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Changes in biochemical signs and electrolyte concentration in acute myocardial infarction

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Acute myocardial infarction (AMI) remains a leading cause of morbidity and mortality worldwide. Electrolyte imbalances, particularly alterations in sodium, potassium, calcium, magnesium, and chloride, play a significant role in cardiac electrophysiology and may influence the clinical course of AMI. To assess the prevalence and patterns of electrolyte disturbances in patients with AMI and to evaluate their association with comorbid hypertension and diabetes mellitus, a case–control study was conducted at the Heart Hospital in Nasiriyah, Southern Iraq, between October 2021 and March 2022. The study enrolled 100 participants, including 51 patients with recent-onset AMI and 49 healthy controls. AMI diagnosis was confirmed according to ESC, ACC, AHA, and WHF guidelines. After an overnight fast, venous blood samples were collected and analyzed for serum sodium, potassium, chloride, calcium, and magnesium using a COBAS C411 analyzer, with verification by spectrophotometry. Among AMI patients (n = 51), hyponatremia was detected in 14 cases (27.5%), hypokalemia in 12 cases (23.5%), and hypocalcemia in 25 cases (49.0%). Electrolyte disturbances were more frequent among patients with hypertension (n = 46) and diabetes mellitus (n = 31) compared with those without these comorbidities. Potassium imbalance emerged as the most significant alteration, present in nearly two-thirds of the cases. Deficiencies in calcium and magnesium showed a strong association with sodium–potassium imbalance. Electrolyte disturbances, particularly hyponatremia and hypokalemia, are common in AMI and may contribute to adverse clinical outcomes. Early detection and correction of these abnormalities are essential for improving prognosis in AMI patients.

Keywords: electrolytes; myocardial infarction; hyponatremia; hypokalemia; hypocalcemia; hypomagnesiumia; hypochloremia.

Introduction

Acute myocardial infarction (AMI) is a very dangerous heart problem that is still one of the main reasons people get sick and die around the world. The pathogenesis of AMI includes the abrupt cessation of coronary blood flow, leading to myocardial ischemia and necrosis. This activates a series of metabolic and biochemical changes that greatly affect cardiac function, of which electrolyte imbalance is a crucial factor influencing the development and prognosis of AMI (Salari et al., 2023).

The process by which necrosis (cell or tissue death) occurs is defined as myocardial infarction, and it is a complication of ischemia that leads to the loss of blood supply (Buja et al., 2023). In addition, AMI is the most common type of cardiovascular disease, often due to coronary artery blockage. When the coronary circulation doesn't bring enough oxygen and nutrients to the heart, cardiogenic shock can happen. This can cause tissue breakdown, either partially or completely (Heusch, 2020; Dong et al., 2023). AMI is characterized by clear symptoms such as pain radiating to the arm and shoulder, nausea, dizziness, and severe chest discomfort lasting for hours (Faraj, 2015; Kucia et al., 2022). Changes in cardiac electrical impulses may also occur irregularly, leading to inadequate pumping of blood and resulting in organ dysfunction and myocardial damage (Näbauer et al., 1993; Albus et al., 2022).

Electrolytes such as sodium, potassium, calcium, magnesium, and chloride play critical roles in myocardial excitability, conduction properties, and contractility. Even small deviations from normal plasma levels can alter the transmembrane potential of cardiac cells, inducing arrhythmias, contractile dysfunction, and increased mortality (Rafaqat et al., 2022; Yadav et al., 2024). For instance, hypokalemia and hyponatremia are strongly associated with poor prognosis in AMI patients, while hypocalcemia and hypomagnesemia can exacerbate myocardial instability and disrupt essential enzymatic processes (Batta et al., 2022; Crintea et al., 2025).

Electrolytes are distributed between two main fluid compartments: extracellular (e.g., magnesium, calcium, sodium) and intracellular (e.g., potassium, chloride), both of which are essential for maintaining myocardial membrane electrophysiological properties (Huang et al., 2023). AMI is also associated with major biochemical alterations representing tissue injury and systemic stress, including electrolyte derangements. These changes are reflected in elevated cardiac biomarkers such as troponins and creatine kinase-MB, and in metabolic variables like blood glucose and lipids, which interact with electrolyte imbalances to worsen patient outcomes (Carrizales et al., 2021; Saetang et al., 2025).

