Review Article

ERA'S JOURNAL OF MEDICAL RESEARCH VOL.11 NO.2

DOI: 10.24041/ejmr2024.34

ASSOCIATION OF SERUM ELECTROLYTE CHANGES WITH ACUTE CORONARY SYNDROME: A NARRATIVE REVIEW

Shubham Saini[1], Jyoti Dwivedi[2], S. Mohd. Shiraz Rizvi[2]

Department of Biochemistry

SCPM College of Nursing and Paramedical Sciences, Gond, U.P. India - 271003[1]

Era's Lucknow Medical College and Hospital, Era University, Lucknow, U.P. India - 226003[2]

Received on : 17-10-2024

Accepted on : 13-12-2024

Address for correspondence

Dr. Shubham Saini

Department of Biochemistry

SCPM College of Nursing and Paramedical Sciences, Gonda U. P., India

Email: shubhamsainisrmsips18@gmail.com

Contact no: +91-8112328488

ABSTRACT

Blended learning (BL), which merges traditional face-to-face instruction with online education, has become a prominent approach in higher education, including dental science education. This review examines the concept of blended learning, its components, advantages, challenges, and applications within dental education. It emphasizes the value of combining in-person teaching with digital tools, such as online platforms, virtual simulations, and patient management software, to enhance learning outcomes. In dental science, blended learning offers a flexible and integrated approach where students engage in theoretical coursework, develop practical skills, and receive clinical training. Online learning modules provide flexibility, allowing students to learn at their own pace, while in-person classes support active interaction with instructors and hands-on experience. The review highlights that blended learning fosters personalized learning paths, increases accessibility, and promotes greater student-teacher engagement. Despite these advantages, the successful implementation of blended learning in dental education requires addressing challenges such as technological limitations, faculty training, and balancing online and hands-on experiences. The review concludes that with thoughtful planning and execution, blended learning has the potential to improve both the theoretical knowledge and clinical proficiency needed by dental professionals in today's rapidly changing healthcare landscape.

KEYWORDS: Acute Coronary Syndrome, Clinical Correlation, Electrolytes, Electrolyte Imbalance, Myocardial Infraction.

INTRODUCTION

"Acute coronary syndrome" describes a cluster of medical conditions marked by sudden reduction in blood flow to the heart. A heart attack and unstable angina are the terms used to describe these conditions (1). A heart attack happens when cells in the heart tissue die or get damaged. Myocardial infarction is another term for a heart attack. Stable angina may develop if there is a decrease in blood flow to the heart (2), there won't be any cardiac arrest or cell death as a consequence. On the other hand, risk of a heart attack may increase due to the reduced blood flow. Symptoms of acute coronary syndrome may range from mild discomfort to severe chest pain (3). Patient need to be evaluated and treated right away since it is a medical emergency. Early diagnosis and efficient treatment planning aims to improve cardiac blood flow, manage ischaemic injury, and prevent secondary complications.

ACS symptoms often appear all at once. Among them are:

- Chest pain or discomfort. Common symptoms include heat, constriction, pain, or pressure. Chest pain is also known as angina.
- Chest pain that spreads to other parts of the body. The areas that fall under this category include the back, neck, jaw, upper belly, shoulders, and arms.
- Queasy or throw up.
- Indigestion.
- Dyspnea, a medical term for shortness of breath.
- Abrupt, profuse perspiration.
- An irregular heartbeat.
- Having vertigo or light-headedness.
- Passing out.
- Atypical exhaustion.

Pain or stiffness in the chest is the most common sign, but the symptoms could be quite different from person to person based on factors including age, sex, and health conditions (4). Symptoms other than chest pain or discomfort are more common in elderly women, especially those with diabetes (5).

ROLE OF KEY SERUM ELECTROLYTES IN CARDIAC FUNCTION

Without the electrolytes calcium (Ca++), magnesium (Mg++), sodium (Na+), and potassium (K+), the myocardium, the heart's muscle tissue, cannot function properly (6). Muscle contraction occurs when there is an increase in voltage across the semi-permeable membrane of a cardiac cell beyond a certain threshold (7). A serum electrolyte test, which is crucial for tracking these critical ions. To guarantee proper cardiac function, electrolytes—which have an electrical charge—are maintained at physiological concentrations using a variety of techniques (for further details, see table-1, 'Standard serum concentrations'). An electrolyte imbalance may harm the heart, either directly or indirectly leading to arrhythmia and cardiac arrest. Serum calcium and magnesium abnormalities are less prevalent causes of life-threatening arrhythmias, but potassium disorders-particularly hyperkalaemia, which is a high potassium level-are often linked to them (8). Wider repercussions of electrolyte imbalances in the body are also possible, but are beyond the purview of this article.

Electrolyte	Standard Range
Potassium	3.5–5.0 mmol/L
Magnesium	0.7–1.1 mmol/L
Sodium	135–146 mmol/L
Calcium	2.20–2.67 mmol/L

Table 1: Standard serum concentrations (9)

Electrolyte concentrations in the heart may be impacted by changes in renal function since renal excretion is crucial to the body's ability to maintain electrolyte balance. Adrenal insufficiency, hypoaldosteronism, and kidney illness may all affect the potassium balance in the electrolyte balance (10). Apart from the significance of renal function in preserving electrolyte equilibrium, several medications may induce notable variations in serum electrolyte concentration via diverse pathways (see to the section under "Common medicines that can cause electrolyte disturbances") (11)

