PCI/year.

The primary end points were in-hospital and six month mor- tality and ventricular arrhythmias, and the secondary end point was hospitalization.

The Local Ethics Committee of the hospital approved the study protocol.

Data Sources

The baseline demographic data was retrospectively collected from medical records. Laboratory parameters for each patient were determined at hospital admission and on a daily basis during the hospital stay. Serum K level was measured by the ISE indirect method using Rosch, Cobas 6000 Biochemistry Auto-Analyzer, USA. The admission sK level was categorized accordingly: <3.5, 3.5-<4, 4-<4.5, 4.5-<5 and ≥5 mmol/L. The estimated glomerular filtration rate (eGFR) was calculated using the Cockcroft–Gault equation (15). The left ventricular ejection fraction (EF) was mea- sured using a modified Simpson's rule on the day of hospitaliza- tion at the coronary care unit following primary PCI (16). Follow-up data were obtained from hospital records or by interviewing the patients (directly or by telephone) or their families.

Coronary Angiography, Medication

Angiographic data of the patients were obtained from the cardiac catheterization laboratory records. Emergency coronary angiography was performed by the percutaneous femoral approach. In all cases, non-ionic low-osmolality contrast media was used. Thrombolysis in myocardial infarction score was used for PCI success. Following angioplasty, patients were admitted to the coronary care unit. The drugs were adminis- tered during and after the PCI procedure according to the European Society of Cardiology PCI Guidelines (17).

Statistical Analysis

Group significance analysis of continuous variables with normal distributions was compared using one-way analysis of variance. The Kruskal-Wallis test was used to compare continuous variables with a skewed distribution. All continuous variables are expressed as mean±SD (standard deviation). Pearson's chi-square or Fisher's exact test were used to evaluate the differences in categorical variables, which were expressed as numbers and percentages. The Kolmogorov-Smirnov test was used for testing normality.

Demographics and clinical characteristics were compared among the patients categorized by the following mean admis- sion sK levels (mmol/L); <3.5, 3.5-<4, 4-<4.5, 4.5-<5, and ≥5. Hierarchical logistic regression was then used to evaluate the independent association between mean admission sK

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levels and mortality after adjustments for the confounders. The con- founders analyzed included age, gender, eGFR, Killip class, left ventricular EF, past history (hypertension, diabetes mellitus, coronary artery disease, hyperlipidemia, and smoking status), diagnosis (anterior STEMI), cardiac enzymes (peak CK-MB lev- els during hospitalization), and medication before hospitaliza- tion. The most comprehensive logistic model was generated by the stepwise model analyze. The group of 3.5-<4 was used as the reference group in multivariate logistic analyze. A two-sided p value of <0.05 was considered to be statistically significant, and 95% CIs were presented for all odds ratios (ORs). Analyses were conducted with the Statistical Package for Social Sciences soft- ware, version 28.0 (SPSS; IBM, Armonk, New York, USA).

Results

A total of 611 patients (mean age 59±13.6 years; male 86%) were included in the present study. There were significant dif- ferences in terms of gender (p=0.006) and age (p=0.001) among the subgroups of K level. However, there was no difference in terms of body mass index (kg/m2). There were complex differ- ences in clinical characteristics among the groups. The history of patients showed diversity only with diabetes mellitus (p=0.032) and hypertension (p=0.024). There was significant difference in terms of Killip Class (p=0.001), anterior STEMI (p=0.009), and heart rate (p=0.003) at admission among the groups of sK levels. With regard to clinical outcomes, the lowest in-hospital and long-term mortality occurred in patients with K levels of 3.5-<4 mmol/L. Moreover, the highest in-hospital mortality (16%) was seen in the group of patients with sK ≥5 (p=0.002). Similarly, long- term (six months) mortality was positively correlated by increas- ing sK levels as well (p=0.007). Ventricular tachycardia/ventricu- lar fibrillation (VT/VF) was different in that its frequency increased by not only increasing sK level but also decreasing sK level (p=0.019). The patients' clinical characteristics stratified by mean sK level are listed in Table 1.

