

Biochemical Study of Blood Electrolytes, Hypokalaemia, and Hyperkalaemia in Genetic Heart Disease

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Abstract:

This study investigated the critical relationship between serum electrolyte homeostatic imbalances and hereditary heart disease within Wasit Governorate, Iraq. Conducted between October 2024 and June 2025 at Al-Karama Hospital and Al-Zahraa Teaching Hospital, the research evaluated peripheral blood samples from 68 patients diagnosed with hereditary cardiovascular conditions alongside a control group of healthy donors. Specifically, patients exhibited severe hyponatraemia (117.49 ± 19.47 mmol/L versus 136.00 ± 2.69 mmol/L), marked hypokalaemia (2.65 ± 0.77 mmol/L versus 4.32 ± 0.44 mmol/L), and critical hypocalcaemia (1.53 ± 0.74 mg/dL versus 8.88 ± 0.77 mg/dL). Sodium was identified as the primary pathogenic driver in cardiovascular deterioration for genetic heart disease. Correlation analyses demonstrated a significant negative correlation between serum sodium concentration and advancing chronological age ($r = -0.258$, $p < 0.05$), alongside a significant positive correlation between chloride levels and age ($r = 0.246$, $p = 0.05$). Conversely, variations in potassium, calcium, and magnesium displayed non-significant negative correlations with age, while Body Mass Index (BMI) demonstrated no statistically significant association with any measured electrolytes. Pathophysiologically, severe deficiencies in essential intracellular ions—particularly magnesium and potassium—were directly implicated as key contributors to disrupted trans-sarcolemmal electrical signalling and cardiac arrhythmias. From a molecular genetics perspective, maintaining rigorous systemic electrolyte homeostasis is vital to stabilise inherently vulnerable ion channels, particularly in cases involving SCN5A gene mutations which underlie Long QT Syndrome Type 3 and Brugada Syndrome. Ultimately, the study concludes that routine, proactive multi-electrolyte monitoring is a fundamental clinical imperative to mitigate life-threatening arrhythmic events and optimise long-term survival in genetic heart disease cohorts.

Keywords: BMI, Body Mass Index, HD Patients, ACE Angiotensin-Converting Enzyme.

1. Introduction

Electrolyte disturbances are a daily and potentially important complication in patients with heart failure. This may

be due to the pathophysiological changes that happen in heart failure and which lead to activation of neurohumoral systems (Polandic, 2012). Recent evidence demonstrates that mutations in cardiac genes altering ion channel subunits directly impair myocardial electrophysiology, fundamentally predisposing individuals to lethal inherited arrhythmias and severe electrolyte imbalances (Smith et al., 2024). This may be due to the pathophysiological changes that occur in heart failure and which lead to action of neurohumoral, and as a result of complications of treatment with diuretics, cardiac

glycosides, or angiotensin-converting enzyme (ACE) inhibitors (Dimeski et al., 2012).

Pathogenic loss-of-function mutations in SCN5A and KCNQ1 directly predispose cells to systemic electrolyte disturbances and lethal cardiac arrhythmias by impairing myocardial sodium and potassium currents (Satvaldina et al., 2025). Patients with heart failure may develop hypernatraemia due to diminished water and desmopressin secretion, and this may be exacerbated by diuretic therapy (Dimeski et al., 2012). Along with potassium and calcium, magnesium touches cardiovascular function. Magnesium and potassium lacks play an important role in the generation of arrhythmias. Magnesium is essential for maintaining potassium within cells (Budak et al., 2012).

The studies of genetic diseases show, especially cardiovascular diseases or, after surgery, metabolism and stress. This balance can be disrupted, leading to fluctuations in the concentration of free ions in the blood, such as Ca^{2+} , K^{+} , Na^{+} , Cl^{-} , and Mg^{2+} . (Jain et al., 2009). Research indicates that sodium plays a role in maintaining fluid balance and proper circulation within the body. Sodium is essential for preserving osmotic pressure and ensuring water is appropriately distributed among tissues. An imbalance—whether an excess or lack—can disturb these critical functions, potentially imperil gross health (Liu & Dudley, 2023). Potassium serves as a crucial component in the intracellular fluid, functioning as its primary cation. It plays a key role in regulating cellular signalling and enabling muscle responses to stimulation, including the activity of the heart's genetics (Mlambo VC et al. 2024). Potassium imbalance can interfere with neuromuscular function and may even lead to arrhythmias. Similar to sodium, maintaining a steady intake of potassium from whole foods is for preserving proper cellular balance (Teymouri et al., 2022).

Clinically, this is manifested in the form of severe systemic and pulmonary congestion, increased risk of ventricular arrhythmias, prolonged hospitalisations and near doubling of long-term risk of mortality (Bhavanandan et al., 2022).

2. Materials and Methods

Patients with hereditary heart disease were seen at Al-Karama Hospital and Al-Zahraa Teaching Hospital from October 2024 to June 2025, according to their treatment schedules. Samples were collected from the patients with their consent for the study – about 5 cc from patients.

