

The Prognostic value of Hyponatremia and Hypokalemia in Myocardial Infarction Patients

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Abstract - Syria is one of the countries with a high risk of ischemic heart disease. It is one of the most causes of morbidity and mortality worldwide. Myocardial infarction does not mean simply occlusion of an epicardial artery. Rather, it is a systemic, metabolic, and neurohormonal process that affects and is affected by other systems, including blood ions. In this research, we will study the changes in sodium and potassium in patients with myocardial infarction and their impact on prognosis. This study included 100 patients. Patients were divided into two groups, a high-risk group and a low-risk group based on the GRACE score, and successive laboratory tests were made for both sodium and potassium from admission to discharge. We found the incidence of hyponatremia was 54% and hypokalemia was 64%. We found that early hyponatremia was associated with short-term prognosis (p -value = 0.001), and hyponatremia at discharge was associated with long-term prognosis (p -value = 0.003). We found that early hypokalemia was associated with short-term prognosis (p -value = 0.001). We found a significant association between the development of cardiac shock and hyponatremia (p -value = 0.001, OR = 40.1), and significant association between the development of arrhythmias and hypokalemia (p -value = 0.002, OR = 41.9). We also found that the return of values to normal ranges at discharge indicates improvement and response to treatment.

Keywords — Myocardial infarction, hyponatremia, hypokalemia.

I. Introduction

At the beginning of the last century, myocardial infarction meant inevitable death, and during these long years, there was a great development in methods of diagnosis and treatment. The death rate that was close to 100% turned to about 10%. In the fifties, treatment was limited to absolute and prolonged rest at home, so there was nothing that could be done to open the occluded artery. Then, the first intensive care unit appeared in Britain to monitor patients in the hospital instead of home to perform CPR in the event of cardiac arrest or life-threatening arrhythmias. Then antiplatelet agents and beta-blockers appeared, then thrombolysis. Until we achieve opening the artery with catheterization and implantation of cardiac stents ⁽¹⁾. The myocardial infarction is part of a spectrum called acute coronary syndrome. The infarction is classified into two

types, the first with ST-segment elevation, which is referred to as STEMI, and the second without ST-segment elevation, which is referred to as NSTEMI. There were 15.9 million infarcts recorded worldwide in 2015 ⁽²⁾. There is no doubt that myocardial infarction is one of the main causes of death and morbidity. It is estimated that 50% of patients die before reaching the hospital ⁽³⁾. The mortality rate during the first year is estimated at 10% ⁽²⁾. And according to the recent guidelines of the European Society of Cardiology, the Syrian Arab Republic was classified in 2019 among the countries of high risk for cardiovascular disease ⁽⁴⁾. The disease prognosis varies greatly from patient to patient, so it is important to determine the severity of the patient upon arrival at the hospital, so-called "risk stratification". Currently, the two most important scores for prognosis are GRACE and TIMI, both of which contain neither sodium nor potassium.

The subject of our research is the study of the changes of sodium and potassium in these patients and their role in diagnosing the disease and determining the prognosis, based on their ease of calibration and availability.

It is known that the balance of electrolytes within the normal range has an important role in maintaining the integrity of the cardiovascular system, and this balance is essential for the transmission of nerve impulses, muscle contraction-relaxation, and acid-base balance ⁽⁵⁾.

Sodium is the main positive ion in the extracellular fluid and is mainly responsible for neuromuscular transmission, excitation-contraction coupling, and balance between intra- and extra-cellular fluids ⁽⁴⁾. Potassium plays an important role in maintaining the stability of the electrical cardiac system. Hypokalemia lowers the threshold of ventricular arrhythmias ⁽⁶⁾.

In myocardial infarction, the cardiac output decreases, the arterial pressure decreases. Then the receptors in the carotid body and renal glomerulus activate the multiple sodium-sparing systems, especially the renin-angiotensin-aldosterone system, sympathetic nervous system, and the arginine-vasopressin system ⁽²⁾. The intensity of activation of neurohormonal hormones is proportional to impaired cardiac function. The worse cardiac function, the greater the neurohormonal activation. The result is the reabsorption of water and sodium from the collecting tubules in the kidney in an attempt to restore normal arterial pressure and cardiac output. However, the reabsorption of water is greater than the reabsorption of sodium, and hyponatremia occurs. Both angiotensin II and vasopressin play a major role in this process ⁽⁶⁾. In addition, the vasoconstriction occurring in the



renal afferent arterioles as a result of noradrenaline and angiotensin II leads to a decrease in the rate of glomerular filtration, meaning a decrease in water excretion, and this contributes to the occurrence of hyponatremia ⁽⁴⁾. Likewise, the hypoxia that occurs in myocardial infarction leads to the destruction of the permeability of the sarcolemma, and this leads to the entry of sodium ions into the cells in an unselected and uncontrolled way, which contributes to the occurrence of hyponatremia ⁽⁷⁾.

