ORIGINAL RESEARCH

Assessment of serum potassium levels in acute myocardial infarction patients

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ABSTRACT
Background: To assess serum potassium levels in subjects with acute myocardial infarction.**Materials & methods:**A total of 40 subjects were enrolled. Demographic, clinical and treatment data were obtained. AMI was diagnosed. Serial serum potassium levels during hospitalization were obtained from the patients' medical records and were reviewed. The results were analysed using SPSS software.**Results:**Out of 40 subjects, number of subjects with lower potassium levels i.e<3.0 mEq/L are 3 and death rate is 33.4 % whereas death rate is less in patients with potassium levels in the range of 3.5 to 4.5 mEq/L.**Conclusion:**There is a U shaped relationship between potassium levels and mortality rate of myocardial infarction.
Keywords: Myocardial infarction, Potassium.

INTRODUCTION

Serum K (sK) level is critical in cardiovascular diseases for the prevention of adverse events. Most of the body K is intracellularly 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.[3,4] Potassium (K) mediates vasodilation by Na-K-ATPase pump and inwardly rectifying K channels.[5] Also, K inhibits vasoconstriction associated with angiotensin-II.[6]

Potassium has an essential role in maintaining myocardial electrical stability. Hypokalemia was first noted to decrease the fibrillation threshold in rabbit hearts over 60 years ago. [7] In the setting of myocardial ischemia, adrenergic stimulation activates the Na–K ATPase pump, lowering plasma potassium levels. [3] Several small, observational studies have suggested that in the setting of acute coronary syndrome (ACS), hypokalemia, typically defined as potassium levels <3.5 mEq/L, is associated with ventricular arrhythmias.[8-10] As a result, both professional guidelines and experts have recommended a target potassium level of at least 4.0 mEq/L in the setting of ACS.[11,12] However, the body of evidence behind this recommendation predates several advances in the management of ACS that are known to reduce the risk of ventricular arrhythmias, specifically the routine use of β blockers and early reperfusion strategies. Two contemporary retrospective cohort studies of patients post-myocardial infarction (MI) observed the lowest in-hospital and three-year mortality rates in patients whose potassium levels were between 3.5 and 4.5 mEq/L.[13,14] Hence, this study was conducted to assess serum potassium levels in subjects with acute myocardial infarction.

MATERIALS & METHODS

A total of 40 subjects were enrolled. Demographic, clinical and treatment data were obtained. AMI was diagnosed. Serial serum potassium levels during hospitalization were obtained from the patients' medical records and were reviewed. Laboratory investigations were done. Patients were categorized into six groups to determine the relation between mean serum potassium and long-term mortality:<3.0, 3.0 to <3.5, 3.5 to <4.0, 4.0 to <4.5, 4.5 to <5.0, and 5 mEq/L. The results were analysed using SPSS software.

RESULTS

Out of 40 subjects, number of subjects with lower potassium levels i.e<3.0 mEq/L are 3 and death rate is 33.4 % whereas death rate is less in patients with potassium levels in the range of 3.5 to 4.5 mEq/L and further mortality increases with increased level of

potassium i.e more than 5.0 mEq/L. This shows a U-shaped relationship between the mortality rate and the potassium levels.

Table: Potassium levels and mortality due to MI

Potassium levels (mEq/L)	Number of patients	Number of deaths due to MI	Mortality percentage(%)
< 3.0	3	1	33.4
3.0 to<3.5	4	1	25
3.5 to <4.0	6	1	16.7
4.0 to <4.5	8	1	12.5
4.5 to <5.0	11	6	54.5
> 5.0	8	5	62.5
Total	40		

DISCUSSION

Most studies reporting an association between hypokalemia and arrhythmic death in ACS predate the era of reperfusion and widespread use of β blockers and, in general, included relatively small patient populations. [9,10] Nonetheless, these studies, together with animal models, were the basis for the common practice of targeting potassium levels of 4.0–5.0 mEq/L in the setting of ACS. [12,13] Since these recommendations were published, two retrospective cohort studies have challenged the benefit of consistently targeting potassium levels >4.0 mEq/L. Hence, this study was conducted to assess serum potassium levels in subjects with acute myocardial infarction.

