RESEARCH ARTICLE

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Electrocardiogram Changes in Patients with Hyponatremia: A Retrospective Study

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Abstract

Background and Aim: Hyponatremia is defined as a serum sodium level lower than 135 mEq/L. It is the most common clinical electrolyte disorder. Electrolyte disorders are known to affect electrical conduction in the human heart. As the severity of sodium deficiency increases, the clinical picture becomes more severe, but this may not always be directly reflected in the electrocardiogram (ECG). In our study, we aimed to detect common ECG findings seen in patients with hyponatremia.

Materials and Methods: The study was designed retrospectively. Patients who applied to the cardiology and/or nephrology outpatient clinic between January 2020 and 2023, were detected to have hyponatremia in laboratory tests, and had an ECG taken on the same day were included in the study. Changes in the ECG were recorded.

Results: Thirty-four patients were included in the study. When the ECG examinations of the patients were evaluated, P mitrale was detected in 8 patients and bundle branch block (left bundle branch block, right bundle branch block and incomplete bundle branch block), was detected in 7 patients. T-wave changes were frequently observed in hyponatremic patients. The most common cause of hyponatremia in patients was determined to be hypovolemic hyponatremia.

Conclusion: In conclusion, the investigation into ECG changes associated with hyponatremia reveals critical insights into the complex interplay between electrolyte disturbances and cardiac function. While the study identified specific ECG abnormalities like P mitrale and various bundle branch blocks, these findings underscore the necessity for careful interpretation, as they may not be solely attributable to hyponatremia. The lack of distinctive ECG patterns correlating exclusively with sodium deficiency suggests that clinicians should employ a holistic approach when evaluating patients with hyponatremia, considering accompanying medical conditions and pharmacological influences. Ultimately, further research is imperative to explore the underlying mechanisms and enhance the diagnostic utility of ECG in managing electrolyte imbalances effectively.

Keywords: P mitrale, hyponatremia, electrocardiogram, bundle branch blocks

INTRODUCTION

The complexity of electrolyte imbalances in clinical medicine requires comprehensive examination, particularly regarding their impact on cardiovascular health. Hyponatremia, characterized by abnormally low serum sodium levels, is a common and potentially dangerous condition that can lead to various health complications.[1] It is frequently encountered in patients with heart failure (HF), where the interplay between neurohormonal activation and fluid retention further exacerbates sodium deficiency. Alterations in serum sodium levels can induce significant changes in cardiac electrical activity, as can be detected by observable electrocardiogram (ECG) abnormalities.

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Symptoms of patients with hyponatremia range from being asymptomatic to coma. Patients with acute (<48 h) or severe (sodium levels less than 120 mmol/L) hyponatremia may present with serious complications such as dizziness, coma, seizures, respiratory depression, and death. Others may present with only non-specific symptoms such as nausea, vomiting, and headache. Chronic moderate (sodium levels 120-129 mmol/L) and mild (sodium levels 130-134 mmol/L) hyponatremia can cause fatigue, cognitive impairment, gait deficits, falls, impaired bone metabolism, and fractures.[2]

Electrolyte imbalances, particularly those affecting sodium levels, have significant implications for health, especially in patients with chronic conditions. Hyponatremia can arise from a myriad of factors, including excessive fluid intake, renal impairment, or the side effects of medications, particularly in the context of cancer treatment.[3]

Identification of changes in the ECG, such as T-wave changes and conduction defects, in cases of hyponatremia may provide early prediction of clinical deterioration.[4]

There is a complex relationship between hyponatremia and ECG changes, and in some cases it can cause arrhythmias with serious cardiac consequences. ECG changes such as P mitrale, bundle branch blocks and T-wave changes have been described in some studies, and it has been noted that more pronounced ECG changes can be seen at lower sodium levels. These changes may occur due to electrolyte imbalances affecting myocardial repolarization and conduction and may lead to increased susceptibility to fatal arrhythmias.[5]

The evaluation of cardiac complications associated with electrolyte imbalances is crucial to understanding their implications in clinical practice. In the context of hyponatremia, the aim of this retrospective study was to identify ECG changes occurring in affected patients. Hyponatremia is often associated with poor health outcomes and, in some cases, affects populations with pre-existing cardiac conditions. Therefore, it is important to detect these changes early. The aim of the study is not only to enable early recognition of ECG changes in hyponatremia, but also to increase awareness and improve patient management and prevent further complications.