Electrolyte	Drug Class Causing Serum Deviation
Potassium	
Hypokalaemia	• Thiazide and similar diuretics (bendroflumethiazide, for example) • Loop diuretics, such as furosemide, • Agonists of beta 2 (such as Salbutamol) • Insulin • Aminoglycosides (gentamicin, for example)
Hyperkalaemia	• Diuretics that spare potassium, such amiloride • Antagonists of testosterone, such as spironolactone • Inhibitors of the angiotensin converting enzyme, such as ramipril • Antagonists of the angiotensin-II receptor, such as losartan • Ibuprofen and other non-steroidal anti-inflammatory medications • Heparin
Magnesium	
Hypomagnesaemia	• Thiazide and similar diuretics (bendroflumethiazide, for example) • Loop diuretics, such as furosemide, • Digoxin • Aminoglycosides (gentamicin, for example)
Hypermagnesaemia	• Antacids (such as trisodium magnesium) • Supplements containing magnesium
Sodium	
Hyponatraemia	• Thiazide and similar diuretics (bendroflumethiazide, for example) • Loop diuretics, such as furosemide, are • Vasopressin • Serotonin reuptake inhibitors that are selective, such as fluoxetine
Hypernatraemia	• Supplements containing sodium, such hypertonic saline • Lithium • Tetracycline (demeclocycline, for example) • Amphotericin • Use caution while taking medications that are rich in salt, such as effervescent formulations and injectable medications like acyclovir and benzyl penicillin.
Calcium	
Hypocalcaemia	• Bisphosphonates (such as alendronic acid) • Calcitonin
Hypercalcaemia	• Thiazide and similar diuretics (bendroflumethiazide, for example) • Vitamin D substitutes • Long-term lithium usage • Alpha Lipoic Acid

Table 2: Class of Drugs Causing Deviation in Serum

When treating moderate, asymptomatic electrolyte imbalances, it is typically best to focus on modifiable causes (13). These may include pharmaceutical side effects or changes in the patient's diet (for example, hyperkalaemia might be caused by drinking too much coconut water). When patients present with symptoms (and/or ECG abnormalities), as indicated above, and

when disrupted electrolyte levels have caused clinical manifestations, it is usually required to promptly provide medication to correct the levels (14).

POTASSIUM

When it comes to positively charged ions (cations) that are found within cells, potassium is by far the most common. Since the concentration inside cells is around twenty times greater than that in the surrounding fluid, a noticeable concentration gradient exists (15). This maintains the excitability of the nerve cells and muscles (16).

Some of the primary regulators of potassium levels include the hormone aldosterone (via renal excretion), catecholamines, insulin, and bicarbonate solutions. Potassium concentrations are also affected by pH (17). In acidemia, when the serum pH is low and potassium is released into the bloodstream, the levels of potassium in the serum increase; in alkaemia, when the serum pH is high and potassium is retained in the cells, the levels of potassium in the serum decrease (18). Heart conduction problems are important whether there is hyperkalaemia or hypokalaemia (19). The severity of changes in potassium levels is not well defined; rather, it is considered a continuum, with the associated clinical symptoms being used to rate the change's severity (20).

HYPERKALAEMIA

If treatment for hyperkalemia is not received, the condition's developing conduction abnormalities might result in cardiac arrest and death (21). Suppressed conduction may result in towering T waves as well as extended PR and QRS intervals. Treatment include transferring potassium into cells, protecting the heart, and eliminating potassium from the body (22). Polystyrene sulphonate resins may be used to progressively lower potassium levels in moderate situations, but they shouldn't be administered to people who have obstructive bowel disease (23). A quick change in extracellular potassium is necessary for moderate hyperkalemia, and patients may need intravenous doses of glucose and soluble insulin (24). Both of these therapies are necessary for severe hyperkalemia, in addition to high-dose beta2 agonists and sodium bicarbonate (25). Calcium gluconate (10%) should be used to treat severe hyperkalemia or ECG abnormalities because it raises the myocytes' threshold potential. Digoxin users should avoid rapid calcium delivery since it increases their risk of arrhythmia (26).

HYPOKALAEMIA

Ventricular tachycardia may result from hypokalemia, a condition in which blood potassium levels are less than 3.5 mmol/l (27). Diarrhoea, hypercalcemia, hyperaldosteronism, medications, renal excretion, diabetes, and excessive fluid replacement treatment may all be contributing factors (28). Premature ventricular or atrial complexes, depressed ST segments, flattened T waves, and U waves may all be seen on an ECG (29). While severe instances need intravenous potassium, mild hypokalaemia may be handled conservatively with or without dietary supplements. Both on-going ECG monitoring and routine monitoring are required (30). In extreme situations, magnesium supplementation is advised for quick treatment of hypokalaemia.

MAGNESIUM

Among cations found within cells, magnesium is the second most prevalent. When magnesium and the enzyme sodium-potassium ATPase interact, the enzyme pumps potassium into cells in return for sodium, mostly regulating cellular concentration gradients (31).

HYPERMAGNESAEMIA

Hypermagnesaemia is an electrolyte disorder in which there is a high level of magnesium in the blood (32). It is mostly eliminated by the kidneys, which have a great secretory capacity. Magnesium levels might rise with significant necrosis or soft-tissue damage. Generally asymptomatic, patients with blood magnesium levels between 1.0 and 2.0 mmol/l may be more harmful if they take digoxin (33). When levels exceed 4.0 mmol/l, patients may become weak, nauseous, and undergo respiratory failure, paralysis, and coma. A systole may also be brought on by hypomagnesaemia. Dialysis is used for individuals with severe renal impairment, whereas intensive intravenous hydration treatment is used for hypovolemia and normal renal function. Treatment is based on the patient's fluid and kidney function (34).