The institutional ethical board's approval was obtained before the trial started, and patients were enrolled in it with their consent and written information.

A total of 68 peripheral blood samples were collected from recently diagnosed patients aged 45-57 years (28 females and 24 males). The control group consisted of individuals who visited the hospital as blood donors, totalling 24 groups (13 females and 11 males). Patients were divided into subgroups according to age. Electrolyte levels in dialysis patients and those with hereditary heart disease (who underwent genetic testing, and their family history was also studied to confirm the genetic predisposition) were divided according to the type of disease. The EXIAS device from Hoffmann-La Roche, Germany, the most advanced device in Iraq, was used to analyse the serum samples extracted from the patients' blood for electrolyte analysis.

Subjects of patients with hereditary heart disease, at the discretion of the attending physician in the endoscopy unit, were included in this study. After explaining study objectives and obtaining written informed consent from all patients, baseline demographic and clinical data were collected from participants by interview and recorded using a study questionnaire. Individuals with atherosclerosis or rheumatoid arthritis were excluded from the study. Another criterion for exclusion is smoking, alcohol intake, and use of antibiotics.

3. Statistical Analysis

All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS), version 22. Continuous variables were tested for normal distribution. All continuous variables were normally distributed; therefore, the data were expressed as mean $\pm$ SD, and all statistical comparisons were made using an independent t-test with $P \leq 0.05$ considered statistically significant.

4. Results and Discussion

4.1 HD Sampling Characteristics

4.1.1 Age

The mean $\pm$ SD of age of HD patients was 56.86 ± 10.43 years with a range of 45–75 years $\pm$ SD, while the mean $\pm$ SD of age of healthy individuals was 55.60 ± 10.38 years with a range of 43–77 years. The age distribution of HD patients was as shown in figure (1):

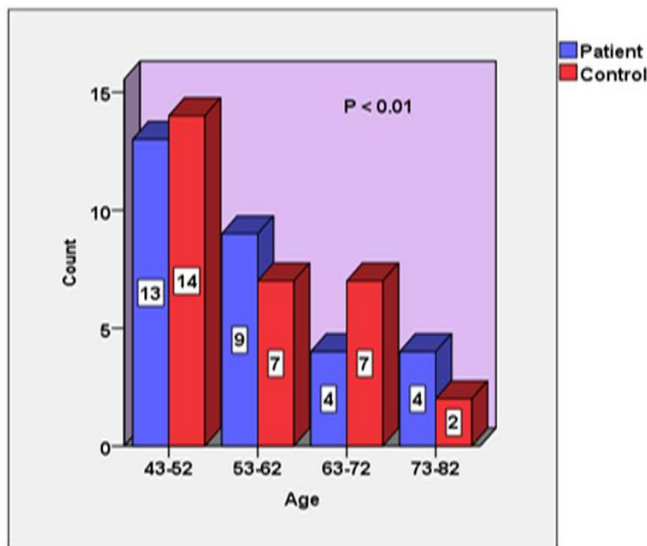


Figure 1. Age distribution of patients and controls with heart genetic diseases

Deficiencies in fluid and electrolytes are a common complication of cardiovascular disease. Fluid and electrolyte imbalance can be jointly life-threatening. Maintaining homeostasis in the ecosystem in terms of fluid and electrolyte balance is critical to achieving optimal cardiac function in all heart patients (Dimeski et al., 2012).

The heart fiasco is because the cardiovascular death rate for morbidity, resulting in more than one million hospitalisations per year in Iraq, and it is the most common hospitalisation among subjects older than 65 years (Polandic et al., 2012).

A recent study by Abdelaal et al. (2025) investigated genetic variants associated with sudden cardiac death (SCD) in young populations and found that inherited phenotypes such as hypertrophic cardiomyopathy (HCM) and dilated

cardiomyopathy (DCM) tend to have acute peaks in younger age groups (<35 years) compared to control populations. The study emphasises that targeted early genetic screening in these specific age brackets is vital for reducing mortality rates associated with severe, early-onset electrical disturbances.

Furthermore, group comparisons between age-matched cohorts have revealed that specific genetic mutations lead to distinct age-related variations in disease severity. In a comparative study of phospholamban (PLN) mutations versus other sarcomeric cardiomyopathies, Afana et al. (2024) highlighted that advancing age is progressively linked to pathological cardiac remodelling and myocardial fibrosis. The age-dependent progression accounts for the late manifestation of severe structural changes in mutation carriers as compared with healthy controls from the same age group not harbouring these genetic alterations (Lewontin et al., 2025).

4.1.2 Body Mass Index

The estimated levels of body mass index for HD patients compared to control (28.75 ± 3.45 vs. 29.06 ± 3.81 ; $p > 0.05$). The BMI distribution of HD patients was as shown in table (1) and figure (2):

Table 1. The means of BMI values for DM patients

Groups	BMI (Mean $\pm$ SD)	P-value
Patients	28.75 ± 3.45 n	$P > 0.05$
Control	29.06 ± 3.81	

*: The difference was significant at $p < 0.05$.