Hypokalemia occurs either by the renal excretion mechanism or by the intracellular shift mechanism. Striated muscles and the liver are the two most important stores of potassium in the body. The high levels of adrenaline that occur during myocardial infarction lead to stimulation of B2 receptors, thus activating the Na/K ATPase pump which leads to the entry of potassium into cells, especially in the striated muscles and liver, and this leads to hypokalemia ⁽⁵⁾. There is an inverse correlation between the level of adrenaline and the level of potassium in the serum, so the higher the adrenaline, the more activated the pump and thus the more hypokalemic. That clarifies the role of beta-blockers in preventing hypokalemia in patients with myocardial infarction and their role in preventing arrhythmias ⁽⁶⁾.

The secretion of corticosteroids is increased in response to the myocardial infarction that leads to the activation of the ENaC sodium channels in the renal tubules that reabsorb the sodium and excrete the potassium ⁽⁸⁾. Respiratory alkalosis occurs in infarction patients as a result of hyperventilation due to anxiety, fear, and pain, or due to hypoxia. And when the serum pH rises, the H/K pump in the collecting tubules is activated, which leads potassium to shift into cells in exchange for removing hydrogen ions to correct the alkalosis.

II. Objectives of Study

The perception of electrolyte changes in acute myocardial infarction is derived from physiological theories, and there are no adequate studies available on the prognostic value of these changes. Our goal of this research is to study the sodium and potassium changes that occur in acute myocardial infarction and their role in determining the prognosis of myocardial infarction.

III. Materials and Methods

Study Population

The sample of the study included 100 patients, who were admitted to ICU with a diagnosis of myocardial infarction at Tishreen University Hospital, from March 2019 until December 2019. All patients agreed to informed consent to include their data in the research.

Study Design

We perform a prognostic prospective study. A detailed history was taken from the patient, including the onset of chest pain, and cardiac risk factors (smoking, hypertension, and diabetes). Electrocardiograms, titration of cardiac enzymes, and determination of myocardial infarction pattern

were done. Then the patients were divided into two groups:

- 1) Group A, which are patients whose GRACE score is greater or equal to 128, and we will refer them as high-risk patients.
- 2) Group B are patients whose GRACE score is less than 128, and we will refer them as low-risk patients.

We took a blood sample during the first 12 hours of the onset of chest pain, a sample on the third day, and a sample on the fifth day. Then we performed a laboratory test of the sodium and potassium of each sample, on the blood gas device located in the intensive care unit. We followed the patients during the hospitalization period and monitored the development of complications (arrhythmias, mechanical complications, cardiac shock).

Inclusion Criteria

STEMI and NSTEMI myocardial infarction patients as defined by the European Society of Cardiology.

Exclusion criteria

- 1) Patients with renal insufficiency stage 3, 4, and 5.
- 2) Patients with chronic diarrhea and vomiting.
- 3) Patients on diuretics.
- 4) Patients on ACE inhibitors and angiotensin receptor blockers before the infarction.
- 5) Patients who died during the hospitalization from a non-cardiac cause.
- 6) Patients who developed a severe sodium or potassium disorder that required therapeutic intervention.

Data Analysis

The data were analyzed using the Statistical Package of Social Sciences (SPSS) software, version 20. The values were expressed as mean and standard deviation. The independent T-test was performed to compare the mean values between the cases and the controls. A significant result means that the p-value test is < 0.05. The confidence intervals (CI) were reported as 95%.

IV. Results

The sample included 100 patients (57 males, 43 females) of myocardial infarction patients admitted to ICU at Tishreen University Hospital. The number of patients with STEMI was 77 patients and 23 patients with NSTEMI. The number of high-risk patients was 56, and 44 low-risk patients. The patients' ages ranged from 27 to 85 years. The mean average age is 60.1 ± 12.6 . For ease of expression, we will denote the sodium value in the first 12 hours as Na1, the third day as Na2, and the fifth day as Na3. The percentage of patients with hyponatremia on admission was 47%, with the mean value of 135.54 ± 4.9 . On the third day, 54%, and the mean value was 133.59 ± 5.7 . On the fifth day, 20% and the mean value was 135.73 ± 5.4 . When studying the distribution of sodium values in both risk groups, it was found that the mean Na1 sodium value in group A was 132.86 ± 4.4 , while in group B it was 138.97 ± 2.9 , with statistical significance p-

