International Journal of Research in Health and Allied Sciences

Journal home page: www.ijrhas.com

Official Publication of "Society for Scientific Research and Studies" [Regd.]

ISSN: 2455-7803

Original Research

Potassium levels and acute myocardial infarction

[1]Mohit Mohindra, [2]Bhagwant Singh

[1]Department of Neurosurgery, Medanta – The Medicity, Gurgaon, Haryana, India
[2]Junior Resident, Department of General Medicine, DMC &H, Ludhiana, Punjab, India

ABSTRACT:
Background: To evaluate the relationship between potassium levels and acute myocardial infarction. **Materials & methods:** A total of 50 patients were enrolled. Demographic, clinical, laboratory, and treatment data were obtained. 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 50 subjects, number of subjects with lower potassium levels i.e <3.0 mEq/L are 4 and death rate is 25 % 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.

Received: 19 November, 2022 Accepted: 27 December, 2022

Corresponding Author: Mohit Mohindra, Department of Neurosurgery, Medanta – The Medicity, Gurgaon, Haryana, India

This article may be cited as: Mohindra M, Singh B. Potassium levels and acute myocardial infarction. Int J Res Health Allied Sci 2023; 9(2):58-60.

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 mediates vasodilation by Na-K-ATPase pump and inwardly rectifying K channels.[5] Also, K inhibits vasoconstriction associated with angiotensin-II.[6] 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.[7,8] The mean admission level of sK was approximately 4 mmol/L.[8] 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. [9]

Hyperkalemia is a common electrolyte abnormality that can lead to serious and potentially fatal cardiac dysrhythmias. [10] Prior work has consistently supported the link between hyperkalemia and adverse cardiovascular outcomes. [11,12] In the modern era, the prevalence of chronic kidney disease and diabetes, both associated with hyperkalemia, is rising. [13] Additionally, there has been widespread adoption of practices that increase the risk of hyperkalemia in acute myocardial infarction survivors with or without incident heart failure. Examples of such therapies include guideline-directed use of beta-blockers, mineralocorticoid receptor antagonists, and renin angiotensin aldosterone system antagonists. [14] The increase in the use of these medications has been associated with significant increases in hyperkalemia-related hospitalization and deaths. [15,16] Further, procedures such as percutaneous intervention and coronary artery bypass grafting, are common in the contemporary management of acute myocardial infarction, and may indirectly contribute to the risk of hyperkalemia via the associated incidence of contrast-induced nephropathy and acute kidney injury, respectively.[17] Hence, this study was conducted to evaluate the relationship between potassium levels and acute myocardial infarction.

MATERIALS & METHODS

A total of 50 patients were enrolled. Demographic, clinical, laboratory, 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 50 subjects, number of subjects with lower potassium levels i.e <3.0 mEq/L are 4 and death rate is 25 % 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	4	1	25
3.0 to<3.5	6	1	16.7
3.5 to <4.0	8	1	12.5
4.0 to <4.5	9	1	11.1
4.5 to <5.0	14	8	57.2
> 5.0	9	6	66.7
Total	50		

DISCUSSION

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.[3,18] 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.[19] 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. [6] Hence, this study was conducted to evaluate the relationship between potassium levels and acute myocardial infarction.

In the present study, out of 50 subjects, number of subjects with lower potassium levels i.e <3.0 mEq/L are 4 and death rate is 25 % whereas death rate is less in patients with potassium levels in the range of 3.5 to 4.5 mEq/L. A study by Goyal A et al, 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 rate, 4.8%; 95% CI, 4.4%-5.2%), mortality was comparable for mean postadmission potassium of 4.0 to less than 4.5 mEq/L (5.0%; 95% CI, 4.7%-5.3%), multivariable-adjusted odds ratio (OR), 1.19 (95% CI, 1.04-1.36). Mortality was twice as great for potassium of 4.5 to less than 5.0 mEq/L (10.0%; 95% CI, 9.1%-10.9%; multivariable-adjusted OR, 1.99; 95% CI, 1.68-2.36), 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.Among inpatients with AMI, the lowest mortality was observed in those with postadmission serum potassium levels between 3.5 and <4.5 mEq/L compared with those who had higher or lower potassium levels.[20]

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 Uluganyan M et al, 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). 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.[21]Recognition of hyperkalemia is especially important in patients with cardiovascular disease, such as those with acute myocardial infarction. Our findings highlight the frequency of hyperkalemia following acute myocardial infarction. Though treatment effect was not evaluated in this analysis, it is noteworthy that resolution of hyperkalemia does portend a better prognosis than persistent hyperkalemia. They feel that the poor prognosis associated with hyperkalemia should prompt clinicians to evaluate their patients for potentially modifiable factors that are associated with hyperkalemia following acute myocardial infarction. In terms of hyperkalemia management, the few more traditional approaches to pharmacologic management of hyperkalemia, including intravenous insulin and

sodium bicarbonate, have conflicting data supporting their efficacy. Several new products are in development, including patiromer calcium and sodium zirconium cyclosilicate (ZS-9). [22,23]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.[24] In a recent study, it was shown that the early administration of beta-blockers is associated with decreased incidence of ventricular arrhythmias in STEMI.[25] Additionally, beta-blockers decrease sudden cardiac death and mortality after MI. [26]

