Clinical Case Reports International

Review Article
Published: 14 Jan, 2023

The Role of Oral Nutrition in Relation to Potassium, Sodium and Creatinine Levels in Acute Myocardial Infarction

*Kanjilal A[1], Datta P[2], Bhattacharjee S[2], Ray A[3]**

[1]Department of Food & Nutrition, University of Calcutta, India

[2]Department of Food & Nutrition, University of Calcutta, India

[3]Department of Food & Nutrition, Maharani Kasiswari College, University of Calcutta, India

Abstract

Myocardial infarction is one of the leading causes of death and severe morbidity in people all over the world. It is the end result of a number of heart conditions which occur due to multifactorial causes. One of the main pathophysiological factors that is related to impaired coronary functioning is the electrolyte balance in the body. The levels of sodium and potassium play a significant role in this regard as does creatinine. These are in turn associated with the kind of food and diet that is consumed by an individual. Although genetics also plays a very crucial role, the interplay of it with food molecules is in primary focus now as a cause of heart diseases as well as other lifestyle diseases like diabetes, cancer etc.

This paper deals with the relation of different levels of sodium and potassium in relation to the occurrence of myocardial infarction and also the role of oral nutrition on their levels and in turn heart conditions in the human body.

Keywords: Myocardial infarction; Sodium; Potassium; Creatinine; Lifestyle diseases

OPEN ACCESS

Correspondence:
Anindita Ray (Chakravarti), Department of Food & Nutrition, Maharani Kasiswari College, University of Calcutta, Kolkata, India,
E-mail: anindita.ray.chakravarti@gmail.com

Received Date: *21 Dec 2022*
Accepted Date: *09 Jan 2023*
Published Date: *14 Jan 2023*

Citation:
Kanjilal A, Datta P, Bhattacharjee S, Ray A. The Role of Oral Nutrition in Relation to Potassium, Sodium and Creatinine Levels in Acute Myocardial Infarction. Clin Case Rep Int. 2023; 7: 1459.

Introduction

Myocardial infarction is an ischemic necrosis of the myocardium, caused by occlusion of the coronary artery and prolonged myocardial ischemia. Myocardial Infarction (MI) is one of the five main manifestations of coronary heart disease, namely stable angina pectoris, unstable angina pectoris, MI, heart failure and sudden death. Differences in lifestyle, behavioral pattern, genetic susceptibility and non-genetic factors such as hypertension, obesity, diabetes are the risk factors associated with MI. Serum electrolyte imbalances after an episode of Acute Myocardial Infarction (AMI) are common. Serum electrolytes play an important role in maintaining electrophysiological homeostasis of the myocardial membrane, and alterations of these electrolyte levels can affect the pathogenesis, complications of myocardial infarction. Major serum electrolytes affecting the myocardial electrophysiological properties are sodium and potassium. Decrease in serum sodium concentration <136 mEq/L (<136 mmol/L) is Hyponatremia. It is relatively common in patients with acute MI and is the most common electrolyte disorder in hospitalized patients in diverse clinical settings. It has been recognized as a worse prognostic indicator in patients with ST-segment Elevation Myocardial Infarction (STEMI). Decrease in serum potassium concentration <3.5 mmol/L is Hypokalemia. It is also common in patients hospitalized with acute myocardial infarction and is associated with increased morbidity particularly arrhythmias and mortality. It is even found to be associated with larger infarcts. Since, Acute Myocardial Infarction (AMI) is one of the leading causes of morbidity and mortality across the world, serum electrolytes with special reference to serum sodium and potassium is observed to detect any changes and treated properly in patients with MI and the correlation of serum sodium and potassium in the severity and outcome of AMI is also studied.