The unadjusted and adjusted models of logistic regression analysis for mortality according to sK levels are listed in Table 2. The mortality had the highest rates at sK levels of ≥5 mmol/L and that had 12.2-times higher mortality rates (95% CI: 2.5-58.5) than sK levels of 3.5-<4 mmol/L, which had the lowest rates and which was used as the reference. Compared with the reference group (3.5-<4.0 mmol/L), the long-term mortality risk was 9.55-times

Table 1. Baseline characteristics of patients by admission serum potassium levels

	Serum potassium (mmol/L) (Mean)					
	<3.5	3.5–<4	4–<4.5	4.5–<5	≥5	***P***
Patients, n	41	196	241	108	25	
Age*, years	62±11	55±12	56±12	58±11	63±15	0.001
Gender, Female	14 (34.1)	23 (11.7)	26 (10.8)	15 (13.9)	6 (24.0)	0.006
BMI, kg/m2*	27.6±4.1	27.3±3.8	27.7±4	27.7±5.5	27.4±5	0.817
Serum K, mEq/L	3.2±0.2	3.8±0.1	4.2±0.1	4.7±0.1	5.2±0.2	0.001
Patient's history						
Smoking, n (%)	24 (58.5)	151 (77)	178 (73.9)	83 (76.9)	19 (76)	0.162
Diabetes, n (%)	8 (19.5)	23 (11.7)	55 (22.8)	25 (23.1)	6 (24)	0.032
Hypertension, n (%)	25 (61)	70 (35.7)	99 (41.1)	36 (33.3)	11 (44)	0.024
Hyperlipidemia, n (%)	15 (36.6)	40 (20.4)	60 (24.9)	22 (20.4)	6 (24)	0.213
CAD, n (%)	7 (17.1)	24 (12.2)	34 (14.1)	15 (13.9)	2 (8)	0.835
Previous medication						
Beta-blocker, n (%)	5 (12.2)	25 (12.8)	30 (12.4)	14 (13)	4 (16)	0.992
ACE/ARB, n (%)	15 (36.6)	41 (20.9)	57 (23.7)	25 (23.1)	6 (25)	0.325
Diuretics, n (%)	2 (4.9)	1 (0.5)	6 (2.5)	3 (2.8)	0 (0)	0.27
ASA, n (%)	8 (19.5)	24 (12.2)	42 (17.4)	18 (16.7)	4 (16)	0.585
CCB, n (%)	6 (14.6)	5 (2.6)	12 (5)	4 (3.7)	1 (4)	0.02
Statin, n (%)	5 (12.2)	13 (6.6)	24 (10)	4 (3.7)	2 (8.3)	0.245

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OAD, n (%)	5 (12.2)	12 (6.1)	28 (11.6)	14 (13)	4 (16)	0.198
Insulin, n (%)	2 (4.9)	4 (2)	13 (5.4)	4 (3.7)	1 (4)	0.503
Laboratory parameters						
Hemoglobin, mg/dL *	13.3±1.5	14±1.6	14.1±1.6	14.4±3	13.8±2	0.180
WBC, 103/µL	11.1±3.3	12.3±3.4	13.4±12.5	12.9±7.9	13.9±5.6	0.225
LDL, mg/dL*	112±39	121±36	121±37	121±37	111±43	0.496
HDL, mg/dL*	39±11.4	39±9.6	39±11.8	38±10.6	39±12.3	0.955
CK-MB, u/L	217±162	162±142	186±150	181±195	216±147	0.051
Admission glucose,	157±51	150±54	161±76	161±74	176±100	0.695
HbA1c, %	6±0.9	6±1	6.4±1.6	6.4±1.4	6.5±1.2	0.016
Creatinine, mg/dL	0.8±0.3	0.8±0.2	0.9±0.3	1±0.3	1.3±0.7	0.001
GFR C-G, mL/min/1.73	104±43	120±45	115±49	103±46	76±40	0.001
At admission						
Killip class	1±0.2	1.1±0.4	1.2±0.6	1.2±0.7	1.8±1.2	0.001
Ejection fraction, %	44.9±6.9	46.3±8.1	44.8±9	45.5±8.8	42.2±10.5	0.119
Anterior MI, n (%)	19 (46.3)	81 (41.3)	136 (56.4)	43 (39.8)	11 (44)	0.009
SBP, mm Hg	123±19	123±22	123±23	126±28	126±42	0.398
DBP, mm Hg	71±10	71±14	71±54	74±19	74±22	0.101
HR, /min	77±11	77±11	77±14	80±13	80±17	0.003
Clinical outcomes						
Mortality, n (%) (in-	1 (2.4)	3 (1.5)	8 (3.3)	2 (1.9)	4 (16)	0.002
Mortality, n (%) (sixth	2 (4.9)	5 (2.6)	13 (5.4)	7 (6.5)	5 (20)	0.007
VT-VF, n (%)	7 (17.1)	15 (7.7)	26 (10.8)	10 (9.3)	7 (28)	0.019
Hospitalization, n (%)	7.3±4.1	6.9±6	7.9±7.8	8.1±5.9	11.2±18.6	0.092