N: non-significant at $p > 0.05$.

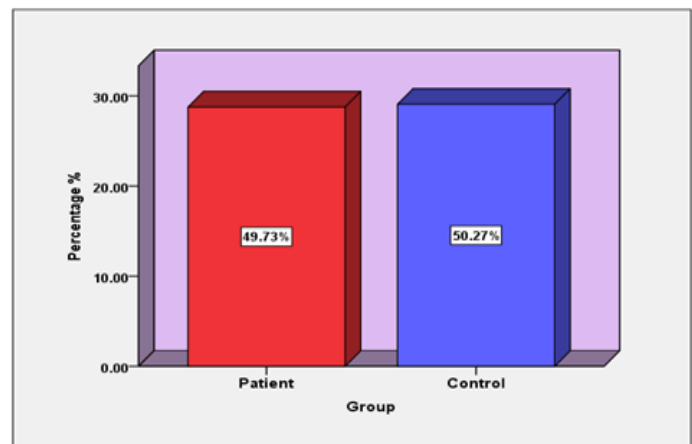


Figure 2. Distribution of Heart genetics disease patients according to body mass index with control group

Electrolyte abnormalities, often in the shadows, result in a significant risk for those who wrestle with heart failure (Teymouri et al., 2022). It's largely tied to the complicated changes within the body during heart failure. This system, the renin-angiotensin-aldosterone system, goes into overdrive by the activation of the sympathoadrenergic system, creating a complex web of responses (van Berkel & Scharnhorst, 2011).

Furthermore, treatments designed to help, such as diuretics, cardiac glycosides, or ACE inhibitors, can further complicate the situation, adding to the risk to patients. Heart failure often brings with it a condition known as hypernatraemia, a situation where sodium levels drop, preventing the body from excreting water effectively (Mlambo et al., 2024).

4.2 Serum Level of electrolytes in HD patients

The mean $\pm$ SD of age of HD patients was 117.49 ± 19.47 years with a range of 45–75 years $\pm$ SD, while the mean $\pm$ SD of age of healthy individuals was 136.00 ± 2.69 years with a range of 43–77 years. The age distribution of HD patients was as shown in table (2), (3) & (4):

Table 2. The means of Sodium (mmol/ l) values for HD patients.

Groups	Sodium (mmol/ l) (Mean $\pm$ SD)	P-value
Patients	$117.49 \pm 19.47^{**}$	P < 0.001
Control	136.00 ± 2.69	

****:** The difference was highly significant at $p < 0.001$.

***:** The difference was significant at $p < 0.05$.

N: non-significant at $p > 0.05$.

Table 3. The means of Potassium (mmol/l) values for HD patients.

Groups	Potassium (mmol/ l) (Mean $\pm$ SD)	P-value
Patients	$2.65 \pm 0.77^{**}$	P < 0.001
Control	4.32 ± 0.44	

****:** The difference was highly significant at $p < 0.001$.

***:** The difference was significant at $p < 0.05$.

N: non-significant at $p > 0.05$.

Table 4. The means of Calcium (mg/dl) values for HD patients.

Groups	Calcium (mmol/ l) (Mean $\pm$ SD)	P-value
Patients	$1.53 \pm 0.74^{**}$	P < 0.001
Control	8.88 ± 0.77	

****:** The difference was highly significant at $p < 0.001$.

***:** The difference was significant at $p < 0.05$.

N: non-significant at $p > 0.05$.

They show study care of the CHF patient includes the recognition and management of these electrolyte disturbances (Rakugi et al., 2021). There's agreement the results from these analysers are interchangeable, as studies using different instruments have shown varying outcomes (Budak et al., 2012; Jain et al., 2009 & Flegar-Mestric & Perkov, 2006). Plasma or serum electrolyte concentrations are the net result of intake, excretion (mainly alimentary and renal), and shifts between extra- and intracellular fluids (Razavi et al., 2010).

4.3 Correlation among Sodium (mmol/ l) and age, BMI, in HD patients.

In table (5) there is a significant correlation between Sodium (mmol/ l) and age, but a non-significant with BMI in patients with HD patients.

There is a negative correlation between Sodium (mmol/ l) concentration and age in patients, and significant at ($r = 0.258$, $p = 0.01$), as shown in Figure (3). The correlation between serum Sodium (mmol/ l) concentration and BMI is positive but not statistically significant ($r = 0.029$, $P = 0.825$) in Figure (3).

Table 5. The correlation among Apelin and age, BMI, in HD patients.