Review

A reduction or interruption of blood flow to a section of the heart, resulting in cardiac muscle necrosis, is the most common cause of acute myocardial infarction, more often known as a heart attack. The most common cause of a myocardial infarction is a reduction or cessation of blood flow

to a portion of the heart, which results in cardiac muscle necrosis. Myocardial Infarction (MI) had already undergone multiple name changes since the 1970s. The distinction between transmural MI and non-transmural MI was made in 1960s and 1970s in the study of MI. Endocardium, myocardium, and epicardium are all involved in transmural MI, which occurs, when the ischemia and damage extend throughout the full thickness of the cardiac muscle. To put it another way, this was often caused by the full obstruction of one of the three layers of the cardiac muscle by an epicardial thrombus. When ischemia and damage do not impact all three layers of the cardiac muscle, it is said to be non-transmural MI, which usually spares the epicardium. This was attributed to a considerable reduction in blood flow to the area, whether or not a coronary artery or branches was completely blocked.

Worldwide, cardiovascular disease ranks high among the leading causes of death and disability. Lifestyle and behavioral risk factors can vary by region in countries like India. Differences between rural and urban areas, as well as between the East and West, the North and the South, are also very prominent. Sodium and potassium are the main serum electrolytes that influence the electrical characteristics of the myocardium [1]. Potassium (K+) being originally extracted in 1807 by electrolyzing potash (plant ashes immersed in pots of water), hence the name. K+ was first isolated from a natural source, but its biological significance was also not understood until the 20th century.

Potassium (K) is an intracellular ion, and renal excretion and exchanges between the intracellular and extracellular space primarily control the lower extracellular K levels. Changes in extracellular K and its effect on the resting membrane potential of myocardial cells can affect the heart's ability to generate and conduct electrical impulses. Although we know that both low and high serum K levels are associated with a worse prognosis in AMI patients, the underlying mechanism through which this occurs is unclear [2].

Several systemic health issues are linked to the stress caused by an AMI. Changes in metabolism include elevated levels of catecholamines, free fatty acids, glucose, glycerol, cortisol, and cyclic Adenosine Monophosphate in the blood (cAMP) are the factors.

Patients with acute coronary syndrome have been shown to have a strong association between the depth of their potassium drop and their blood sugar levels, initially observed by Sekiyama et al. Patients with a greater K drop during hospitalization also had a longer hospital stay, a higher incidence of myocardial infarction, and a higher peak of creatine kinase levels. Additionally, the elevated catecholamines in AMI may stimulate the Na + -K + -ATPase pump, leading to intracellular K shift, which may reflect the hypokalemia mostly in acute period of AMI. As per some studies patients diagnosed with AMI who were administered beta-blockers did not experience an early hypokalemic drop [3].

Nevertheless, dietary potassium could be just as potent a driver of cardiovascular disease and death as dietary salt. Potassium depletion has been associated with a rise in stroke and sudden cardiac death, while increasing potassium intake (without altering dietary sodium) can decrease blood pressure in both experimental and therapeutic hypertension. This may also inhibit the activity of the sympathetic nervous as well as renin-angiotensin mechanisms and reduce the risk of vascular injury [2].

Physiological mechanisms that conserved sodium and excreted potassium arose through evolutionary processes because prehistoric animals and human beings ingested a diet low in sodium while enriched in potassium. It was observed in some studies that sodium levels in the diet have risen dramatically with the rise of civilized societies, while potassium levels have fallen. Excess sodium has been linked to high blood pressure and heart failure. The potential link between low potassium intake and neurodegenerative disorders including Alzheimer's, Parkinson's, and Parkinson's has been overlooked in favor of the more familiar focus on sodium. Potassium has diuretic impact, can alter the heart's electrical characteristics, but also can decrease the occurrence and severity of possibly fatal ventricular tachyarrhythmias in people who have chronic heart failure [4].

Acute myocardial infarction is frequently followed by heart failure, which necessitates the use of a diuretic to alleviate the symptoms including water retention. Patients may experience ventricular and supraventricular arrhythmias, as well as cardiac arrest, when using diuretics, which has been linked to hypokalemia and complicated cardiac arrhythmias. Cardiomyocyte resting membrane potentials are influenced by serum potassium displacement, resulting in hyperpolarized cells and enhanced excitability and automaticity. After a MI, it's crucial to look at whether or not specific potassium levels within the expected level put patients at danger or seems particularly safe. Both hypo- and hyperkalemia are linked to an increased mortality rate.