HYPOMAGNESAEMIA

Due to genetic predispositions, inadequate magnesium intake, or adverse reactions to beta2-agonists, corticosteroids, or theophylline, hypomagnesaemia-a disorder that impairs the absorption of magnesium from the gastrointestinal tract-can develop in people with either acute or chronic asthma. Neuromuscular signs such as tremors, seizures, delirium, and psychosis are among the symptoms. Prolonged PR and QT intervals brought on by severe hypomagnesaemia might result in longer QRS durations and torsades de pointes (35). Individuals who have hypomagnesaemia brought on by diuretics should stop taking their medication or get a potassium-sparing diuretic. Intravenous magnesium

should be used to treat significant hypomagnesaemia. This may be done as a 6-hour infusion or as an 8–20 mmol bolus dose (36).

SODIUM

Serum osmolality is significantly affected by sodium, the principal extracellular cation in the human body (37). Because it modulates myocardial membrane potentials with potassium, it is crucial for the regulation of cardiac action potentials. Changes in blood sodium levels, in contrast to potassium, do not cause major cardiac problems until there is a significant deviation from normal physiological standards.

Sodium variations often cause nausea, vomiting, weakness, and disorientation. If treatment is not received, these symptoms might worsen and perhaps put a person in a coma or seizure. ECG alterations that are consistent are uncommon (38).

Patients with severe heart failure often exhibit excess total body water relative to sodium, which is caused by inadequate compensatory mechanisms for sodium control (leading in hypervolaemic hyponatraemia). Patients have to be put on a fluid restriction regimen and given diuretics, which will lower water retention and progressively raise sodium levels in the blood.

HYPERNATRAEMIA

When the body loses more water than sodium, the sodium content in the blood rises over 145 mmol/L, a condition known as hypernatraemia. Dehydration is often the source of this, particularly in those with defective thirst systems and people who are physically or mentally disabled. Lethargy, muscular weakness, and irritability are some of the symptoms. Patients may endure disorientation, convulsions, and in extreme situations, a coma or even death, as the illness worsens. There are many situations in which hypernatraemia may arise, such as hypopovolemic, euvolemic, and hypervolemic hypernatraemia. In order to prevent cerebral edema, treatment focuses on replenishing lost fluids and reducing salt levels gradually rather than quickly. Oral water consumption or intravenous hypotonic solutions are often enough in mild instances. Intravenous fluids are given cautiously and gradually in severe instances to lower salt levels and restore water balance without causing brain edema. It's critical to treat the underlying causes of hypernatraemia, such as dehydration or a high salt diet. Throughout the course of therapy, careful monitoring of neurological state and serum salt levels is important.

HYPONATRAEMIA

When the blood's sodium content drops below normal—typically below 135 mmol/L—it is referred to as hyponatraemia. This results in an imbalance between salt and water in the blood, which is typical for people suffering from various medical disorders that cause fluid overload or severe heart failure. From moderate to severe symptoms, early indicators include nausea, headaches, dizziness, and vomiting. As the disease progresses, swelling of the brain cells may cause neurological symptoms. Based on the amount of fluid consumed, hyponatraemia can be categorized as follows: hypopovolemic hyponatraemia, which occurs when sodium loss outweighs water loss; euvolemic hyponatraemia, which results in a normal fluid level but an imbalance of sodium; and hypervolemic hyponatraemia, which results in an increase in both sodium and water but a greater degree of water retention. The intensity and underlying cause determine the course of treatment. Fluid restriction is often enough to minimize water retention and progressively raise salt levels in moderate situations. Hypertonic saline may be used to restore sodium levels in extreme situations, however excessively harsh therapy might result in osmotic demyelination syndrome, a dangerous neurological disorder.

CALCIUM

In the myocardium, calcium plays a crucial role in conduction, intracellular signaling, and fiber contraction. In instance, calcium levels might affect cardiac conduction by changing the duration of the plateau phase (phase 2) of the myocardial action potential.

A short QT interval may be caused by high calcium levels, whereas a longer QT interval can be caused by low calcium levels. Cardiac arrest may result from conduction anomalies at even greater extremes (39).

HYPERCALCAEMIA

It may cause QT intervals to shorten, and AV block may ensue if treatment is not received. Furthermore, hypercalcemia damages smooth muscle fibers, which results in muscular weakness (40).

Patients whose calcium levels are high (>2.67 mmol/l) and whose electrocardiograms reveal a shortened QT interval need urgent treatment. If an electrocardiogram (ECG) shows that a patient has a longer QRS interval, loop diuretics may be used to increase calcium excretion. Thiazides and similar diuretics, on the other hand, might cause hypercalcaemia. Aggressive fluid administration, such sodium chloride 0.9%, may be used to control hypercalcaemia initially. Standard practice for individuals undergoing treatment for both cancer and osteoporosis is the intravenous administration of bisphosphonates to reduce blood calcium levels and slow the rate of bone turnover.

HYPOCALCAEMIA

The QT interval will prolong with hypocalcemia, increasing the risk of AV block and cardiac arrest. Tetany and cramping are signs of hypocalcaemia. Individuals who exhibit these symptoms and have blood calcium levels less than 2.1 mmol/l need to get intravenous calcium therapy as soon as possible. If at all feasible, reversible causes of hypocalcaemia such as medication-induced hypocalcaemia—should be addressed. Intravenous magnesium should be administered to patients receiving treatment for hypocalcaemia in order to assist in restoring serum calcium levels.