	R	P-value	Type
Sodium	0.258*	0.05	Negative
Age			
Sodium	0.029	0.854	Positive
BMI			

***:** Correlation was significant at $p < 0.05$.

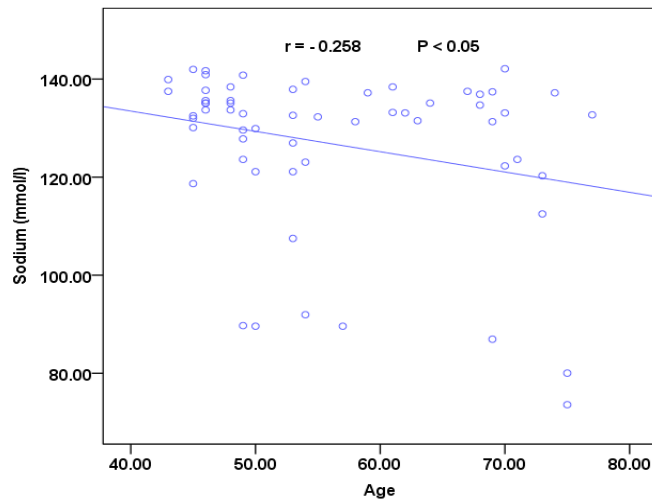


Figure 3. Correlation between serum Sodium (mmol/ l) concentration and age for patient groups

They Show that pathophysiological nexus of age, serum sodium concentrations and cardiovascular genetic disease outcomes. Serum sodium concentrations are homeostatically controlled between 135 mmol/L and 145 mmol/L. This is important for optimal cellular function, plasma osmolality and cardiovascular stability.

A complex, non-linear correlation between increasing chronological age and alterations in serum sodium is found, which has a substantial impact on the morbidity and mortality profiles of cardiovascular patients (Liamis et al., 2013). Age-Dependent Changes in Serum Sodium Regulation Epidemiological cohorts imply that ageing shifts the baseline distribution of serum sodium, mainly because of senescent physiological declines and polypharmacy (Filippatos et al., 2017).

Renal senescence is the ageing process is associated with a gradual decrease in the glomerular filtration rate (GFR) and a loss of cortical nephron mass (Gankam Kengne et al., 2019). The ageing kidney has a blunted response to aldosterone and to natriuretic peptides, impairing the ability to conserve sodium during deficits and to efficiently excrete free water in the event of volume overload. Neuro-Hormonal Dysregulation: Elderly patient cohorts often exhibit a reset osmostat mechanism. Ageing influences the hypothalamic thirst threshold, predisposing people to voluntary dehydration (Liamis et al., 2013). However, there is often a paradoxical

increase in basal secretion of arginine vasopressin (AVP) and older adults are very susceptible to dilutional states induced by water retention (Filippatos et al., 2017).

In cohorts of patients with cardiovascular disease, deviations from the optimal natremic ranges are clearly J- or U-shaped with mortality. Both hypo- and hypernatraemia are major independent prognostics markers of poor outcomes (Rusinaru et al., 2012).

- **Hyponatraemia** (<135 mmol/L) in Cardiovascular Disease Hyponatraemia is the most common electrolyte disorder present in patients admitted to hospital with acute heart failure (AHF) (Bhavanandan et al., 2022). Pathophysiology: In heart failure, diminished effective arterial blood volume leads to significant non-osmotic activation of the sympathetic nervous system (SNS) and the renin-angiotensin-aldosterone system (RAAS). This neurohormonal cascade results in continuous release of AVP via V2 receptors in the collecting ducts of the kidney. Water reabsorption is greater than sodium retention. This leads to dilutional hyponatraemia and hypervolemia (Verbrugge et al., 2015). Clinical Impact on the Failing Myocardium: The extracellular hypotonic environment affects the cardiac trans-sarcolemmal sodium-calcium exchanger (NCX) at the cellular level. This alters the calcium dynamics inside the cell, which impairs the contractility of cardiomyocytes (Gankam Kengne et al., 2019).
- **Hypernatraemia** (>145 mmol/L) in Cardiovascular Disease Hypernatraemia is less common than hyponatraemia but is associated with very high mortality, particularly in frail, elderly cardiac patients. Pathophysiology Hypernatraemia is a hyperosmolar state typically due to unreplaced free water losses (e.g. secondary to high-dose loop diuretic therapies, impaired cognitive access to water or blunted thirst sensations) (Filippatos et al., 2017). Clinical Impact on the Failing Myocardium Hypertonicity causes intracellular dehydration as water follows its osmotic gradient out of myocytes, causing cellular shrinkage

and oxidative stress (Rusinaru et al., 2012). Such excess electrolyte increases endothelial stiffness, inhibits endothelial nitric oxide release and acutely increases systemic vascular resistance (afterload).

Studies show that genetic mutations in cardiac sodium channels (specifically the SCN5A gene) alter intracellular sodium currents (INa), where a gain-of-function mutation leads to Long QT Syndrome Type 3 (LQT3) through prolonged repolarisation (Ackerman & Wilde, 2025; Strachan & Read, 2019).

On the other hand, loss of function mutations in these same channels lead to Brugada syndrome and progressive cardiac conduction disease, which impair depolarisation, greatly increasing the risk of fatal ventricular arrhythmia and sudden cardiac death (Libby et al., 2022; Ackerman & Wilde, 2025).

4.4 Correlation among Potassium (mmol/l) and age, BMI, in HD patients.

In table (6) there is a non-significant correlation between potassium (mmol/l) and age and BMI in patients with HD.