Electrolytes influence cardiac electrophysiology by modulating ion conduction across the myocardial membrane. Potassium, one of the most important cations, works in conjunction with calcium and sodium to affect the rate and direction of ion movement. Hypokalemia (<3.5 mmol/L) and hyperkalemia (>5.5 mmol/L) are particularly linked to arrhythmias (Xianghua et al., 2010). Calcium and sodium interactions help regulate polarization changes related to potassium levels (Solini et al., 2006). These processes are mediated by mechanisms such as the Na^+-K^+ ATPase pump and the opening of calcium channels during action potentials, facilitating ionic fluxes essential for cardiac contraction (Goldberg et al., 2004). Accumulation of intracellular calcium also plays a key role in apoptosis initiation (Szerencsei et al., 2013).

Subtle changes in extracellular potassium profoundly impact cardiovascular and neuromuscular function, while normal calcium levels protect the myocardium and prevent coronary artery spasm. Hyponatremia, defined as serum sodium <133 mEq/L, is relatively common in AMI patients (Bueno-Orovio et al., 2014; Teymouri et al., 2022). Chloride, the primary anion, works with sodium to maintain cellular osmotic balance, pressure, and pH. Disturbances arise from reduced cardiac output, impaired renal excretion, and neurohormonal activation affecting both cardiovascular and electrolyte balance. Magnesium, an abundant element essential for over 300 enzymatic reactions, plays a protective role via beta-adrenergic receptor interaction

and helps stabilize ionic gradients (Rasmussen, 2002). The present study aimed to investigate alterations in serum electrolyte levels among patients with acute myocardial infarction (AMI), particularly those with comorbid hypertension and diabetes.

Materials and methods

The study was approved by the Human Ethics Committee of Department of Chemistry, College of Education, University of Sumer, Al-Refaee, Thi-Qar, Iraq, Everyone who took part in the study was told about it and asked to sign a consent form. Each patient was also guaranteed that his information would be kept private.

This study was conducted by Sumer University at the Heart Hospital in the city of Nasiriya ,Southern Iraq, from October 2021 to March 2022 on 51 patients with AMI and 49 healthy patients carefully selected. In both the groups together , 61 male patients and 39 females, aged 35–75 years (57.82 ± 1.94, mean ± SD) completed the study. The AMI diagnosis was confirmed according to the guidelines of the European Society of Cardiology (ESC), the American College of Cardiology (ACC), the American Heart Association (AHA), and the World Heart Federation (WHF). The people who had heart attacks were all from the same hospital. The study's goal was clear: to find out how changes in electrolytes affect people who have had an AMI, taking into account their age, gender, smoking habits, high blood pressure, and diabetes.

After obtaining ethical approval and documentation from the hospital under reference number 35674, blood samples were collected from all participants following standardized procedures to minimize pre-analytical errors. Patients were instructed to fast for at least 13 hours overnight prior to sample collection to prevent postprandial variations in biochemical parameters and avoid the influence of recent food intake or medications on serum electrolyte levels. On the day of collection, each participant was seated comfortably in a dedicated phlebotomy area to reduce stress-induced changes. Venous blood samples (5 mL) were drawn from the antecubital vein using sterile disposable syringes. A tourniquet was applied briefly to facilitate venous access but released before venipuncture to prevent hemoconcentration and artificial elevation of biochemical values.

The collected blood was distributed into two tubes: a GEL tube containing clot activator for serum separation, used to determine sodium, potassium, chloride, calcium, and magnesium levels, and an EDTA tube containing anticoagulant for additional analyses if required. Blood in the GEL tubes was allowed to clot for 15–30 minutes at room temperature and then centrifuged using a TDZ5-WS centrifuge at 3000 rpm for 10 minutes (Max RCF: 4745, time range 0–99). Serum separation was completed within 2 hours to maintain sample integrity.

Electrolyte concentrations (Na^+, K^+, Cl^-, Ca^{2+}, Mg^{2+}) were determined using an automated chemistry analyzer (COBAS C411, Roche Diagnostics International Ltd., Rotkreuz, Switzerland) according to the manufacturer's protocol. Internal quality control was ensured through the use of calibrators and commercial control sera, in compliance with Clinical and Laboratory Standards Institute (CLSI) guidelines. To verify accuracy, a subset of samples underwent manual confirmation using spectrophotometry with a Hitachi Double-Beam Spectrophotometer UH5300 (Hitachi High-Tech, Tokyo, Japan). Ion-selective electrode (ISE) principles were used for sodium and potassium, calcium was assessed via the colorimetric Arsenazo III method, magnesium by the xylidyl blue method, and chloride using the mercuric thiocyanate technique. All samples were processed in duplicate, and results were accepted only when duplicate values showed a coefficient of variation below 5%.

