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ORIGINAL RESEARCH

Assessment of Serum potassium levels in acute myocardial infarction patients: A cross-sectional study from a tertiary hospital

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Abstract
Background: An acute myocardial infarction (AMI) is a subset of a spectrum of IHD that includes unstable angina and AMI with or without ST elevation. The present study was conducted to assess levels of serum potassium levels in patients with acute myocardial infarction.
Materials & Methods: 92 patients of AMI of both genders were enrolled. Patients with AMI were put in group I and control in group II. Venous blood samples were collected for the estimation of potassium level.
Results: Age group <40 years had 30 patients in group I had 36 in group II and age group >40 years had 62 in group I and 56 in group II. The mean serum potassium level in group I was 3.42mEq/L and in group II was 4.89mEq/L. The difference was significant (P< 0.05).
Conclusion: Patients with AMI had lower serum potassium level as compared to healthy subjects. Changesin potassium levels might act asa predictor for assessing the prognosis.
Key words: acute myocardial infarction, hypokalemia, ventricular arrhythmia

Introduction

An Acute Myocardial Infarction (AMI) is a subset of a spectrum of IHD that includes unstable angina and AMI with or without ST elevation. Potassium is critical to the maintenance of cardio-vascular health.[1] Several studies have demonstrated a relationship between low serum potassium levels and the risk of ventricular arrhythmias in patients with AMI. Acute MI is accompanied by a catecholamine surge.[2] Catecholamine by stimulating Na-K-ATPase pump shifts K intracellularly, thus causing re-distributional hypokalemia, and as a result, non-ischemic myocardium is hyperpolarized. As a consequence, electrical inhomogeneity occurs and leads to ventricular arrhythmia.[3] 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. 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 recommended a serum level of >4-4.5 mmol/L in acute MI.[4,5]

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Potassium homeostasis is critical to prevent adverse events in patients with cardiovascular disease. Several studies have demonstrated a relationship between low serum potassium levels, usually less than 3.5mEq/L, and the risk of ventricular arrhythmias in patients with acute myocardial infarction (AMI).[6]The present study was conducted to assess levels of serum potassium levels in patients with acute myocardial infarction.

Materials & Methods

The present study comprised of 92 patients of acute myocardial infarction of both genders. The consent was obtained from all patients.

Demographic profile such as name, age, gender etc. was recorded. Patients were put in group I and healthy subjects in group II. Physical examination performed was height and weight measurement and body mass index (BMI) was calculated. 5 ml venous blood samples were collected from anti-cubital vein under aseptic precautions on the day of admission within 12 hours. All the samples were sent for analysis in laboratory for the estimation of potassium level. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

Results

Table I Distribution of patients

Age groups (years)	Group I	Group II
<40	30	36
>40	62	56

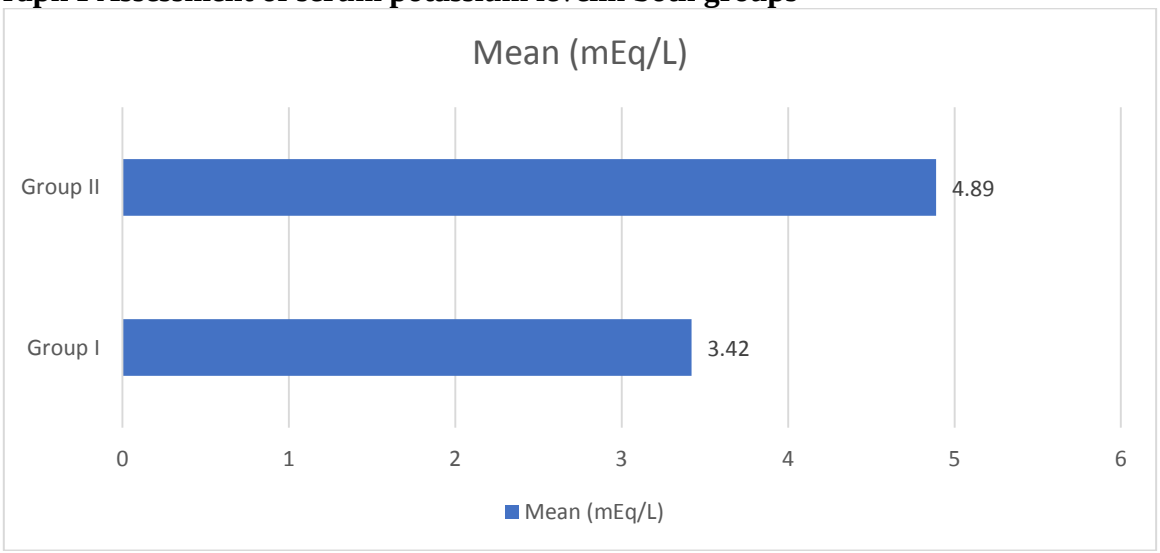
Table I shows that age group <40 years had 30 patients in group I had 36 in group II and age group >40 years had 62 in group I and 56 in group II.

Table II Assessment of serum potassium level in both groups

Groups	Mean (mEq/L)	P value
Group I	3.42	0.01
Group II	4.89	

Table II, graph I shows that mean serum potassium level in group I was 3.42 mEq/L and in group II was 4.89mEq/L. The difference was significant (P< 0.05).