There is a negative correlation between potassium (mmol/l) concentration and age in patients, which is non-significant at ($r = -0.10$, $p = 0.44$), as shown in Figure (4).

The correlation between serum potassium (mmol/l) concentration and BMI is positive but not statistically significant ($r = 0.16$, $P = 0.21$) in figure (4).

Table 6. The correlation among Potassium (mmol/l) and age, BMI, in HD patients.

	R	P-value	Type
Potassium Age	- 0.10	0.44	Negative
Potassium BMI	0.16	0.21	Positive

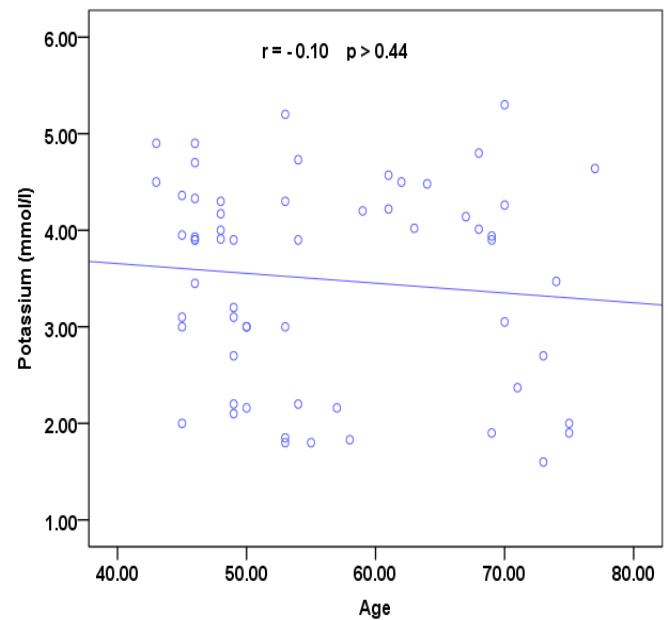


Figure 4. Correlation among serum Potassium (mmol/l) concentration and age for patient groups.

Studies reiterate clearly that potassium plays a vital role as the major cation of intracellular fluid. Thus, one could speculate that hypokalaemia potentiates continued activation of the renin-angiotensin-aldosterone centre in CHF. (Nanda et al., 2015) Potassium also modulates the activity of the sympathetic nervous system; when aloft, potassium enhances the uptake of norepinephrine by sympathetic nerve terminals (Patel, 2009 & Birukov et al., 2016). For these causes, potassium in CHF likely has a main role in thermodynamic regulation and, perhaps, myocardial protection (Dimeski et al., 2012).

Potassium is the main positive ion within cells, critical for neuromuscular cells to respond to stimulants. This element is vital in the body, especially regarding the cardiovascular system, where its concentration significantly impacts the heart muscle's ability to be stimulated, the conduction of electricity, and the heart rate. (Birukov et al., 2016) Nutritional markers and body composition can influence potassium levels independently of BMI and age. (Chow et al., 2008). Therefore, showing maintained meticulous systemic electrolyte homeostasis is crucial for stabilising these genetically vulnerable ion channels to prevent life-threatening arrhythmic events (Libby et al., 2022).

4.5 Correlation among Calcium (mg/dl) and age, BMI, in HD patients.

In table (7) there is a non-significant correlation between calcium (mg/dl) and age and BMI in patients with HD. There is a negative correlation between calcium (mg/dl) concentration and age in patients, which is non-significant at ($r = -0.11$, $p = 0.381$), as shown in figure (5). The correlation between serum calcium (mg/dl) concentration and BMI is positive but not statistically significant ($r = 0.045$, $P = 0.732$) in figure (6)

Table 7. The correlation among Calcium (mg/dl) and age, BMI in HD patients.

	R	P-value	Type
Age	- 0.115	0.381	Negative
BMI	0.045	0.732	Positive

*: Correlation was significant at $p < 0.05$.

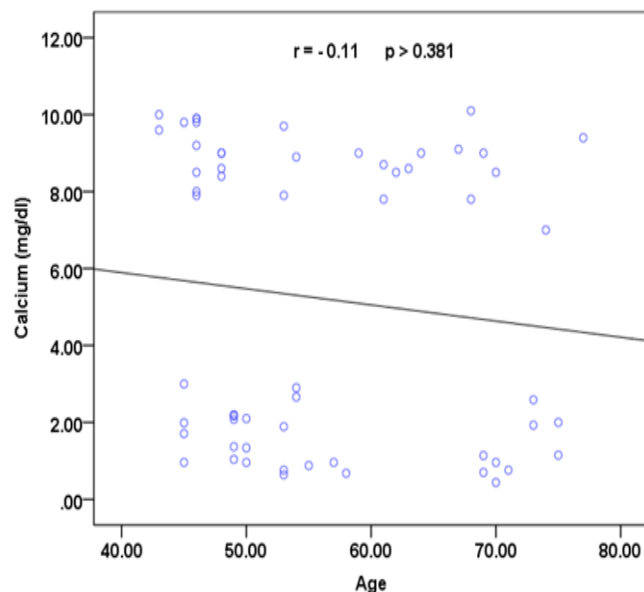


Figure 5. Correlation among serum Calcium (mg/dl) concentration and age for patient groups