SPSSv10 software was used to collect and analyze data. Mean ± SD (standard deviation) was used to pool the results and compare them with healthy cases. Statistical tools such as Z-value (Guide to Statistical Methods for Use in Health and Nutrition) were used, which are important in finding the significance of the difference in mean values between groups. An independent t-test was used to compare quantitative data across groups. Pearson's correlation coefficient was used to determine the correlation between quantitative data. A $P < 0.05$ was considered significant.

Results

The results of the demographic analysis showed that the mean age in both groups was close (57.82 ± 1.94 years), with no significant differences between the patients and the control group (P = 0.990). Examining the distribution of ages within the groups, it was found that the most represented group among patients with infarction was the 46–60 year age group (39.2%), followed by the >60 year age group (33.3%), while the 35–45 year age group was the least common (27.4%), with no significant statistical difference (P = 0.820). As for gender, the results showed that males constituted the largest proportion in both groups (60.8% in the patient group versus 61.2% in the control group), while the proportion of females was (39.2%) among patients versus (38.8%) among the control group, with no significant differences (P = 0.910). These results indicate that mean age and gender were not associated with statistically significant differences between infarction patients and the control group as shown in Table 1.

Table 1
Socio-demographic characteristics of study participants

Variable	AMI cases (n = 51)	Controls (n = 49)	P-value
Age, years	57.82 ± 1.94	57.82 ± 1.94	0.990
Age groups			
35–45	14 (27.4%)	12 (24.5%)	0.820
46–60	20 (39.2%)	16 (32.6%)	
>60	17 (33.3%)	21 (42.8%)	
Gender			
Male	31 (60.8%)	30 (61.2%)	0.910
Female	20 (39.2%)	19 (38.8%)	

The serum electrolyte levels in AMI patients with and without hypertension are shown in Table 2 .The amounts of sodium (121.0 ± 5.2 vs. 136.0 ± 8.0, P = 0.370), potassium (2.94 ± 1.06 vs. 3.40 ± 0.93, P = 0.080), chloride (88.0 ± 8.6 vs. 89.0 ± 11.8, P = 0.830), and calcium (2.11 ± 0.34 vs. 2.16 ± 0.38, P = 0.980) were not statistically different between the two groups. The magnesium levels were statistically different between the high blood pressure AMI group (0.81 ± 0.16) and the normal blood pressure AMI group (0.82 ± 0.18, P = 0.017).

Table 2
Electrolyte levels in AMI cases with and without hypertension

Serum, mmol/L	Case (mean ± SD)		P-value
	AMI with hypertension (n = 46)	AMI with non-hypertension (n = 5)	
Sodium	121.0 ± 5.2	136.0 ± 8.0	0.370
Potassium	2.94 ± 1.06	3.40 ± 0.93	0.080
Chloride	88.0 ± 8.6	89.0 ± 11.8	0.830
Calcium	2.11 ± 0.34	2.16 ± 0.38	0.980
Magnesium	0.81 ± 0.16	0.82 ± 0.18	0.017

The results regarding serum electrolyte levels in the control groups with hypertension (N = 21) and non-hypertension (N = 28) are shown in Table 3 .These showed statistically significant differences in sodium levels, where the value was 130.0 ± 9.4 in the group with hypertension compared to 133.0 ± 9.0 in the non-hypertensive group ($P < 0.001$). Potassium levels also showed a statistically significant difference, where it was 3.92 ± 0.97 in the group with hypertension compared to 4.30 ± 0.75 in the non-hypertensive group (P = 0.026). As for chloride levels, there were no statistically significant differences (79.0 ± 14.9 vs. 93.0 ± 10.3, P = 0.220). As for calcium levels, a statistically significant difference was recorded, as the result was 2.09 ± 0.34 in the hypertensive group compared to 2.45 ± 0.47 in the non-hypertensive group (P = 0.050). Magnesium levels did not show a statistically significant difference (0.83 ± 0.13 vs. 0.87 ± 0.17, P = 0.260) as shown in Table 3.