ACE - angiotensin-converting enzyme; ARB - angiotensin II receptor blocker; ASA - acetylsalicylic acid; BMI - body mass index; CAD - coronary artery disease; CCB - calcium channel blocker; CK-MB - creatinine kinase-myocardial band; DBP - diastolic blood pressure; GFR C-G - glomerular filtration rate; Cockcroft-Gault; HDL - high density lipoprotein; HR

Table 2. Logistic regression models for mortality. All-cause mortality or ventricular fibrillation by mean admission serum potassium levels

	Serum potassium levels (mmol/L)				
Mortality	[<3.5]	[3.5 <4]	[4-<4.5]	[4.5-<5]	[≥5]
Model 1	1.6 (0.16 15.8)	1 [Reference]	2.2 (0.57 8.4)	1.2 (0.2 7.3)	12.2 (2.5 58.5)
Model 2	0.97 (0.09 10)	1 [Reference]	2.1 (0.56 8.4)	1.02 (0.16 6.3)	7.81 (1.5 39.5)
Model 3	0.91 (0.08 9.5)	1 [Reference]	2.04 (0.51 7.8)	0.76 (0.12 4.8)	3.42 (0.65 18.1)
Model 4	1.08 (0.08	1 [Reference]	1.24 (0.27 5.7)	0.37 (0.05 2.7)	1.77 (0.25 12.4)
VT-VF					
Model 1	2.48 (0.94 6.5)	1 [Reference]	1.45 (0.75 2.8)	1.23 (0.53 2.8)	4.69 (1.69 13)
Model 2	2.47 (0.92 6.6)	1 [Reference]	1.44 (0.74 2.8)	1.21 (0.52 2.7)	4.55 (1.6 12.8)
Model 3	2.46 (0.91 6.6)	1 [Reference]	1.33 (0.68 2.6)	1.08 (0.46 2.5)	2.95 (1.02 8.7)
Model 4	2.7 (0.93 7.8)	1 [Reference]	0.93 (0.43 1.9)	0.84 (0.32 2.1)	1.38 (0.34 5.5)
Long-term Mortality					
Model 1	1.95 (0.36-	1 [Reference]	2.18 (0.76 6.2)	2.64 (0.81 8.5)	9.55 (2.54 35.8)
Model 2	1.26 (0.22 6.9)	1 [Reference]	2.19 (0.76 6.3)	2.32 (0.71 7.6)	6.45 (1.64 25.3)
Model 3	1.23 (0.22 6.8)	1 [Reference]	2.07 (0.71 6.1)	1.93 (0.58 6.4)	3.48 (0.85 14.2)
Model 4	1.62 (0.24	1 [Reference]	1.53 (0.47 4.9)	1.58 (0.42 5.9)	2.27 (0.44 11.5)

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All data are presented as odds ratio (95% confidence interval) VT-VF - ventricular tachycardia-ventricular fibrillation

Logistic Models: Model 1-unadjusted model. Model 2-adjusted model for age and sex. Model 3- adjusted

Discussion

The results of the present study revealed that admission sK level of >4.5 mmol/L was associated with increased long-term mor- tality in patients with STEMI treated with primary PCI. A significant relation was detected between admission sK level of <3 mmol/L and ≥5 mmol/L and ventricular arrhythmia. An sK level of 3.5-4 mmol/L was found to be associated with the lowest mortality. Acute MI is accompanied by a catecholamine surge (3).