In the present study, out of 40 subjects, number of subjects with lower potassium levels i.e<3.0 mEq/L are 3 and death rate is 33.4 % whereas death rate is less in patients with potassium levels in the range of 3.5 to 4.5 mEq/L. A study by Patel RB et al, showed that NSVT lasting for at least eight consecutive beats occurred more frequently at potassium levels <3.5 mEq/L than at potassium levels ⩾5 mEq/L (10.1 vs. 4.5%, p=0.03 for trend), whereas the inverse pattern was observed for ventricular pauses >3 s, which occurred more frequently at potassium levels ⩾5 mEq/L than at potassium levels <3.5 mEq/L (5.9 vs. 2.0%, p=0.03 for trend). There was a U-shaped relationship between the potassium level at admission and both early and late risk of cardiovascular death. Compared with patients with potassium levels of 3.5 to <4 mEq/L, a potassium level <3.5 mEq/L was associated with an increased risk of cardiovascular death at day 14 (2.4 vs. 0.8%, HRadj 3.1, p=0.02) and at one year (6.4 vs. 3.0%, HRadj 2.2, p=0.01). The risk of cardiovascular death at one year was also significantly increased at potassium levels ⩾4.5 mEq/L and a similar trend was noted at potassium levels ⩾5 mEq/L. The lowest risk of cardiovascular death was observed in patients with admission potassium levels between 3.5 and 4.5 mEq/L. Both lower and higher levels of potassium were associated with tachyarrhythmias and bradyarrhythmias, suggesting a potential mechanistic explanation for the increased risk of cardiovascular death at the extremes of potassium homeostasis. [15]

In the present study, further mortality increases with increased level of potassium i.e more than 5.0 mEq/L. This shows a U- shaped relationship between the mortality rate and the potassium levels. Another study by Ma W et al, showed patients with potassium levels of 4.0 to <4.5 mEq/L had the lowest predefined event rates, which were 6.4% for 7-day malignant arrhythmia, 3.7% for 7-day mortality, and 5.3% for 30-day mortality. Compared with the reference group (4.0 to <4.5 mEq/L), multivariate regression analysis revealed significantly higher 30-day mortality risk in patients with potassium level of 4.5 to <5.0 (hazard ratio [HR]: 1.52, 95% confidence interval [CI]: 1.17-1.98; P = .002) and even higher risk in patients with potassium level of ≥5.0 mEq/L (HR: 1.80, 95% CI: 1.22-2.66; P = .002). The lowest 30-day mortality was observed in patients with STEMI having potassium levels between 4.0 and 4.5 mEq/L, and a level >4.5 mEq/L significantly increased mortality risk. [16] Acute MI is accompanied by a catecholamine surge.[3] Catecholamine by stimulating Na-K-ATPase pump shifts K intracellularly, thus causing redistributional hypokalemia, and as a result, non-ischemic myocardium is hyperpolarized. As a consequence, electrical inhomogeneity occurs and leads to ventricular 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.[9,10] 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. [13]Goyal et al. [17] noted a lower in-hospital mortality in a cohort of over 30,000 patients post-MI with potassium levels of 3.5–4.5 mEq/L. Subsequently, Choi et al. [18] noted similar findings in a cohort of over 2000 patients in South Korea.Acute MI is accompanied by a catecholamine surge. [3] Catecholamine by stimulating Na-K-ATPase pump shifts K intracellularly, thus causing redistributional hypokalemia, and as a result, non-ischemic myocardium is hyperpolarized. As a consequence, electrical inhomogeneity occurs and leads to ventricular 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. [10]

CONCLUSION

There is a U shaped relationship between potassium levels and mortality rate of myocardial infarction.

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