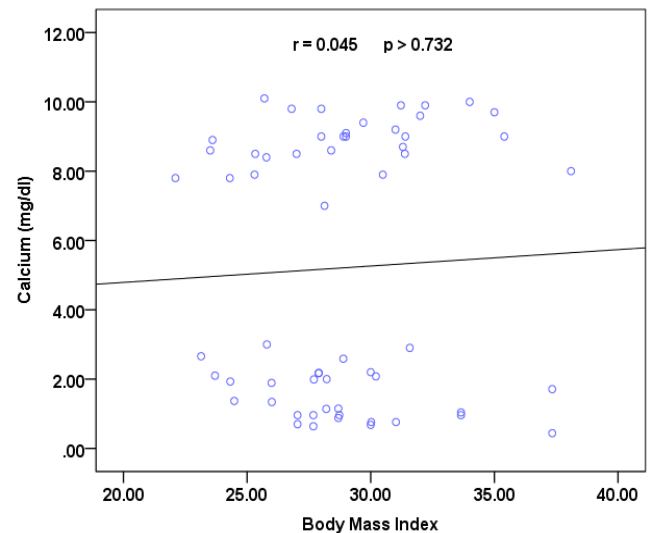


Figure 6. Correlation among serum Calcium (mg/dl) concentration and BMI for patient groups

Calcium is crucial for a number of bodily processes, particularly in the action of blood coagulation (Razavi et al., 2010).

In patients undergoing maintenance haemodialysis (HD), serum calcium (Ca) regulation is a dynamic process governed by altered bone remodelling, dietary intake, and dialysis prescriptions (Nakanishi et al., 2018). High-impact nephrology literature reveals a complex, multi-directional metabolic axis among serum calcium, advancing chronological age, and body mass index (BMI) that profoundly impacts cardiovascular and mineral-bone disorder (MBD) profiles in end-stage renal disease (ESRD) cohorts (Nakanishi et al., 2018).

This metabolic axis operates primarily through two distinct physiological mechanisms:

- **Nutritional and Protein Binding Interference:** Because a substantial fraction of serum calcium binds to albumin, malnourished, low-BMI patients suffering from protein-energy wasting (PEW) frequently exhibit falsely depressed total measured serum calcium due to hypoalbuminemia, necessitating precise albumin-correction formulas to reveal accurate physiological calcium status. Conversely, higher-BMI patients possess superior nutritional reserves, leading to more stable protein-bound calcium fractions.

- Adipokine and Parathyroid Axis Interactivity:**
 Adipose tissue functions as an active endocrine organ; elevated BMI and increased adiposity are correlated with enhanced parathyroid gland sensitivity and altered calcium-sensing receptor (CaSR) expression. Tentori et al. (2014) demonstrate that HD patients exhibit a high vulnerability to vascular calcification when calcium levels fluctuate even slightly. Consequently, large-scale registries underscore that maintaining both serum calcium and BMI within optimal median bands is critical; clinical deviations—specifically the combination of hypercalcaemia and low BMI—strongly accelerate calcific uremic arteriolopathy (calciphylaxis) and elevate cardiovascular mortality risks (Rivara et al., 2015).

4.6 Correlation among Chlorine (mmol/l) and age, BMI, in HD patients.

In table (8) there is a significant correlation between chlorine (mmol/l) and age, while there is a non-significant correlation between chlorine and BMI in patients with HD. There is a positive correlation between chlorine (mmol/l) concentration and age in patients, which is significant at ($r = 0.246$, $p = 0.05$), as shown in Figure 7. The correlation between serum chlorine (mmol/l) concentration and BMI is positive but not statistically significant ($r = 0.134$, $P = 0.306$) in Figure (8).

Table 8. The correlation among Chlorine (mmol/l) and age, BMI, in HD patients.

	R	P-value	Type
Chlorine	0.246 *	0.05	Positive
Age			
Chlorine	0.134	0.306	Positive
BMI			

*: Correlation was significant at $p < 0.05$.