The serum electrolyte levels in AMI patients with acute myocardial infarction (AMI) with and without diabetes (N = 31) are shown in Table 4 .These showed statistically significant differences in sodium levels, which were 125.0 ± 7.2 in the group with diabetes compared to 132.0 ± 10.3 in the group without diabetes (P = 0.012). Differences in calcium levels, which were 2.11 ± 0.50 in the group with diabetes compared to 2.17 ± 0.34 in the group without diabetes, were statistically significant (P = 0.040). However, potassium (3.60 ± 1.05 vs. 4.02 ± 1.04, P = 0.740), chloride (78.0 ± 11.3 vs. 89.0 ± 10.6, P = 0.940) and magnesium (0.83 ± 0.16 vs. 0.84 ± 0.13, P = 0.190) levels did not record any statistically significant differences as shown in Table 4.

Table 3
Electrolyte levels in control group with and without hypertension

Serum, mmol/L	Control group (mean ± SD)		P-value
	CG with hypertension (n = 21)	CG with non-hypertension (n = 28)	
Sodium	130.0 ± 9.4	133.0 ± 9.0	<0.001
Potassium	3.92 ± 0.97	4.30 ± 0.75	0.026
Chloride	79.0 ± 14.9	93.0 ± 10.3	0.220
Calcium	2.09 ± 0.34	2.45 ± 0.47	0.050
Magnesium	0.83 ± 0.13	0.87 ± 0.17	0.260

Table 4
Electrolyte levels in AMI case group with and without diabetes

Serum, mmol/L	Case (mean ± SD)		P-value
	AMI with diabetes (n = 31)	AMI without diabetes (n = 31)	
Sodium	125.0 ± 7.2	132.0 ± 10.3	0.012
Potassium	3.60 ± 1.05	4.02 ± 1.04	0.740
Chloride	78.0 ± 11.3	89.0 ± 10.6	0.940
Calcium	2.11 ± 0.5	2.17 ± 0.34	0.040
Magnesium	0.83 ± 0.16	0.84 ± 0.13	0.190

The results regarding serum electrolyte levels in the control group with diabetes (n = 14) and without diabetes (n = 14) are shown in Table 5. These showed no statistically significant differences in sodium (120.0 ± 5.8 vs. 138.0 ± 10.0, P = 0.822), potassium (3.00 ± 1.05 vs. 4.32 ± 0.67, P = 0.810) and chloride (80.9 ± 11.7 vs. 90.3 ± 13.2, P = 0.640) levels. However, a statistically significant difference was observed in calcium levels, with the result being (2.02 ± 0.35) in the group with diabetes compared to (2.55 ± 0.51) in the group without diabetes (P = 0.040). However, magnesium levels (0.80 ± 0.25 vs. 0.91 ± 0.05, P = 0.250) did not record any statistically significant differences as shown in Table 5.

Table 5
Electrolyte levels in control group with and without diabetes

Serum, mmol/L	Control (mean ± SD)		P-value
	CG with diabetic (n = 14)	CG without diabetic (n = 14)	
Sodium	120.0 ± 5.8	138.0 ± 10.0	0.822
Potassium	3.00 ± 1.05	4.32 ± 0.67	0.810
Chloride	80.9 ± 11.7	90.3 ± 13.2	0.640
Calcium	2.02 ± 0.35	2.55 ± 0.51	0.040
Magnesium	0.80 ± 0.25	0.91 ± 0.05	0.250

Pearson correlation analysis results showed a strong negative relationship between sodium levels and hypertension (r = –0.62, P = 0.001), as well as with diabetes (r = –0.58, P = 0.002), indicating that low sodium levels are significantly associated with the presence of these conditions. Potassium levels also showed a moderate negative relationship with hypertension (r = –0.36, P = 0.021), while there was no significant relationship with diabetes (P = 0.318). Chloride showed a weak and insignificant relationship with both hypertension (P = 0.184) and diabetes (P = 0.410). Calcium showed a very weak relationship with hypertension (r = –0.04, P = 0.732), while there was a moderate, but significantly negative relationship with diabetes (r = –0.28, P = 0.041). Finally, magnesium showed a moderate negative association with hypertension (r = –0.42, P = 0.017), while its association with diabetes was weak and insignificant (P = 0.292). These results indicate that sodium and magnesium are the most affected by pathological conditions, especially hypertension, reflecting their importance in the physiological changes accompanying myocardial infarction as shown in Table 6.