ELECTROLYTE IMBALANCES IN ACS

ACS is a medical disorder caused by a reduction in blood flow to the heart's coronary arteries. The ACS is divided into three subgroups:

1. Elevated myocardial infarction with ST segment (STEMI)
2. Elevated MI not in the ST segment (NSTEMI)
3. Unstable Angina (UA), determined by cardiac biomarkers and ECG findings.

The hallmarks of STEMI are total blood artery blockage, which causes a transmural infarct to the myocardium and an increase in troponin levels. Plaque rupture and thrombus development in NSTEMI lead to partial blood artery blockage, which causes a myocardial infarct and an increase in troponins. In unstable angina, angina pain that occurs at rest or increases quickly over a short period of time is accompanied with partial blockage of the artery and normal troponins due to plaque ruptures and thrombus development that occur without an infract.

One of the signs and symptoms of coronary heart disease (CHD) is angina. Cardiovascular disease accounts for 7.2 million annual deaths worldwide and 12.8% of all fatalities. In India, the prevalence of CHD is 2.2% in rural areas and 6.4% in urban areas. The "global burden of disease study age standardized estimates (2010)" states that cardiovascular illnesses account for around 24.8% of all deaths in India (41).

Electrolytes play a critical role in the proper operation of cells and key organs, such as the heart. Calcium (Ca++), potassium (K+), and sodium (Na+) are some of the most crucial electrolytes for the body's essential functions. Other significant electrolytes include phosphate, bicarbonate, magnesium (Mg++), and chloride ions. Normal levels of Na+, K+, and Ca++ control the heart's electrical activity. The proper balance of these electrolytes is essential for the heart to operate normally. After an ACS episode, electrolyte imbalance is often seen, and it may play a significant role in modifying the outcome of ACS.

Myocyte depolarization and myocardial contractility are mediated by calcium. Serum calcium levels (total) should be between 8.5 and 10.5 mg/dL. Atherosclerotic plaque disruption, thrombus development, and coronary spasm are all possible consequences of hypocalcemia.

Early on following ACS episodes, electrolyte imbalance is important for prognosis. Therefore, the purpose of the current research was to evaluate the electrolyte imbalance (Na+, K+, and Ca++) in ACS patents (42).

METHOD

DATA SOURCES AND TERMS OF SEARCH

The project will use patient records from the hospital's cardiac care unit to gather data, with a focus on patients who have been hospitalized with acute coronary syndromes. Serum electrolyte values (sodium, potassium, calcium, magnesium, and chloride) will be provided via laboratory results both at admission and during the patient's hospital stay. Furthermore, pertinent patient data, including demographics, medical history, comorbidities, and therapy specifics, will be retrieved from clinical databases. Key phrases like "acute coronary syndrome," "serum electrolytes," "electrolyte imbalance," "mortality," "morbidity," and "cardiac arrhythmias" will be used to search databases like PubMed, Scopus, and Cochrane Library for the literature review.

DATA EXTRACTION

In order to extract data, pertinent information must be gathered from databases and medical records. Electrolyte levels, clinical outcomes (mortality, arrhythmias, ICU hospitalization), and patient demographic information will be extracted by skilled experts. To guarantee uniformity, a pre-established data collecting sheet will be used (43). Baseline electrolyte levels, variations throughout the hospital stay, and results after discharge will all be included in the retrieved data. Important information about the studies that are part of the literature review, such sample size, electrolyte measures, clinical results, and research findings, will be methodically taken out and compared.

DATA ANALYSIS

An overview of studies on serum electrolytes and associated indicators in different clinical circumstances is provided in the table. The emphasis was on the relationship between baseline electrolyte

imbalances and illness outcomes in COVID-19 patients, as shown by the paper Electrolyte abnormalities in COVID-19: Association with disease outcomes. The significance of these anomalies on the disease's severity and prognosis was brought to light by this inquiry. Rhabdomyolysis: A prevalent condition with different causes and therapeutic options is a noteworthy research that offered a thorough analysis of the etiology, consequences, and therapy of Rhabdomyolysis. It emphasized the range of etiological variables and approaches to treating this illness, which often include abnormalities in electrolytes.

Furthermore, how different blood laboratory values may be utilized for diagnosing and forecasting the course of epilepsy was examined in the review titled Diagnostic and prognostic usefulness of laboratory values in epilepsy. The significance of laboratory testing in differentiating between seizure types and their therapeutic implications was highlighted in this paper. Finally, the function of cardiac and electrolyte indicators in determining myocardial damage in Acute Myocardial Infarction (AMI) was the focus of markers of myocardial damage and their connection with AMI prognosis. This research described the potential predictive significance of these indicators in AMI and how they may indicate the degree of myocardial damage.

ELECTROLYTES AND CLINICAL OUTCOMES IN ACS

One of the most important factors affecting the clinical course of individuals with ACS is the electrolyte balance in the serum. Electrolyte abnormalities are often important factors in determining a patient's prognosis and may directly affect how well the heart functions, raising the risk of problems (49).

IMPACT ON MORTALITY AND MORBIDITY

Patients with ACS often have electrolyte abnormalities, such as hypokalemia, hyperkalemia, hyponatremia, and hypocalcemia. These abnormalities are strongly associated with increased risks of death and morbidity. If these imbalances are not properly handled, they often result in serious difficulties because they impair vital physiological processes in the heart and circulatory system.