value = 0.004. The mean Na² sodium value for group A was 130.52 ± 5.2 , while for group B it was 137.52 ± 3.9 , with statistical significance p-value = 0.001. The mean value of Na³ in group A was 133.57 ± 5.9 while in group B it was 138.47 ± 3 , with statistical significance p-value = 0.003. We found that 72.1% of patients who developed arrhythmias had hyponatremia (p-value = 0.002, OR = 7.5). And we found that 100% of patients who developed cardiogenic shock had hyponatremia (p-value = 0.001, OR = 40.1). And we found that 88.2% of patients who died had hyponatremia (p-value = 0.001, OR = 8.4). Also, for ease of expression, we will denote the value of potassium in the first 12 hours as K1, the third day as K2, and the fifth day as K3. The percentage of patients with hypokalemia on admission was 26%, and the mean value was 4.2 ± 0.8 . On the third day, 64%, the mean value was 3.4 ± 0.4 . On the fifth day, 16%, and the mean value was 3.7 ± 0.4 . When studying the distribution of potassium values in both risk groups, it was found that the mean value of K1 in group A was 4.18 ± 0.96 , while in group B it was 4.28 ± 0.66 with insignificant statistical p-value = 0.548. The mean value for K2 in group A was 3.31 ± 0.38 , while for group B it was 3.6 ± 0.35 , with statistically significant p-value = 0.001. The mean value for K3 in group A was 3.64 ± 0.5 , while for group B it was 3.85 ± 0.37 , with insignificant statistical p-value = 0.030. We found that 90.1% of patients who developed arrhythmias had hypokalemia (p-value = 0.001, OR = 41.9). And we found that 92.3% of patients who developed cardiogenic shock had hypokalemia (p-value = 0.001, OR = 11.3). And we found that 88.2% of the patients who died had hypokalemia (p-value = 0.004, OR = 5.7).

V. Discussion

Hyponatremia is common in patients with myocardial infarction, and it is more common than in heart failure patients which are estimated at 25% of them ⁽⁹⁾. We found that hyponatremia is a hemodynamic process that changes from day to day, and this change is related to disease progression. We noticed that when sodium levels continued to decline beyond the third day and did not start to rise, had accompanied by a prolonged hospitalization (p-value = 0.001). Hyponatremia is related with mortality during the hospitalization (p-value = 0.001, OR = 8.4). The lower the sodium value, the worse the prognosis.

We found a significant difference in the distribution of hyponatremia between the high-risk group and the low-risk group (p-value = 0.001). We also found an inverse correlation between the sodium value and the GRACE score in the same patient. the higher the patient's GRACE score value, the lower the sodium value. And when studying the sodium value at discharge, we found that the sodium values of 20% of patients did not return normal level at discharge, and 85.5% of those did not have successful reperfusion criteria for either thrombolysis or cardiac catheterization.

Hypokalemia is also common in patients with infarction ⁽¹⁰⁾. Potassium values are also a hemodynamic process and their changes are subject to disease progression. In our study, we

found a strong correlation between hypokalemia and arrhythmias (p-value = 0.001, OR = 41.9). Hypokalemia is associated with mortality, specifically dip potassium value (p-value = 0.004, OR = 5.7). We found a significant difference in the distribution of hypokalemia between the high-risk group and the low-risk group on the third day only (p-value = 0.001). We also found an inverse correlation between the potassium value and the GRACE score in the same patient. The greater the patient's GRACE score value, the lower the sodium value was. We noticed that the best prognosis patients are those who maintained normal potassium values (p-value = 0.004).

VI. Limitations

We only followed patients during the hospitalization period, monitored the development of complications, and recorded deaths during this period only. We relied on the GRACE score to predict long-term prognosis without directly studying it.

VII. Conclusion

We conclude that hyponatremia and hypokalemia are markers of myocardial infarction. We also derive the correlation of hyponatremia with both short-term and long-term prognosis. The dip sodium value is related to short-term prognosis, and the discharge value is related to long-term prognosis. We also conclude that hypokalemia, either upon admission or upon discharge, is related to short-term prognosis and predicts the development of life-threatening arrhythmias.

We conclude the importance of periodic monitoring of the values of sodium and potassium, as their continued decrease indicates the deterioration of the disease and its return to normal indicates the improvement and the effectiveness of the treatment.

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