CLINICAL STUDY OF SERUM SODIUM AND POTASSIUM LEVELS IN MYOCARDIAL INFARCTION PATIENTS

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ABSTRACT: *Lower levels of Sodium (Na^+) and Potassium (K^+) in Myocardial Infarction Patients cause complications with cardiac arrhythmias and sudden death. Worldwide, there are 3 million cardiac deaths per year due to myocardial infarction. This study was conducted to determine the levels of sodium and potassium in Myocardial Infarction patients. Seventy blood samples of Myocardial Infarction patients were collected from Gulab Devi and Ittefaq Hospital, Lahore, Pakistan, and thirty blood samples of control subjects were collected from different areas of Lahore, Pakistan. All samples were tested using the Beckman Coulter Synchron System to estimate the plasma levels of sodium and potassium. It was found that the levels of sodium and potassium were non-significantly lower than those of control subjects. Results were discussed in the light of previous reports of different populations. These results suggested that levels of sodium and potassium may be useful for the detection of Myocardial Infarction and its outcomes.*

Key Words: Electrolyte, Sodium, Potassium, Myocardial Infarction

INTRODUCTION

Acute myocardial infarction (AMI) is a dreadful complication of cardiovascular disease, which is one of the leading causes of morbidity and mortality across the world. The World Health Organisation (WHO) has declared cardiovascular disease a modern epidemic [1].

The incidence of myocardial infarction is increasing in developing countries [2]. The electrolyte levels play a crucial role in influencing the prognosis of MI patients. These electrolyte level changes have been reported to be used to monitor AMI. The major electrolytes in the body are potassium, sodium, magnesium, calcium and chloride [3]. Abnormalities of these electrolytes due to different causes can lead to significant cardiac life-threatening events [4]. Serum sodium and potassium play an important role in altering the prognosis of myocardial infarction patients [5]. A recent studies provide evidence for the effect of the sodium potassium ratio on myocardial infarction [6].

Hyponatremia and hypokalemia are indicators of acute myocardial infarction. Serum sodium and potassium levels are prognostic indicators. Therefore, estimation of sodium and potassium levels in acute MI patients can help assess their prognosis [7]. Some other studies have also shown a decrease in sodium level in patients with AMI compared with healthy people [5,7,8]. However, in a studies the levels of serum sodium (Na^+) were showing significant increase in acute coronary syndrome patients as compared to control group [9]. Higher sodium intake is associated with an increased risk of cardiovascular disease [10,11]. Some studies demonstrate that sodium may not have a strong relationship with cardiovascular mortality, and its association with cardiovascular mortality is less established [12]. Another studies show that higher potassium variability was associated with post-infarction outcomes but not sodium variability [13].

Potassium is one of the electrolytes that play an important role in cardiac disease, especially AMI [3]. Some previous studies also demonstrate a close association between hypokalemia and myocardial infarction [5,7,9]. Serum electrolyte changes in AMI have not been studied extensively, and there is a paucity of information [7].

All the above observations and results have led to the conclusion that estimation of sodium and potassium levels in acute MI patients can help assess their prognosis. We carried out a study to demonstrate the relation between Myocardial Infarction and variation in serum sodium and potassium levels.

MATERIALS AND METHODS

The present study was based on 70 Myocardial Infarction (MI) patients and 30 control subjects belonging to different age groups. The blood samples of MI patients were collected from Gulab Devi Chest Hospital and Ittefaq Hospital, Lahore, Pakistan. Patients with Myocardial Infarction were already diagnosed by cardiologist on the basis of ECG, Cardiac biomarker (Myoglobin, CKMB, Troponin T and I), echocardiography and angiography. Samples of control subjects were collected from the general population of Lahore, Pakistan. The electrolytes (sodium and potassium) of MI patients were compared with those of control subjects to find out the association between electrolytes and MI. Blood samples of MI patients and control subjects were drawn by the venipuncture method. Blood samples were allowed to clot for 20-25 minutes, and then, for the separation of serum, they were centrifuged at 4000 rotations per minute. The serum was separated out from the top of the clotted blood and then used. The electrolytes (sodium and potassium) were determined by the ion-selective electrodes. The Beckman Coulter AU system ISE module for sodium and potassium employs crown ether membrane electrodes for sodium and potassium.

The system reagents were used for the qualitative determination of sodium and potassium on Beckman Coulter AU clinical chemistry analysers. Mean ± S.D. of serum samples of control subjects and myocardial infarction were calculated. Statistical analysis of the data was carried out by employing the Student's test.

RESULTS

Thirty control subjects and seventy Myocardial infarction patients were involved in the study. The mean serum sodium level of control subjects and myocardial infarction patients was studied. Serum sodium level in myocardial infarction

patients was lower than that of control subjects. In control subjects, these values were 140.2 ± 0.57 mmol/l, while in myocardial infarction patients, these values were 139.4 ± 0.52 mmol/l. The observed difference was non-significantly lower (P>0.05) in myocardial infarction patients (Table 1).

The mean serum potassium level of control subjects and myocardial infarction patients was also studied. Serum potassium level in myocardial infarction patients was lower than that of control subjects. In control subjects, these values were 4.32 ± 0.14 mmol/l, whereas in myocardial infarction patients, these values were 4.27 ± 0.06 mmol/l. The observed difference was non-significantly lower (P>0.05) in myocardial infarction patients (Table 1).

Table 1: Mean Serum Sodium and Potassium Levels in Control Subjects and Myocardial Infarction Patients.