References	Topic Covered	Research Study	Title
Nitesh MS *et.al.,(*2023) (44)	Serum electrolyte concentrations in ACS patients	electrolyte imbalances (sodium, potassium, calcium, and magnesium) and the onset/severity of ACS are correlated, emphasizing the significance these imbalances have in cardiac function and possible consequences	Acute Coronary Syndrome: A Serum Electrolyte Study
Challoob H *et.al.*,(2023) (45)	Serum electrolytes and trace elements in AMI patients	assessed the blood electrolytes and trace elements (zinc, copper, magnesium) in patients with AMI, finding notable changes in the electrolyte levels and their significance for the prognosis of AMI	Assessment of Certain Trace Elements and Serum Electrolytes in Acute Myocardial Infarction Patients
Abdalla *et. al.,*(2023) (46)	Relationship between MI, electrolytes, and vitamin D deficiency	investigated how vitamin D insufficiency affects electrolyte imbalances (calcium, potassium, magnesium) and how it affects the severity of myocardial infarction in individuals with acute ACS	Relationship between Electrolyte Level, Vitamin D Deficiency, and Myocardial Infarction in ACS Patients
Hasan *et. al.,* (2019) (47)	Serum electrolyte levels in healthy people, VHD, and MI	highlighted electrolyte abnormalities by comparing the amounts of electrolytes (sodium, potassium, calcium, and magnesium) in normal people, VHD patients, and MI patients	A Comparison of Normal Subjects' Serum Electrolyte Concentrations with VHD and MI
Patil *et.al.,* (2016) (48)	Unbalanced electrolytes in AMI patients	examined the frequency and trends of electrolyte abnormalities (sodium, potassium, and calcium) in patients with AMI, finding hypokalemia and hyponatremia to be prevalent and high-risk variables	An Examination of Electrolyte Disproportion in Patients with Acute Myocardial Infarction at a Maharashtra Tertiary Care Facility

Table 3: Research Study Data

Potassium (K^+) is one of the most important electrolytes for heart function. For people with ACS, low potassium levels (hypokalemia) or high potassium levels (hyperkalemia) may both have detrimental effects. Hypokalemia impairs normal cardiac conduction, which may lead to potentially fatal arrhythmias such ventricular fibrillation. This raises the risk of sudden cardiac death, especially in those with weakened hearts who are already at risk. Conversely, hyperkalemia may make the heart excessively arrhythmogenic, which may result in cardiac arrest. Patients with reduced renal function or those using drugs that affect potassium levels, such as ACE inhibitors or diuretics, are more vulnerable to this imbalance. Thus, there is a significant chance of death in ACS associated with both types of potassium imbalance.

Another essential electrolyte is sodium (Na^+), and hyponatremia in particular is strongly linked to heart failure and worse outcomes in ACS patients. A typical symptom of fluid overload in individuals with compromised cardiac function is hyponatremia. Low sodium levels are a substantial predictor of higher mortality in these individuals, according to several studies. Furthermore, hyponatremia is associated with increased rates of rehospitalization and longer hospital stays, indicating its effect on the exacerbation of heart failure and general decline in health.

The electrolyte calcium (Ca^{++}), which is necessary for cardiac conduction and contractility, is also critical to the prognosis of individuals with ACS. Too high (hypercalcemia) or too low (hypocalcemia) of a calcium level imbalance may cause severe arrhythmias in the heart. Hypercalcemia may lengthen the QT interval on an ECG, which puts patients at risk for arrhythmias, while hypocalcemia can impair the heart's capacity to contract efficiently. These disruptions raise the possibility of death and morbidity while also complicating the clinical course of ACS (50).

Despite being often disregarded, magnesium (Mg^{++}) is essential for preserving adequate cardiac function, especially when it comes to controlling ventricular rhythms. Hypomagnesemia, or low magnesium levels, is closely linked to a higher risk of arrhythmias, especially in individuals who are very sick. Because restoring magnesium levels may greatly lower the occurrence of arrhythmias and improve patient outcomes, magnesium monitoring is crucial in the treatment of ACS patients. Maintaining the proper balance of magnesium is especially crucial since it influences not only heart rhythm but also the stability of other electrolytes such as potassium and calcium via interactions.

ELECTROLYTE MANAGEMENT IN ACUTE CORONARY CARE UNITS

Maintaining electrolyte balance is an essential part of patient treatment in acute coronary care units (ACCUs). Since ACS is associated with potentially fatal consequences, it is essential to continuously monitor blood electrolytes in order to preserve cardiovascular stability. By using this technique, electrolyte abnormalities that would otherwise cause serious consequences can be identified early on. Abnormal levels of potassium, calcium, sodium, or magnesium are examples of electrolyte abnormalities that may exacerbate heart failure and raise the risk of arrhythmias and cardiac arrest. Clinicians may improve patient outcomes and lower the likelihood of further problems during hospitalization by routinely monitoring electrolyte levels and avoiding these potentially harmful episodes.

Crucial treatment choices in ACCUs are also heavily influenced by efficient electrolyte control. For example, low potassium levels, or hypokalemia, may cause dangerous cardiac arrhythmias. Potassium supplements can help treat hypokalemia. On the other side, drugs like calcium gluconate to steady the heart, insulin-glucose combinations to transfer potassium into cells, or potassium-binding resins to remove excess potassium may be used to treat hyperkalemia (high potassium levels), which can cause cardiac arrest. By helping to restore electrolyte balance, these procedures guarantee that the heart continues to beat at its best and lower the chance of unexpected cardiac events.