Mechanisms that kept sodium in the body and excreted potassium were able to evolve because humans developed a diet low in sodium and high in potassium. The modern diet, which is high in sodium, causes sodium excess while simultaneously depleting potassium levels [5]. Hypokalemia is a factor in the development of cardiovascular disease, which also plays a role in the aggravation of hypokalemia by a variety of cardiovascular illnesses and medications. As a consequence of this, hypokalemia is a prevalent and treatable component in the clinical history of MI. Depending on intracellular and external potassium concentrations, the resting transmembrane potential differential can vary. Hypokalemia promotes cellular hyper polarity, resting potential, depolarization. All of which lead to hyperpolarized cells. Hypokalemia expands the action potential, which suppresses hypokalemic ventricular ectopy, while potassium supplementation shortens the action potential. Hypokalemia, on the other hand, raises the probability for ventricular arrhythmia and abrupt death from heart failure.

Terminal renal disease has been established in the past to be a strong indicator of death. It is not yet clear how impaired kidney function & coronary artery disease interact, however the blood creatinine ratio may be a signal for coexisting cardiovascular risk factors such as diabetes, general hypertension, as well as advanced age. Acute coronary syndromes can lead to a considerable increase in mortality if there is a decrease in creatinine clearance or Glomerular Filtration Rate (GFR) despite the reason [6]. A recent research study has shown that long-term mortality increases as creatinine levels rise above 1.3 mg/dL, and the difference is statistically significant at this point. According to the GRACE registry, a one-milligram/deciliter rise in baseline Creatinine found linked to 1,2 times increase in the probability of dying in the hospital. Creatinine levels have been proven to have a stronger predictive value than cardiac enzyme activities. For patients with acute myocardial infarction, it was found that patients who had increased serum creatinine concentrations died more quickly in the hospital. In both individuals with severe renal failure and people who had mild renal dysfunction, this unfavorable

result was observed. Patients with mild creatinine levels (1.2 mg/dL to 2.0 mg/dL) died at a rate of 17.1%, whereas patients with normal creatinine levels (1.2 mg/dL) died at a rate of 3.9%.

There is a correlation between renal failure and an increased risk of cardiovascular disease and cardiovascular results in patients [6]. Having renal failure has been found to be a significant determinant of survival in patients with acute coronary syndrome & acute myocardial infarction in such a number of epidemiologic research and clinical trials (AMI). Acute revascularization with Percutaneous Coronary Intervention (PCI) is rapidly replacing fibrinolytic therapy as the gold standard treatment for Acute Myocardial Infarction (AMI). Some research has shown that patients with AMI who have an elevated blood creatinine content, including those who have undergone a successful primary PCI, have a higher risk of dying while hospitalized. Patients with mild renal impairment were also at risk for this negative outcome, which was shown in those with more severe renal dysfunction [7]. One study looked at the correlation amongst patients' admission creatinine levels & their risk of dying within a year after suffering an AMI; this study included 160 AMI patients (127 men & 33 women, with only an average age of 59-13). Following 12 h of an AMI, serum creatinine levels were checked. Mortality rates at one year have also risen significantly. Death rates in patients with AMI are dramatically higher after one year if their serum creatinine levels were even slightly raised at admission [8].