CONCLUSION

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

REFERENCES

1. Macdonald JE, Struthers AD. What is the optimal serum potassium level in cardiovascular patients? J Am Coll Cardiol. 2004;43:155–61.
2. Schulman M, Narins RG. Hypokalemia and cardiovascular disease. Am J Cardiol. 1990;65:4–9.
3. Brown MJ, Brown DC, Murphy MB. Hypokalemia from beta-2 receptor stimulation by circulating epinephrine. N Engl J Med. 1983;309:1414–9.
4. Friedensohn A, Faibel HE, Bairey O, Goldbourt U, Schlesinger Z. Malignant arrhythmias in relation to values of serum potassium in patients with acute myocardial infarction. Int J Cardiol. 1991;32:331–8.
5. Dawes M, Sieniawska C, Delves T, Dwivedi R, Chowienczyk PJ, Ritter JM. Barium reduces resting blood flow and inhibits potassium-induced vasodilation in the human forearm. Circulation. 2002;105:1323–8.
6. Cohn JN, Kowey PR, Whelton PK, Prisant LM. New guidelines for potassium replacement in clinical practice:a contemporary review by the National Council on Potassium in Clinical Practice. Arch Intern Med. 2000;160:2429–36.
7. Madias NE, Shah B, Chintalapally G, Chalavarya G, Madias NE. Admission serum potassium in patients with acute myocardial infarction:its correlates and value as a determinant of in-hospital outcome. Chest. 2000;118:904–13.
8. Campbell WB, Schmitz JM. Effect of alterations in dietary potassium on the pressor and steroidogenic effects of angiotensins II and III. Endocrinology. 1978;103:2098–104.
9. Sekiyama H, Nagoshi T, Komukai K, Matsushima M, Katoh D, Ogawa K, et al. Transient decrease in serum potassium level during ischemic attack of acute coronary syndrome:paradoxical contribution of plasma glucose level and glycohemoglobin. Cardiovasc Diabetol. 2013;12:4.
10. Goyal A, Spertus JA, Gosch K, et al. Serum potassium levels and mortality in acute myocardial infarction. Jama. 2012 Jan 11;307(2):157–164.
11. An JN, Lee JP, Jeon HJ, et al. Severe hyperkalemia requiring hospitalization: predictors of mortality. Critical care (London, England) 2012;16(6):R225.
12. McCullough PA, Beaver TM, Bennett-Guerrero E, et al. Acute and chronic cardiovascular effects of hyperkalemia: new insights into prevention and clinical management. Reviews in cardiovascular medicine. 2014;15(1):11–23.
13. Services USDoHaH. National Diabetes Statistics Report: Estimates of Diabetes and Its Burden in the United States. Centers for Disease Control and Prevention. 2014
14. Juurlink DN, Mamdani MM, Lee DS, et al. Rates of hyperkalemia after publication of the Randomized Aldactone Evaluation Study. The New England journal of medicine. 2004 Aug 5;351(6):543–551.
15. Wozakowska-Kaplon B, Janowska-Molenda I. Iatrogenic hyperkalemia as a serious problem in therapy of cardiovascular diseases in elderly patients. Polskie Archiwum Medycyny Wewnetrznej. 2009 Mar;119(3):141–147.
16. Fordjour KN, Walton T, Doran JJ. Management of hyperkalemia in hospitalized patients. The American journal of the medical sciences. 2014 Feb;347(2):93–100.
17. Garovic VD, Textor SC. Renovascular Hypertension and Ischemic Nephropathy. Circulation. 2005 Aug 30;112(9):1362–1374. 2005.
18. Obeid AI, Verrier RL, Lown B. Influence of glucose, insulin, and potassium on vulnerability to ventricular fibrillation in the canine heart. Circ Res. 1978;43:601–8.
19. Kafka H, Langevin L, Armstrong PW. Serum magnesium and potassium in acute myocardial infarction. Influence on ventricular arrhythmias. Arch Intern Med. 1987;147:465–9.
20. Goyal A, Spertus JA, Gosch K, Venkitachalam L, Jones PG, Van den Berghe G, Kosiborod M. Serum potassium levels and mortality in acute myocardial infarction. JAMA. 2012 Jan 11;307(2):157-64.
21. Uluganyan M, Ekmekçi A, Murat A, Avşar Ş, Ulutaş TK, Uyarel H, Bozbay M, Çiçek G, Karaca G, Eren M. Admission serum potassium level is associated with in-hospital and long-term mortality in ST-elevation myocardial infarction. Anatol J Cardiol. 2016 Jan;16(1):10-5.
22. Weir MR, Bakris GL, Bushinsky DA, et al. Patiromer in patients with kidney disease and hyperkalemia receiving RAAS inhibitors. The New England journal of medicine. 2015 Jan 15;372(3):211–221.
23. Kosiborod M, Rasmussen HS, Lavin P, et al. Effect of sodium zirconium cyclosilicate on potassium lowering for 28 days among outpatients with hyperkalemia: the HARMONIZE randomized clinical trial. Jama. 2014 Dec 3;312(21):2223–2233.
24. Johansson BW, Dziamski R. Malignant arrhythmias in acute myocardial infarction:relationship to serum potassium and effect of selective and nonselective beta-blockade. Drugs. 1984;28:77–85.
25. Park KL, Goldberg RJ, Anderson FA, López-Sendón J, Montalescot G, Brieger D, et al. GRACE Investigators. Beta-blocker Use in ST-segment Elevation Myocardial Infarction in the Reperfusion Era (GRACE) Am J Med. 2014;127:503–11.
26. The Norwegian Multicenter Study Group. Timolol-induced reduction in mortality and reinfarction in patients surviving acute myocardial infarction. N Engl J Med. 1981;304:801–7.