Vigilant monitoring is even more important in light of the link between electrolyte levels and drugs used to treat ACS. Treatments like beta-blockers, ACE inhibitors, and diuretics may all have a big impact on electrolyte balance. For example, it is well known that diuretics deplete potassium whereas ACE inhibitors increase potassium levels. These drugs may aggravate pre-existing electrolyte imbalances and worsen the patient's health if they are not carefully monitored. In order to prevent difficulties, healthcare providers must make sure that the drugs they administer do not cause harmful changes in electrolytes. This will enable them to make necessary modifications to therapy as required.

In more extreme situations, electrolyte imbalances may need to be corrected quickly, often including intravenous (IV) electrolyte treatment. In patients whose potassium or magnesium levels are dangerously low, IV treatment provides a quick and regulated way to bring their bodies back into balance. In situations when oral supplements may be

inadequate or too slow to treat the critical imbalance, prompt action is essential to stabilize the patient's state. In order to keep patients on a stable recovery path by halting the development of acute illnesses to more severe states, including arrhythmias or heart failure, IV electrolyte treatment is a routine procedure in ACCUs.

COMPARISON OF OUTCOMES WITH AND WITHOUT ELECTROLYTE CORRECTION

Patients with ACS who undergo electrolyte correction on time or not often have extremely different clinical outcomes, which may have an impact on both short- and long-term survival. In critical care environments, stabilizing cardiac function, averting potentially fatal consequences, and guaranteeing the best prognosis for patients depend on the appropriate treatment of electrolyte imbalances.

There are significant advantages for patients who obtain electrolyte correction on time. Heart rhythms are directly impacted by imbalances such as hypo- or hyperkalemia, which must be corrected in order to normalize the heart's electrical activity and lower the risk of potentially deadly arrhythmias. For instance, ventricular fibrillation, a dangerous arrhythmic condition that may be fatal if left untreated, is known to be brought on by hypokalemia (low potassium levels). Healthcare professionals may greatly reduce the risk of these harmful events by restoring potassium levels via supplements or other therapies, giving the heart a more stable environment for healing. In a similar vein, inadequate management of hyperkalemia (high potassium levels) may result in cardiac arrest. Treatments such as potassium-binding drugs, insulin-glucose therapy, or calcium gluconate may be used to rapidly restore potassium levels to a range that is safer and delay the start of sudden cardiac death.

For ACS patients, especially those who have heart failure, maintaining a balanced sodium intake is crucial. Low sodium levels, or hypernatremia, are a typical indicator of declining heart health and are highly correlated with unfavourable outcomes including lengthier hospital admissions and increased death rates. Shortness of breath and fluid retention are two heart failure symptoms that may be improved by promptly adjusting sodium levels by fluid management or sodium supplements. This improves the patient's quality of life and lowers the risk of readmissions to the hospital since stable sodium levels help better control the symptoms of heart failure over the long run. In this situation, electrolyte management may result in a more predictable and effective healing process, assisting patients in regaining stability more quickly and smoothly returning to their regular lives.

On the other hand, patients who do not obtain the proper electrolyte correction will have far severe consequences. Untreated or improperly controlled electrolyte imbalances may have catastrophic consequences for cardiovascular health. For instance, since chronic hyperkalemia severely disrupts heart electrical activity, it might result in cardiac arrest. The likelihood of unexpected, deadly occurrences like asystole—the total stoppage of cardiac electrical activity—increases with the length of time hyperkalemia is left untreated. In a similar vein, untreated hypokalemia may lead to protracted arrhythmias that exacerbate cardiac injury and raise the risk of death. Due to consequences from these imbalances, these patients are also more prone to have longer hospital admissions, which may hinder their recovery and raise their medical expenses.

If left untreated, hyponatremia makes heart failure symptoms worse by making the already weak heart even more strained and retaining more fluid. This worsens cardiac function, raises the possibility of morbidity, and increases the likelihood of unfavorable long-term consequences. Due to inadequate electrolyte management, these patients' conditions worsen and they often need hospital stays, with no improvement in their quality of life. In addition to raising death rates, electrolyte imbalances may cause a vicious cycle of declining health and recurrent medical emergencies if left untreated.

CONCLUSION

The investigation into serum electrolytes in ACS highlights the critical role that key electrolytes such as sodium, potassium, calcium, and magnesium play in cardiac function and their influence on clinical outcomes. Electrolyte imbalances, commonly exacerbated by certain drug classes and disease progression, are strongly associated with increased mortality and morbidity in ACS patients. Effective electrolyte management, particularly in acute coronary care settings, has been shown to improve clinical outcomes, with corrective interventions leading to better patient survival rates and reduced complications. Therefore, precise monitoring and timely correction of electrolyte disturbances are essential for optimizing care in ACS.

Abbreviation

ACS	Acute coronary syndrome
AMI	Acute myocardial infarction
CHD	Coronary heart disease
ECG	Electrocardiogram
MI	Myocardial Infarction

Ca^{++}	Calcium
Mg^{++}	Magnesium
Na^{+}	Sodium
K^{+}	Potassium
VHD	Valvular heart disease
UA	Unstable angina