Catecholamine by stimulating Na-K-ATPase pump shifts K intra- cellularly, thus causing redistributional hypokalemia, and as a result, non-ischemic myocardium is hyperpolarized. As a conse- quence, electrical inhomogeneity occurs and leads to ventricu- lar arrhythmia (3, 4). Most prior studies had proposed an increased rate of ventricular arrhythmia during the acute course of MI that was found to be associated with hypokalemia (7, 18-20). Most of these studies were conducted prior to modern treatment modalities such as beta-blocker and early reperfusion treatment. Based on these previous studies, guidelines recom- mended a serum level of >4-4.5 mmol/L in acute MI (1, 11). Beta- blockers increase sK level and inhibit ventricular arrhythmias by blocking catecholamine-induced depression of K level that is derived by the inhibition of Na-K-ATPase pump by beta-2 receptors (6, 21-22). In a recent study, it was shown that the early administra- tion of beta-blockers is associated with decreased incidence of ventricular arrhythmias in STEMI (23). Additionally, beta-blockers decrease sudden cardiac death and mortality after MI (24, 25). In the present study, there was a significant difference at the extremes of sK levels. This finding is similar to the study by Goyal et al. (12) but is different from that by Choi et al. (13). In our center, all patients following the emergent PCI were administrated evidence-based treatment including appropriate beta-blocker drugs.

A high volume study performed by Goyal et al. (12) interest- ingly revealed that mean sK level above 4.5 mmol/L is associated with increased mortality. They suggested that K level between 3.5 and 4.5 mmol/L is the optimal range for acute MI patients (12). This finding was a challenge against the guidelines' recommen- dation for sK level. A very recent study conducted by Choi et al.

(13) supported Goyal et al's (12) finding. They demonstrated that mean sK level of >4.5 mmol/L is associated with increase in in- hospital and long-term mortality (13). Even though in that study, the K level of >4.5 mmol/L group was less frequently treated with beta-blockers and angiotensin-converting enzyme inhibitors, after the adjustment of confounders, the mean sK level of >4.5 mmol/L was associated with increased long-term mortality (13). Although the present study evaluated the admission sK level rather than the mean sK level, our results showed similarity with both studies (12, 13). We found that long-term mortality increased in sK level of >4.5 mmol/L.

The present study also showed that as the sK level increased, the KILLIP class also increased, and at the highest sK level, EF was the lowest. This finding was supported by Choi et al. (13). This could be partially explained by the effect of hypokalemia impairing the contractility and relaxation of myocardium because of the high levels of vasopressors (26, 27).

At the acute phase of MI, some kind of insulin resistance is seen (28, 29). It has been shown that a higher level of admission serum glucose level is seen during the acute phase of MI and that this is associated with increased in-hospital and long term mortality (28, 29). In this regard, the present study showed a positive correlation between admission sK level and admission glucose and HgA1c levels.

Serum K level is maintained by intra and extracellular shifts and renal excretion (1). As eGFR decreases, sK level increases. eGFR is an independent predictor of mortality and complications after MI (30). In the present study, a similar finding was detect- ed. Admission sK level is positively correlated with admission creatinine level and is negatively correlated with eGFR.

Study Limitations

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The present study had some limitations. This was a single- center, retrospective, observational study. Post discharge sK levels were not recorded; thus the effect of post-discharge K level on clinical outcomes could not be determined. The effect of medication on sK level and thus the outcomes were not evaluated. Hormonal changes such as serum catecholamine, insulin, and cortisol were not evaluated. Thus, the exact relation of sK level with hormonal changes was not assessed. The time of ventricular arrhythmias was not noted.

Conclusion

It can be concluded that admission sK level was signifi- cantly associated with in-hospital and long-term mortality in ASC. An sK level of >4.5 mmol/L was associated with increased long-term mortality. Ventricular arrhythmia risk was increased at extreme levels of sK. In association with recent studies, we thought that the ideal sK level could be different from that that has been proposed. To address this issue, randomized con- trolled trials should be undertaken.

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