Table 6
Correlation between serum electrolytes and clinical conditions in AMI patients

Electrolyte	Hypertension (r)	P-value	Diabetes (r)	P-value
Sodium	–0.62	0.001	–0.58	0.002
Potassium	–0.36	0.021	–0.12	0.318
Chloride	–0.18	0.184	–0.09	0.410
Calcium	–0.04	0.732	–0.28	0.041
Magnesium	–0.42	0.017	–0.15	0.292

Discussion

In the current study 100 subjects, including control, were investigated to determine the lowest and highest value for the electrolytes potassium, sodium, chloride, calcium and magnesium in acute myocardial infarction with hypertension and diabetes. Electrolyte imbalances are common in MI compared with the control group and lead to mortality and cardiovascular disease. Due to early detection and treatment, the mortality rate has decreased over the past three decades (Despa & Bers, 2007). In fact, we can know the changes that occur to the heart muscle through disruption of enzymes such as (CK-MB and troponin) or ECG, but they often show a global pattern. Therefore, biochemical indicators are used to determine the progression of myocardial damage and confirm the diagnosis (Despa et al., 2012).

Researchers have found that potassium inside cells is very important for preventing MI from happening. If it can fix the heart's depolarization and contracting, it can prevent further damage from happening (El Sherif & Turitto, 2011). This study found that potassium levels were 3.92 ± 0.97 times lower in high blood pressure patients with AMI compared to non-high blood pressure patients 4.30 ± 0.75 (P = 0.026). This was because of β-agonism. Potassium can flow into cells when the body is under short-term stress. Catecholamines work on β_2-receptors to make this happen. A lot of K^+ is lost when the Na^+, K^+-ATPase pump is stopped by factors like insufficient oxygen or magnesium. The amount of magnesium went down by 0.81 ± 0.16. People with high blood pressure are more likely than people without high blood pressure to have their magnesium levels drop (P = 0.017).

This study showed a decrease in Mg^{2+} in hypertensive cases. Low level of magnesium predisposes one to increase in arterial pressure as Mg^{+2} acts peripherally to produce peripheral vasodilation and hence fall in blood pressure. In the present study, serum potassium concentration was decreased significantly in patients with MI with hypertension. which is similar to the results of Solomon & Cole (1981).

Edemas happen when people don't get enough salt. This is when fluid fills up the spaces between cells. If you look for pits in people's legs while they walk or in their pelvic area while they lie down, you can easily find it. When the amount of blood that works drops, this can happen. This can happen because of heart failure or low albumin levels. The body reacts to this by making secondary hyperaldosteronism happen. By storing sodium (and water), aldosterone increases the size of the extracellular fluid (ECF). When someone has an edema, their sodium levels are low even though they are hanging on to sodium. This is because hypovolemic people also release AVP, which makes them hold on to even more water. In other words, people with high blood pressure have a lot of sodium in their blood, while people who have had a heart attack don't have enough sodium in their blood (Gaw et al., 2013).

Therefore, tests show low amounts of blood sodium, as shown in the results of our study in Table 1. A study conducted by Flear et al. showed 45% of infarcted patients had hyponatremia and were associated with increased mortality (Rosen., 2008). And on the contrary, the calcium level was 2.11 ± 0.34 significantly lower with AMI in hypertensive patients compared with non-hypertensive patients (2.16 ± 0.38) and the control group (2.11 ± 0.34) due to an increase in the amount of sodium inside the cell and a decrease in the amount of

magnesium, in addition to a decrease in the amount of albumin and vitamin D (Belin & He, 2007; Catalano et al., 2012). A similar study showed a lower calcium level (Adkins & Curtis, 2015).

There was more chloride in the blood (88.0 ± 8.6) because opening cardiac chloride channels changes the membrane potential and action potential length in the sino-atrial node, which can cause irregular heartbeats. If a person with diabetes has an AMI, they might not have enough nutrients. Some of the main signs of diabetes are having to go to the bathroom more often, being thirsty, being tired, losing weight, having blurry vision, being hungry, and having diabetes dermopathy (Hasona & Elasbali, 2016). Many different factors, like age and other health problems, can change the relationship between minerals and blood sugar (Haffner, 2000). Glucose is an osmotically active chemical. When blood sugar levels are too high, it raises serum osmolality. This makes cells lose water and lowers sodium levels by diluting the serum (Barbagallo et al., 2007).