Graph I Assessment of serum potassium levelin both groups



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Discussion

The definition of MI requires cardiac myocyte necrosis with an increase and/or a decrease in plasma of cardiac troponin (cTn).[7,8] At least one cTn measurement should be greater than the 99th percentile normal reference limit during: symptoms of myocardial ischemia; new (or presumably new) significant ECG ST-segment/T-wave changes or left bundle branch block; the development of pathological electrocardiographic (ECG) Q waves; new loss of viable myocardium or regional wall motion abnormality identified by an imaging procedure; or identification of intracoronary thrombus by angiography or autopsy.[9]Research showed that at the acute phase of myocardial infarction (MI), hypokalemia occurs that as a consequence could lead to ventricular arrhythmia.[10] Potassium mediates vasodilation by Na-K-ATPase pump and inwardly rectifying K channels. Also, K inhibits vasoconstriction associated with angiotensin-II. As a consequence, a low level of K further enhances infarction and ischemia.[4] Previous studies showed that hypokalemia is a fairly common finding on admission in acute MI patients. The mean admission level of sK was approximately 4 mmol/L. This level is not defined as hypokalemia. It was reported that after ischemic attack, during the stable phase, the sK level significantly increases with a mean value of 4.4 mmol/L.[11,12]The present study was conducted to assess levels of serum potassium levels in patients with acute myocardial infarction.

We found that age group <40 years had 30 patients in group I had 36 in group II and age group >40 years had 62 in group I and 56 in group II. Uluganyan et al[13]conducted a study on 611 patients with ST-elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention. Admission sK levels were categorized accordingly: <3.5, 3.5-<4, 4-<4.5, 4.5-<5, and ≥5 mmol/L. 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 for sK levels of 4.5-<5 mmol/L and ≥5 mmol/L. At sK levels <3 mmol/L and ≥5 mmol/L, the incidence of ventricular arrhythmias was higher.

We found that mean serum potassium level in group I was 3.42 mEq/L and in group II was 4.89mEq/L. Singh et al[14]assessed levels of serum potassium levels in 100 acute myocardial infarction patients. Mean serum potassium levels were higher in the control group (4.52 mEq/L) in comparison to the study group (3.99mEq/L), the values of which were found to be statistically significant. Friedensohn A et al[15] evaluated 11 patients presenting with AMI. Thirteen percent of the overall patients 11 studied had significant hypokalemia. The average initial level of potassium in patients who developed malignant arrhythmias was (4.10 mmol/liter) significantly lower than those patients who did not develop such arrhythmias (4.19 mmol/liter). To determine whether the level of potassium was, in itself, the primary cause of malignant arrhythmias following MI, a subgroup analysis of factors influencing these levels was performed. It was determined that diabetics have a higher level of potassium than non-diabetics (4.2 mmol/liter versus 4.11 mmol/liter - P = 0.01) and a lower incidence of malignant arrhythmias.

Goyal et al[16]determined the relationship between serum potassium levels and in-hospital mortality in AMI patients in the era of -blocker and reperfusion therapy.There was a U-shaped relationship between mean postadmission serum potassium level and in-hospital mortality that persisted after multivariable adjustment. Compared with the reference group of 3.5 to less than 4.0 mEq/L, mortality was comparable for mean postadmission potassium of 4.0 to less than 4.5 mEq/L. Mortality was twice as great for potassium of 4.5 to less than 5.0 mEq/L and even greater for higher potassium strata. Similarly, mortality rates were higher for potassium levels of less than 3.5 mEq/L. In contrast, rates of ventricular fibrillation or cardiac arrest were higher only among patients with potassium levels of less than 3.0 mEq/L and at levels of 5.0 mEq/L or greater.

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Bowling et al[17] found that of the 7788 patients with chronic HF, 2793 had chronic kidney disease. Of these, 527 had hypokalemia and 2266 had normokalemia. Propensity scores for hypokalemia were used to assemble a balanced cohort of 522 pairs of patients with hypokalemia and normokalemia. All-cause mortality occurred in 48% and 36% of patients with hypokalemia and normokalemia, respectively, during 57 months of follow-up (matched hazard ratio when hypokalemia was compared with normokalemia. Matched hazard ratios for cardiovascular and HF mortalities and all-cause, cardiovascular, and HF hospitalizations were 1.65, 1.82, 1.16, 1.27 and 1.29 respectively. Among 453 pairs of balanced patients with HF and chronic kidney disease, all-cause mortality occurred in 47% and 38% of patients with mild hypokalemia (3.5 to 3.9 mEq/L) and normokalemia, respectively (matched hazard ratio, 1.31; 95% CI, 1.03 to 1.66; P=0.027). Among 169 pairs of balanced patients with estimated glomerular filtration rate <45 mL/min per 1.73 m(2), all-cause mortality occurred in 57% and 47% of patients with hypokalemia (<4 mEq/L; mild) and normokalemia, respectively.
The limitation the study is small sample size.

Conclusion

Authors found that patients with AMI had lower serum potassium level as compared to healthy subjects. Changesin potassium levelsmight act asa predictor for assessing the prognosis.

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