REFERENCES

1. Mandole MB, Howale DS, Mamatha MT, et al. Evaluation of renal function tests and serum electrolytes in patients with acute myocardial infarction. Int J Biomed Res. 2016;7(9):676-9.
2. Amin MN, Ali AM, Hussain AB, et al. Estimation of serum electrolytes dysfunction in acute MI patients presented at tertiary care hospital in Pakistan. Atherosclerosis. 2020 Dec 1;315:e227.
3. Aziz A, Abdul-Hassan M, Mohammed K. Plasma atrial natriuretic peptide level and serum electrolyte status among patients with acute myocardial infarction complicated with cardiac arrhythmia. Eur. J. Mol. Clin. Med. 2020;7(2):4352-60.
4. Whitted AD, Dube P, Komolafe BO, et al. A dyshomeostasis of electrolytes and trace elements in acute stressor states: impact on the heart. The American journal of the medical sciences. 2010 Jul 1;340(1):48-53.
5. Bo X, Liu Y, Hao C, et al. Risk stratification and predictive value of serum sodium fluctuation for adverse prognosis in acute coronary syndrome patients. Clinica Chimica Acta. 2023 Aug 1;548:117491.
6. Hamim HA, Al-Timimi RJ, Al-Taweel RB. Association of chemerin with biochemical parameters and electrolyte imbalance in iraqi patients with acute coronary syndrome. Biochemical & Cellular Archives. 2021 Oct 1;21(2).
7. Nieper HA, Blumberger K. Electrolyte transport therapy of cardiovascular diseases. Electrolytes and Cardiovascular Diseases. 1966;2.
8. Dafalla AM, Dafalla LA, Khalafalla AS, et al. Impact of Cardiac Disorders on Renal Function Tests and Serum Electrolytes in Sudanese Patients with Acute Myocardial Infarction. Muthanna Medical Journal. 2022;9(2).
9. Fan Q, Bai Z, Ndjana Lessomo et al. Variations in Post-Operative Electrolyte in Coronary Artery Intervention. International Journal of General Medicine. 2023 Dec 31:4629-36.
10. Jain S, Sharma R. Evaluation of Electrolyte Imbalance in Myocardial Infarction Patients at Tertiary Care Center. International Journal of Medical Science and Education. 2018;5(1):117-21.
11. Fernando MB, Ferreira I, Lourenço P. Admission and discharge sodium: Chloride ratio in acute heart failure–The importance of electrolytes. International Journal of Cardiology. 2024 May 22.
12. Mohammadifard N, Gotay C, Humphries KH, et al. Electrolyte minerals intake and cardiovascular health. Critical reviews in food science and nutrition. 2019 Aug 22;59(15):2375-85.
13. Klimenko LL, Skalny AV, Tutna AA, et al. Serum electrolytes are associated with markers of neural damage in transient ischemic attack and ischemic stroke patients. Trace Elements and Electrolytes. 2017 Jan 1;34(1):29-33.
14. Seyedi SZ, Zahedi M, Farahani P, et al. Electrolyte Disturbances in Post-Acute Myocardial Infarction Arrhythmias. Tabari Biomedical Student Research Journal. 2023 Sep 10;5(2):39-44.
15. Kothavale S, Patil J, Kulkarni G, et al. Evaluation of Serum Sodium Levels in Acute Myocardial Infarction patients: A Clinical Study. Age (years).;42(41.6):0-5.
16. Cao Z, Zhu W, Shen C, et al. Association of baseline serum sodium with long-term outcomes in newly diagnosed coronary heart disease patients without heart failure: a prospective cohort study. Scientific Reports. 2024 Aug 6;14(1):18154.
17. Milionis HJ, Alexandrides GE, Liberopoulos EN, et al. Hypomagnesemia and concurrent acid–base and electrolyte abnormalities in patients with congestive heart failure. European journal of heart failure. 2002 Mar;4(2):167-73.
18. Shiraz Rizvi SM, Sunny S, Wani IA, et al. Influence of electrolyte imbalance on regional wall motion abnormalities in STEMI patients of North Indian origin. Frontiers in Cardiovascular Medicine. 2023 Nov 30;10:1223954.
19. Terlecki M, Kocowska-Trytko M, Kurzyca A, et al. The sodium–chloride difference: A marker of prognosis in patients with acute myocardial infarction. European Journal of Clinical Investigation. 2024 May;54(5):e14157.
20. Altura BM, Altura BT. Magnesium, electrolyte transport and coronary vascular tone. Drugs. 1984

Nov;28:120-42.