Because glucose changes the osmotic pressure, it also causes osmotic diuresis. This lowers the flow of blood through the body and dehydrates cells by removing water from inside them (Abu Marzoq et al., 2016). It was discovered that 78.3% of patients do not have enough electrolytes. This is different from the findings of Abu Marzoq et al. (2016), who only found an issue with potassium levels (Liamis et al., 2006). Hyponatremia has been linked to a higher chance of death in hospitalized patients (Hao et al., 2017). Hyponatremia (124.0 ± 12.2) was the most common electrolyte problem. Almost a third of the people who took part had sodium levels below 130 mEq/L. 68% of the people in this study had mild hyponatremia, while 26% had serious hyponatremia. As expected, people whose blood sugar was not under control or who had a MI were more likely to develop hyponatremia. This was because their kidneys were unable to control homeostatic processes like the feeling of thirst, release of antidiuretic hormone (ADH), or how they handled filtered sodium and diuretics. Riphagen et al. (2015) found that diabetic patients with MI had lower sodium levels. However, another study did not find a significant difference in the mean sodium levels between people with T2DM and people who did not have diabetes (Shahid et al., 2005).

72% of the people in this study had low potassium levels and 28% had high potassium levels. It was because the renin-angiotensin-aldosterone pathway was stopped. This pathway is very important for keeping fluid and electrolyte levels in check. Scientists have studied how this system of enzymes affects diabetes and other heart and hormone diseases (Milionis et al., 2001). Another study found that 16% of the people who took part had low potassium levels and 6% had high potassium levels. There wasn't a big difference in how often these chemical problems happened between diabetics who were treated and those whose blood sugar was not under control (Nilsson et al., 2017). Fluid and electrolyte balance can go wrong for people with diabetes mellitus if they don't have enough insulin, if their blood sugar is too high, or if their ketones level is too high (Nilsson et al., 2017).

What we found also was that 91% of the patients had low chloride levels (78.0 ± 11.3%) and only 9% had high chloride levels. A lot of things depend on serine-threonine kinases. They can tell when the amount of chloride inside cells, the size of cells, and the osmolarity outside of cells change. According to Kitabchi et al. (2006), when blood chloride levels drop, the WNK family makes the Na-K-2Cl cotransporter in the thick ascending limb of the loop of Henle and the Na-Cl symporters in the distal convoluted tubule work harder to help the body take back chloride. When you make these carriers work harder, your body may lose potassium and have trouble keeping its rhythm (Kazory & Ronco, 2020).

The amount of chloride inside cells can change when the potassium level in the plasma changes. This changes the function of WNK and the Na-Cl symporter. There is no WNK activity when there is a lot of potassium in the blood. This is because aldosterone is released. New studies show that chloride attaches directly to a catalytic site on WNK. This phosphorylates sodium regulatory pathways and helps keep blood pressure and electrolyte balance in check (Adkins & Curtis, 2015). One way in which that HF pathophysiology and chloride problems might be linked is through this. Low amounts of calcium and magnesium were also found (2.11 ± 0.50 and 0.83 ± 0.16, respectively). This happened because when people were thirsty, they drank and urinated copiously (Adkins & Curtis, 2015).

Correlation analysis showed significant negative correlations of sodium with hypertension ($r = –0.62$, $P = 0.001$) and diabetes ($r = –0.58$, $P = 0.002$), which is in accord with the results obtained by Feng et al. (2025), who demonstrated an association between hyponatremia and a worse cardiovascular prognosis. Additionally, magnesium had a weak negative correlation with hypertension and as reported by Qirjazi et al. (2021), this points to its role in the regulation of vascular tone. Nevertheless, the weak association between chloride and calcium with hypertension is in disagreement with findings by Klemens et al. (2021), who described the important role of calcium in the regulation of vascular tone. This gap may be explained by differences in sample size and patient comorbidities. On the whole, electrolyte disturbance, especially sodium and magnesium depletion, seems to be central in AMI pathophysiology.

Conclusion

Low levels of potassium and sodium in the blood are signs of a sudden myocardial infarction. Doctors can use a patient's serum sodium and potassium levels to guess how healthy they are. One sign that a patient is recovering is when the salt levels rise again after falling at first. So, measuring the amounts of sodium and potassium in people who have had an acute MI can help doctors figure out how likely it is that they will recover. Electrolytes are more likely to present problems for people who have diabetes. Most of the time, electrolyte problems were caused by low sodium, chloride, or potassium levels.

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