	No.	Serum Sodium (mmol/l) (Mean ± SEM)	Serum Potassium (mmol/l) (Mean ± SEM)	P value
Control Subjects	30	140.2 ± 0.57	4.32 ± 0.14	> 0.05
Myocardial Infarction patients	70	139.4 ± 0.52	4.27 ± 0.06	

DISCUSSION

The present study was conducted to check the levels of sodium and potassium in Myocardial Infarction patients. Our results showed that serum sodium level in Myocardial Infarction patients was non-significantly lower compared to control subjects. These results are comparable with some previous studies. Many researchers in their studies described lower levels of sodium in Myocardial Infarction patients compared to control subjects in India [7], in Palestine [8], in Udairpur, Rajasthan, India [14], in Gujrat, India [15] and in Satlur, Dharwad, Karnataka [16].

Sodium is the most abundant extracellular cation that helps to balance fluid levels in the body and facilitates neuromuscular functioning. Sodium imbalance has been recorded in the early phase of AMI in some studies [17]. Many patients with heart failure have decreased sodium levels due to neurohormonal mechanisms [18].

Hyponatremia is relatively common in patients with acute MI. Some studies have shown that hyponatremia is associated with poor outcomes in patients with STEMI and NSTEMI, and the risk of mortality increases with the severity of hyponatremia [4,19].

In AMI, non-osmotic release of vasopressin may occur due to acute development of left ventricular dysfunction due to pain, nausea and major stress, the most common mechanisms of hyponatremia in adults, or may be due to the administration of analgesics or diuretics. This could result in low sodium levels in the blood [20,21]. In this setting, vasopressin level increases concomitantly with activation of other neurohormones such as renin and norepinephrine [22]. Moreover, the renal effect of vasopressin is enhanced in heart failure, as the vasopressin-regulated water in the collecting duct is upregulated [23].

The relation between the development of hyponatremia and time of myocardial injury seems to be important, because as the time from acute injury increased, the prognostic relation with hyponatremia became less significant [24]. MI patients with hyponatremia were also found to have lower ejection fraction when compared to non-hyponatremic patients [25]. Hyponatremia could probably be attributed to the non-osmotic secretion of vasopressin, impairing the water excretion, causing dilutional hyponatremia [24]. Vasopressin is known to regulate vascular tone and cardiac contraction and may adversely affect cardiac hemodynamics and myocardial remodelling [4].

The sarcolemma is impermeable to sodium in the resting state. It has a potassium ATPase pump that plays an important role in establishing the resting potential. This pump exports sodium from the cell and imports potassium inside the cell against its concentration gradient. Thus, intracellular potassium is relatively high, and sodium is low, and extracellular sodium is high and potassium is low. There are four phases of action potential dependent on sodium, potassium and calcium. Serum electrolyte imbalances after an episode of acute myocardial infarction are common. But the clinical importance of these imbalances in both ST-segment elevation myocardial infarction and non-ST-segment myocardial infarction has not been fully understood. These electrolytes play an important role in altering the prognosis of such myocardial infarction patients [4,19].

The result of our tests revealed that serum potassium levels in Myocardial Infarction patients were significantly lower compared to control subjects. These results are in line with some previous studies. Many researchers in their studies described lower level of potassium in Myocardial Infarction patients as compare to control subjects in India [7], in Palestine [8], in Sri Dev raj Urs medical college, Kolar [2], in Udairpur, Rajasthan, India [14], in New York [27], in Gujrat, India [15] and in Satlur, Dharwad, Kamataka [16].

Potassium is the main component of cellular fluid. This positive electrolyte helps to regulate neuromuscular function and osmotic pressure. Approximately 98% of this electrolyte is intracellular. Its main regulation is by the renal excretion and shift between the intracellular and extracellular compartments. Potassium is one of the electrolytes that play an important role in cardiac disease, especially AMI [28].

The prevalence and role of hypokalemia in myocardial infarction have been under investigation for a long time [3,29]. Hypokalemia in patients with AMI is thought to predict increased in-hospital morbidity, particularly arrhythmias and mortality [27]. Several studies have shown an association between hypokalemia and increased occurrence of cardiac arrhythmia in AMI patients [28]. Hypokalemia was even found to be associated with larger infarcts [27]. Hyperkalemia is also associated with increased mortality and should be avoided. The total body potassium levels in the body are 3500 mmol, out of which 98% is intracellular. Its main regulation is by the renal excretion and shift between the intracellular and extracellular

compartments. Mainly, the sodium-potassium ATPase pump is responsible for preserving the intracellular potassium. Aldosterone and vasopressin stimulate potassium secretion by up-regulating the luminal sodium potassium ATPase pump and opening the luminal sodium and potassium channels [30]. The sudden cardiac death after MI is mainly due to alterations in the environment at the level of myocytes and Purkinje fibres that are mainly regulated by the electrolyte imbalances and autonomic nervous system activity [17].

Hypokalemia observed in our study may be due to stress-induced Catecholamine, which increases potassium uptake by the cells [31]. This is an acute stress effect and is due to the shift of potassium from extracellular to intracellular space, and is a result of stimulation of beta-2 adrenoceptor agonists linked to sodium potassium ATPase [32].

Hypokalemia may result in membrane hyperpolarisation with subsequent insufficient muscle contraction. Symptoms of hypokalemia include weakness, respiratory compromise, and paralysis. Electrocardiogram changes can occur, including T flattening, T wave inversion, ST segment depression, and the presence of U waves. The most serious complications associated with hypokalemia are cardiac arrhythmias and sudden death [33-35].

The association of hyponatremia or hypokalemia with early presentation in acute MI may alert the clinician about the acuteness and severity of the patient's illness, since these attributes constitute substrates for the emergence of complications in acute MI patients.

Conclusion:

Levels of sodium and potassium may be useful for the detection of Myocardial Infarction and its outcomes. As a consequence, it must be properly observed in all patients suffering from Myocardial Infarction. However, in order to analyse the severity of the disease, further studies on other parameters.

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