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ORIGINAL RESEARCH

Assessment of changes occurring in serum potassium profile in AMI patients: A biochemical study

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***ABSTRACT*:**
Background: Acute myocardial infarction (AMI) is one of the leading causes of death in the developed world. Changes in intracellular and extracellular potassium levels modify the electrophysiological properties of the resting membrane potential in cardiac cells and subsequently influence myocardial impulses generation and conduction. Hence; the present study was undertaken to assess changes occurring in serum potassium profile in AMI patients. **Materials & methods:** A total of 50 AMI patients and 50 age and gender matched healthy controls were included in the present study. Serum samples were obtained from all the subjects and were sent to the central laboratory for assessment of serum potassium profile. Auto-analyzer was used for evaluating the serum potassium levels. All the results were recorded and analyzed by SPSS software **Results:** Mean potassium levels of the AMI group and the control group were found to be 3 mEq/L and 3.8 mEq/L respectively. Significant results obtained while comparing the mean serum potassium profile of the AMI patients and healthy controls. **Conclusion:** Serum potassium alterations play a significant role in the pathogenesis of AMI.
Key words: Acute myocardial infarction, Potassium

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INTRODUCTION

Acute myocardial infarction (AMI) is one of the leading causes of death in the developed world. Acute myocardial infarction can be divided into two categories, non-ST-segment elevation MI (NSTEMI) and ST-segment elevation MI (STEMI). Unstable angina is similar to NSTEMI.[1] The pathogenesis of AMI is multifactorial; however, several studies have implicated impaired lipid metabolism as one of the crucial factors in the development of this disease. Serum potassium levels play a major role in the outcome of cardiovascular (CV) events.[2, 3] Changes in intracellular and extracellular potassium levels modify the electrophysiological properties of the resting membrane potential in cardiac cells and subsequently influence myocardial impulses generation and conduction. Serum potassium levels are maintained between 3.5 and 5.2 mEq/L by renal excretion, and shift between intracellular and extracellular fluid compartments.[4- 6] Hence; the present study was undertaken to assess changes occurring in serum potassium profile in AMI patients.

MATERIALS & METHODS

The present study was undertaken with the aim of analyzing the serum potassium profile in AMI patients. Ethical approval was obtained before the starting of the study. Written consent was also obtained from all the patients after explaining in detail the entire research protocol. A total of 50 AMI patients and 50 age and gender matched healthy controls were included in the present study. Exclusion criteria for the present study included:

- Subjects with history of any other metabolic disorders,
- Subjects with history of any other systemic illness,
- Subjects with any known drug allergy

Serum samples were obtained from all the subjects and were sent to the central laboratory for assessment of serum potassium profile. Auto-analyzer was used for evaluating the serum potassium levels. All the results were recorded and analyzed by SPSS software. Unpaired t test was used for assessment of level of significance.

RESULTS

In the present study, a total of 100 subjects were analyzed. Among these 100 subjects, 50 were AMI subjects while the remaining 50 were healthy control subjects. Mean age of the subjects of the AMI group and the healthy control group was 45.8 years and 46 years respectively. There were 30 males and 20 females in the AMI group and there were 28 males and 22 females in the control group. Mean potassium levels of the AMI group and the control group were found to be 3 mEq/L and 3.8 mEq/L respectively. Significant results obtained while comparing the mean serum potassium profile of the AMI patients and healthy controls.

DISCUSSION

In the early phases of acute myocardial infarction (AMI), the sympathetic nervous system is activated, as reflected by elevated levels of plasma catecholamines and modulation of β adrenergic receptor signaling. This activation leads to

intracellular influx of potassium and decrease in serum potassium levels. Low potassium levels have been shown to increase the automaticity and excitability of myocardial cells, leading to the propensity for ventricular arrhythmias.[7-9]

Table 1: Comparison of demographic data

Parameter		AMI group	Control group
Mean age (years)		45.8	46
Gender	Males	30	28
	Females	20	22

Table 2: Comparison of mean serum potassium profile

Parameter	AMI group	Control group	p- value
Mean serum potassium levels (mEq/L)	3	3.8	0.00 (Significant)

In the present study, a total of 100 subjects were analyzed. Among these 100 subjects, 50 were AMI subjects while the remaining 50 were healthy control subjects. Mean age of the subjects of the AMI group and the healthy control group was 45.8 years and 46 years respectively. In most cases, an AM is caused by occlusion (blockage) of one or more coronary blood vessels by a thrombus (blood clot), and it is accompanied by severe crushing chest pain. In a minority of AMIs, but more commonly in the elderly, patients experience no pain. In some cases the reduced blood flow is caused by a blood vessel problem other than a thrombus.[10]

There were 30 males and 20 females in the AMI group and there were 28 males and 22 females in the control group. Mean potassium levels of the AMI group and the control group were found to be 3 mEq/L and 3.8 mEq/L respectively. Pohl W et al analyzed the serum magnesium concentration (MGK) and serum potassium concentration (KK) in 176 patients with acute myocardial infarction (AMI) during the first 48 hours after AMI. The patients rhythm was continuously recorded. In a subgroup of 70 patients a signal averaging-ECG was performed. 4.5% of the patients had a low, 55.7% a normal and 39.8% a high MGK. 14.8% of the patients had a low, 80.1% a normal and 5.1% a high KK. Ventricular arrhythmias > or = Lown IV b were found in 25% of the patients with low MGK, in 38.8% with normal and in 52.9% with high MGK. 50% of the patients with low, 62.2% with normal and 61.3% with high MGK had late potentials. There was no relation between hypomagnesemia and ventricular arrhythmias as between hypomagnesemia and late potentials. Thus, hypomagnesemia in AMI patients is rare and does not correlate with ventricular arrhythmia or delayed ventricular potentials.[9]

In the present study, significant results obtained while comparing the mean serum potassium profile of the AMI patients and healthy controls. The prognosticator role of normal range admission potassium levels among patients with AMI have been described in the past. Higher potassium levels (≥4.3 mEq/L) were shown to be an independent risk factor for target lesion revascularization among AMI patients. In addition, higher admission potassium levels (≥4.3 mEq/L) were also found to be associated with a larger scintigraphic infarct size in patients with ST-elevation myocardial infarction. These findings suggest that higher potassium levels, although in the normal range, may be associated with complications that are not necessarily related to immediate changes in the electrophysiological properties of the myocardium, and thus can be associated with late morbidity and mortality.[9, 10]

CONCLUSION

Changes in intracellular and extracellular potassium levels modify the electrophysiological properties of the resting membrane potential in cardiac cells and subsequently influence myocardial impulses generation and conduction. Therefore; serum potassium alterations play a significant role in the pathogenesis of AMI.

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