21. Omland T, Samuelsson A, Hartford M, et al. Serum homocysteine concentration as an indicator of survival in patients with acute coronary syndromes. Archives of internal medicine. 2000 Jun 26;160(12):1834-40.
22. Singla I, Zahid M, Good CB, et al. Effect of hyponatremia (< 135 mEq/L) on outcome in patients with non–ST-elevation acute coronary syndrome. The American journal of cardiology. 2007 Aug 1;100(3):406-8.
23. Grodin JL, Simon J, Hachamovitch R, et al. Prognostic role of serum chloride levels in acute decompensated heart failure. Journal of the American College of Cardiology. 2015 Aug 11;66(6):659-66.
24. Wannamethee SG, Shaper AG, Lennon L, et al. Mild hyponatremia, hypernatremia and incident cardiovascular disease and mortality in older men: a population-based cohort study. Nutrition, Metabolism and Cardiovascular Diseases. 2016 Jan 1;26(1):12-9.
25. Kwoczynski Jk, Ekmekci A, Toyoshima H, et al. Electrocardiographic ischemic patterns without coronary artery disease. Diseases of the Chest. 1961 Mar 1;39(3):305-16.
26. NACB Writing Group Members, Morrow DA, Cannon CP, Jet al. National Academy of Clinical Biochemistry Laboratory Medicine Practice Guidelines: clinical characteristics and utilization of biochemical markers in acute coronary syndromes. Circulation. 2007 Apr 3;115(13):e356-75.
27. Steg PG, Dabbous OH, Feldman LJ, et al. Determinants and prognostic impact of heart failure complicating acute coronary syndromes: observations from the Global Registry of Acute Coronary Events (GRACE). Circulation. 2004 Feb 3;109(4):494-9.
28. Urso C, Brucculeri S, Caimi G. Acid–base and electrolyte abnormalities in heart failure: pathophysiology and implications. Heart failure reviews. 2015 Jul;20:493-503.
29. Kato TS, Nakamura H, Murata M, et al. The effect of tolvaptan on renal excretion of electrolytes and urea nitrogen in patients undergoing coronary artery bypass surgery. BMC Cardiovascular Disorders. 2016 Dec;16:1-9.
30. Rivera FB, Alfonso P, Golbin JM, et al. The role of serum chloride in acute and chronic heart failure: a narrative review. Cardiorenal Medicine. 2021 Apr 19;11(2):87-98.
31. Gu X, Ding X, Sun H, et al. Usefulness of serum calcium in the risk stratification of midterm mortality among patients with acute coronary syndrome. BioMed Research International. 2019;2019(1):9542054.
32. Breen TJ, Brueske B, Sidhu MS, et al. Abnormal serum chloride is associated with increased mortality among unselected cardiac intensive care unit patients. PloS one. 2021 Apr 26;16(4):e0250292.
33. Alcalai R, Planer D, Culhaoglu A, et al. Acute coronary syndrome vs nonspecific troponin elevation: clinical predictors and survival analysis. Archives of internal medicine. 2007 Feb 12;167(3):276-81.
34. Jain S. Evaluation of Serum Sodium Levels in Acute Myocardial Infarction Patients at a Tertiary Care Teaching Centre: A Clinical Study.
35. Ahmad F, Ilyas M, Rauf MA, et al. Electrolyte disorders and renal dysfuntion in patients with chronic heart failure. Pakistan Heart Journal. 2013;46(3).
36. Vamne A, Pathak S, Thanna RC, et al. Status of ggt, uric acid and serum electrolytes in myocardial infarction.
37. Patel AA, Mohanty A, Garg S, et al. Serum sodium levels in acute myocardial infarction and their relationship to the severity and consequences of myocardial infarction. Journal of Advanced Medical and Dental Sciences Research. 2022 Apr 1;10(4):48-51.
38. Christenson RH, Duh SH, Sanhai WR, et al. Characteristics of an albumin cobalt binding test for assessment of acute coronary syndrome patients: a multicenter study. Clinical Chemistry. 2001 Mar 1;47(3):464-70.
39. Kral M, Skoloudik D, Sanak D, et al. Assessment of relationship between acute ischemic stroke and heart disease-protocol of a prospective observational trial. Biomedical Papers. 2012 Sep 1;156(3):284-9.
40. Hjortshøj S, Kristensen SR, Ravkilde J. Diagnostic value of ischemia-modified albumin in patients with suspected acute coronary syndrome. The American journal of emergency medicine. 2010 Feb 1;28(2):170-6.
41. Jewad AM, Jihad IA. Role of heart failure in variation of serum alt, ast, alp, bilirubin and electrolytes. Biochemical & Cellular Archives. 2021 Oct 1;21(2).

42. Inokubo Y, Hanada H, Ishizaka H, et al. Plasma levels of matrix metalloproteinase-9 and tissue inhibitor of metalloproteinase-1 are increased in the coronary circulation in patients with acute coronary syndrome. American heart journal. 2001 Feb 1;141(2):211-7.
43. Pramanik SS, Harley KN, Jambhulkar RK, et al. Investigating pre and post-operative blood gases and serum electrolyte in patients undergoing coronary artery bypass surgery (CABG).
44. Nitesh MS. A study of serum electrolytes in acute coronary syndrome. Int J Acad Med Pharm. 2023;5(3):1166-70.
45. Hussein Challoob M, Mohammed MT. Evaluation of Some Trace Elements and Serum Electrolytes in Patients with Acute Myocardial Infraction. Egyptian Journal of Chemistry. 2023 May 1;66(5):93-7.
46. Abdalla NA, Mohamed AA, Ibrahim FA, et al. Association between Vitamin D Deficiency and Electrolyte Level and Myocardial Infarction in Patients Presenting with Acute Coronary Syndrome. The Egyptian Journal of Hospital Medicine. 2023 Jul 1;92(1):5570-6.
47. Hasan R, Serafi A, Javed A, et al. A study to compare serum electrolytes concentrations of normal individuals with valvular heart disease and myocardial infarction patients. Int J Cardiovasc Dis Diagn. 2019;4(1):022-7.
48. Patil S, Gandhi S, Prajapati P, et al. A study of electrolyte imbalance in acute myocardial infarction patients at a tertiary care hospital in western Maharashtra. International Journal of Contemporary Medical Research. 2016 Dec;3(12):3568-71.
49. Al Suwaidi J, Reddan DN, Williams K, et al. Prognostic implications of abnormalities in renal function in patients with acute coronary syndromes. Circulation. 2002 Aug 20;106(8):974-80.
50. Zandijk AJ, van Norel MR, Julius FE, et al. Chloride in heart failure: the neglected electrolyte. Heart Failure. 2021 Dec 1;9(12):904-15.

Orcid ID:

Shubham Saini - https://orcid.org/0009-0001-1302-3434

Jyoti Dwivedi - https://orcid.org/0009-0009-2277-0085

S. Mohd. Shiraz Rizvi - https://orcid.org/0009-0004-6361-6088

How to cite this article:

Saini S, Dwivedi J, Rizvi SMS. Association of Serum Electrolyte changes with Acute Coronary Syndrome: A Narrative Review. Era J. Med. Res. 2024; 11(2): 199-209.