Role of Oral Nutrition in AMI Patients

One study found that individuals with STEMI had a day's nutritional condition when measured using the proper nutritional indices. A person's health, including their energy level, protein metabolism, and ability to fight off infections, can be gauged by looking at their nutritional status. Mortality from cardiovascular disease is reduced with increased potassium consumption, according to both prospective cohort studies and outcome trials. While serum blood potassium levels were not affected by either the low sodium diet or eplerenone, heart injury was averted in this trial. We used to eat a lot of potassium-rich foods, but that's changed in recent times [9,10]. In most industrialized countries, the average daily potassium intake is now approximately 70 mmol day 1, which is just one third of our ancestral intake, due to the rise in the consumption of processed food, from which potassium has been eliminated, and the decline in the consumption of fruits and vegetables. There is a substantial body of information demonstrating the health benefits of increasing one's potassium consumption. Multiple researches, both epidemiological and clinical, have demonstrated the beneficial effects of a high-potassium diet on lowering blood pressure in people with hypertension and in the general population. One study found that individuals with STEMI had a day's nutritional condition when measured using the proper nutritional indices. A person's health, including their energy level, protein metabolism, and ability to fight off infections, can be gauged by looking at their nutritional status. The construction and performance of the myocardium depend on adequate amounts of magnesium and potassium ions in the tissue. This study looked at the amounts of these components in the myocardium of males who had had sudden cardiac death to see if there was a link between a lack of these compounds and unexpected cardiac death in predisposed individuals.

Conclusion

A heart attack, or myocardial infarction, is a type of ischemic necrosis that develops when the coronary artery supplying the heart muscle becomes blocked. One of the five main symptoms of coronary heart disease is Myocardial Infarction (MI), along with unstable angina pectoris, heart failure, sudden death, and stable angina pectoris. MI risk factors include variations in lifestyle, behavior pattern, genetic vulnerability, and non-genetic factors including weight, blood pressure, and glucose levels [11]. A severe decline in potassium levels has been linked to elevated glucose levels in patients with acute coronary syndrome. A decrease in creatinine clearance or Glomerular Filtration Rate (GFR), regardless of cause, can significantly increase mortality in patients with acute coronary syndromes [12]. Lower levels of magnesium and potassium in myocardial tissue were seen in subjects in some research studies who died unexpectedly from ischemic heart disease.

References

1. Barkas F, Elisaf M. Serum potassium levels and mortality in acute myocardial infarction: Myth or fact? Angiology. 2018;69(8):657-9.
2. Packer M. Potential role of potassium as a determinant of morbidity and mortality in patients with systemic hypertension and congestive heart failure. Am J Cardiol. 1990;65(10):E45-E51.
3. Francis GS. Interaction of the sympathetic nervous system and electrolytes in congestive heart failure. Am J Cardiol. 1990;65(10):E24-E27.
4. Young DB. Role of potassium in preventive cardiovascular medicine. Springer Science & Business Media. 2012;8.
5. Schulman M, Narins RG. Hypokalemia and cardiovascular disease. Am J Cardiol. 1990;65(10):E4-E9.
6. Choi JS, Kim YA, Kim HY, Oak CY, Kang YU, Kim CS, et al. Relation of serum potassium level to long-term outcomes in patients with acute myocardial infarction. Am J Cardiol. 2014;113(8):1285-90.
7. Yamaguchi JI, Kasanuki H, Ishii Y, Yagi M, Ogawa H; HIJC Study Group, et al. Prognostic significance of serum creatinine concentration for in-hospital mortality in patients with acute myocardial infarction who underwent successful primary percutaneous coronary intervention (from the Heart Institute of Japan Acute Myocardial Infarction [HIJAMI] Registry). Am J Cardiol. 2004;93(12):1526-8.
8. Cakar MA, Gunduz H, Vatan MB, Kocayigit I, Akdemir R. The effect of admission creatinine levels on one-year mortality in acute myocardial infarction. Sci World J. 2012.
9. Maronde RF, Chan LS, Vlachakis N. Hypokalemia in thiazide-treated systemic hypertension. Am J Cardiol. 1986;58(2):A18-A21.
10. Pitt B, Collins AJ, Reaven N, Funk S, Bakris GL, Bushinsky DA. Effect of cardiovascular comorbidities on the mortality risk associated with serum potassium. Circulation. 2014;130(suppl 2):A13320-A13320.
11. Weaver CM. Potassium and health. Adv Nutr. 2013;4(3):368S-377S.
12. 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(16):2429-36.