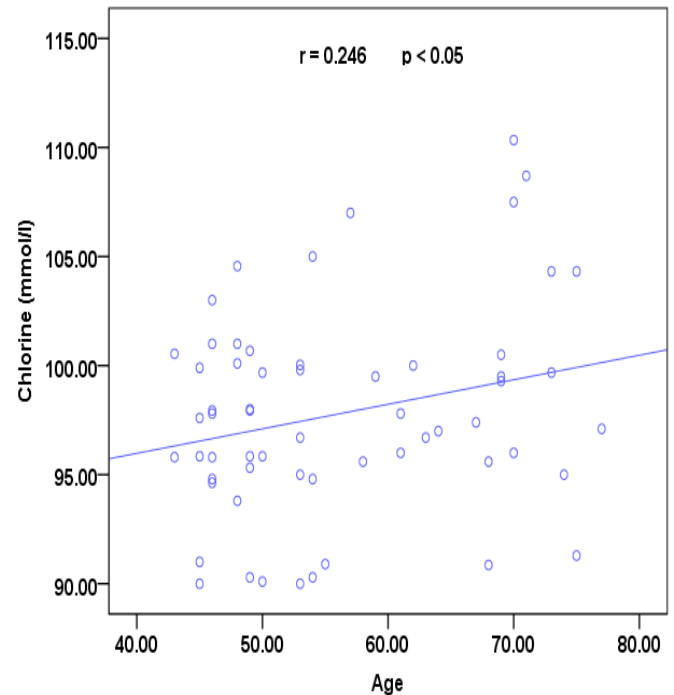
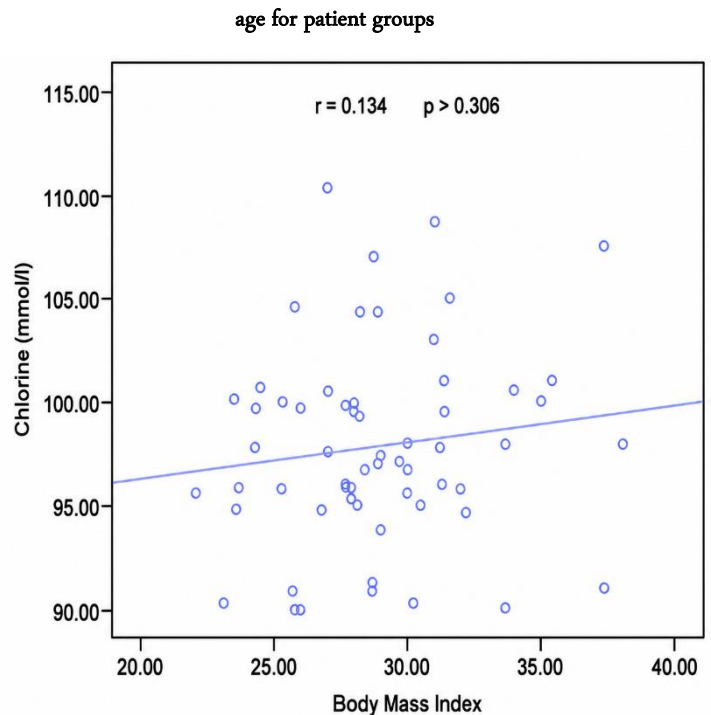


Figure 8. Correlation among serum Chlorine (mmol/l) concentration and BMI for patient groups.

Figure 7. Correlation among serum Chlorine (mmol/l) concentration and

Birukov et al. (2016) found that they have reported 93% mortality in hyperchloraemia cases. A minimum chloride greater than or equal to 110 mEq/L was auxiliary to increased odds of a complicated course (odds ratio, 1.9; 95% CI, 1.1-3.2; $p = 0.023$) and mortality (odds ratio, 3.7; 95% CI, 2.0-6.8; $p < 0.001$). Studies have shown that hypochloremia is more prevalent in elderly patients and is often associated with lower BMI and poor nutritional status (Mandai et al., 2017).

Furthermore, there is a relation between chloride levels and BMI, where patients with lower BMI and lower chloride levels face a higher risk of cardiovascular mortality (Wu et al., 2020).

4.7 Correlation among Magnesium (mg/dl) and age, BMI, in HD patients.

In table (9) there is a non-significant correlation between magnesium (mg/dl) and age and BMI in patients with HD. There is a negative correlation between magnesium (mg/dl) concentration and age in patients, and it is non-significant at $r = -0.32$, $p = 0.81$, as shown in Figure 9.

The correlation between serum magnesium (mg/dl) concentration and BMI is negative but not statistically significant ($r = -0.061$, $P = 0.642$) in Figure (10)

Table 9. The correlation among Magnesium (mg/dl) and age, BMI, in HD patients.

	R	P-value	Type
Magnesium Age	- 0.32	0.81	Negative
Magnesium BMI	- 0.061	0.642	Negative

*: Correlation was significant at $p < 0.05$.

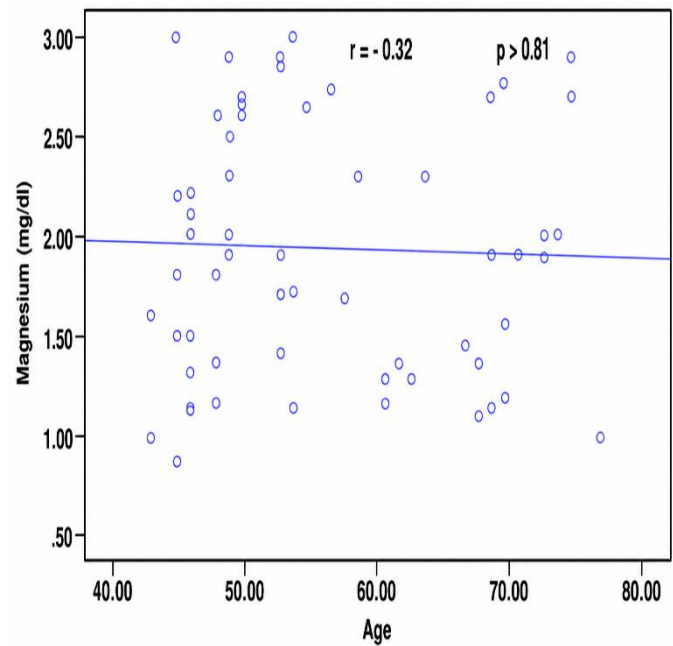


Figure 9. Correlation among serum Magnesium (mg/dl) concentration and age for patient groups.

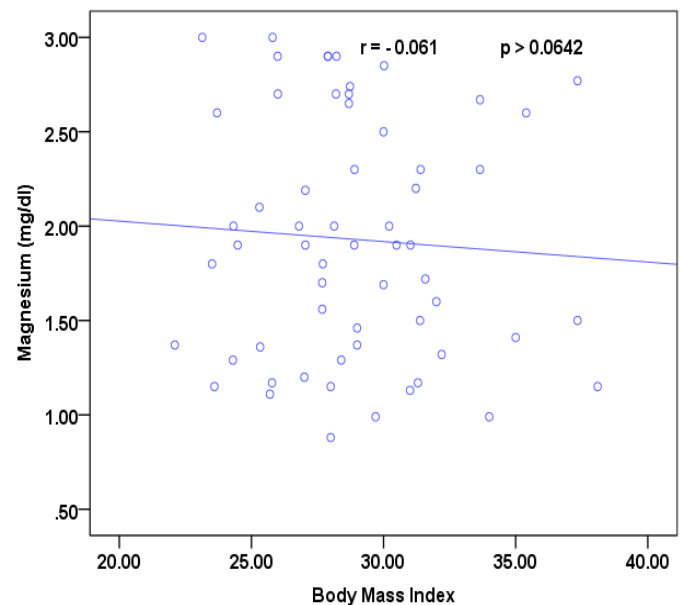


Figure 10. Correlation among serum Magnesium (mg/dl) concentration and BMI for patient groups

Magnesium is crucial for keeping potassium levels in balance within our cells. (Metheny, 2012) Given that magnesium and potassium are predominantly inside our cells, simply testing serum or plasma levels doesn't show a complete portrait of magnesium status. (Naveda & Naveda, 2016) There's a bit when it comes to the prevalence of hypomagnesaemia—a condition characterised by low magnesium levels—among patients who wrestle with chronic heart failure. This controversy has numbers dancing between 7% and 37%. However, patients with heart failure tend to show lower magnesium levels when contrasted to those with healthy hearts (Kwiatkowski et al., 2017).

A study showed that serum magnesium had a strong negative correlation with BMI (Jia et al., 2026). Genetic cardiovascular diseases are often associated with low or pathologically deficient levels of magnesium (Shugaa Addin et al., 2024). The natural electrical protection of the heart is removed by this hypomagnesaemia, resulting in a marked deterioration of the clinical course and precipitation of severe symptoms in patients with inherited cardiac disorders. According to a large-scale real-world clinical analysis by Xu et al. (2024), patients with higher BMI and abdominal obesity exhibit significantly lower serum magnesium levels.

5. Conclusion

This comprehensive study underscores the critical and complex interplay between systemic electrolyte homeostasis and the pathophysiological trajectory of genetic heart diseases. Our biochemical analysis identifies serum sodium as a primary determinant in the onset and progression of these cardiac conditions, demonstrating a significant inverse correlation with advancing chronological age that exacerbates myocardial vulnerability. Crucially, the precise regulation of potassium, calcium, magnesium, and chloride concentrations is indispensable for stabilising genetically susceptible cardiac ion channels. The research highlights those genetic mutations, particularly within cardiac sodium channels such as the SCN5A gene, render patients acutely sensitive to ionic fluctuations; these shifts can precipitate lethal channelopathies, including Long QT and Brugada syndromes, by impairing normal depolarisation and repolarisation phases. Furthermore,

the data reveal that pronounced deficiencies in essential intracellular cations—specifically magnesium and potassium—severely disrupt transmembrane electrical signalling and sarcolemmal integrity. This disruption acts as a primary catalyst for life-threatening ventricular tachyarrhythmias and suppressed myocardial conduction velocities. Demographic and physiological modifiers, notably advanced age and diminished renal senescence, further distort this fragile homeostatic balance, thereby magnifying the clinical risks associated with dysnatremia and elevated serum chloride levels. While body mass index exhibited a statistically non-significant correlation with the primary measured electrolytes, the convergence of poor nutritional status and ionic derangements substantially amplifies cardiovascular mortality. Ultimately, the clinical management of hereditary cardiac conditions mandates rigorous, routine multi-electrolyte monitoring. Proactive therapeutic stabilisation of these vital macro-minerals is a fundamental clinical imperative necessary to neutralise the arrhythmic risks inherent in genetic predispositions, reduce rehospitalisation rates, and optimise the long-term survival of cardiovascular patients.

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