METHODS

This retrospective study included patients who presented to the cardiology and/or nephrology outpatient clinic between January 2020 and January 2023, were diagnosed with hyponatremia, and had their ECG taken on the same day. Data were collected from patient records, specifically targeting those diagnosed with hyponatremia, defined as serum sodium levels less than 135 mEq/L. The study included 34 patients, mostly older adults, with a significant prevalence of chronic health conditions such as HF, which is known to complicate the clinical picture of hyponatremia. It was planned to evaluate the possible effects of hyponatremia by detecting all abnormal changes in the ECG.

The selection of participants for the study on ECG changes in patients with hyponatremia involved stringent inclusion and exclusion criteria to ensure valid and reliable results. Individuals aged 18 and older, with confirmed hyponatremia defined as serum sodium levels less than 135 mEq/L, were included to create a focused cohort that accurately reflects the population affected by this electrolyte imbalance. Furthermore, patients exhibiting significant comorbid conditions, such as chronic kidney disease (CKD) or cardiac disorders, were scrutinized to assess their impact on ECG results, which aligns with findings suggesting that underlying health issues complicate hyponatremia and its cardiac implications. Exclusion criteria eliminated those with psychiatric illnesses or previous electrolyte disorders to avoid confounding variables, thereby enhancing the study's power and ensuring that the captured ECG changes were indeed reflective of hyponatremia itself. The study protocol was reviewed and approved by the Ethics Committee of Hatay Mustafa Kemal University (decision number:19, date:12.10.2023).

Statistical Analysis

The SPSS 20 package program was used to analyze statistical data for evaluation. Descriptive analytic methods were used in this study. Data statistics, number, and percentage distributions were analyzed using the chi-square and Mann-Whitney U tests. A P of <0.05 was considered significant.

RESULTS

This study included 34 patients, of whom 20 (58.5%) were women and 14 (41.5%) were men. The average age of the patients was 67.5±13.0 (34-94) years. Hypertension was present in 18 (52.9%) of the patients; CKD in 13 (38.2%) of the patients, with four undergoing hemodialysis; coronary artery disease in 7 (20.5%) of the patients. HF was diagnosed in 5 (14.7%) patients. Twenty-one of the 34 patients included in the study were aged 65 years and older. When the etiologies of hyponatremia were investigated, hypervolemic hyponatremia was found in 10 patients, hypovolemic hyponatremia was found in 19 patients (2 of whom had malnutrition), and euvolemic hyponatremia was found in five patients.

One patient had hypokalemia. However, the ECG was found to be normal in this patient. Potassium levels were increased in two patients (5.5 mmol/L). These patients did not exhibit any signs of hyperkalemia, such as T peaking and QRS widening. Furthermore, twelve (35%) patients were using diuretics (6 of them were using loop diuretics, while the others were using thiazides). Notably, patients with hypervolemic hyponatremia

are generally associated with HF and/or CKD. Eight of the hypervolemic hyponatremia patients (ten overall) had accompanying CKD, with five of these patients having HF and three having HF and CKD. Four of the patients had inappropriate antidiuretic hormone (ADH) syndrome (12.5%). Twelve patients were taking diuretics (35.2%). The demographic, laboratory, and ECG results of the patients are shown in Table 1.

When the ECGs of the patients were evaluated, P mitrale was found in ten patients, and bundle branch block was detected in eight patients (left bundle branch block in four patients, right bundle branch block in three patients, and incomplete bundle branch block in one patient). Two patients experienced atrial fibrillation (AF) (this finding was confirmed by information in the patients' files). One patient had supraventricular tachycardia at the time of admission. Other patients were in the sinus rhythm. T flattening was found in five patients. T negativity was found in thirteen patients. However, the regions of these changes varied from patient to patient (in some patients, they were seen in D1-aVL, whereas in others, they were in the chest leads). ECG changes according to hyponatremia levels are shown in Table 2.

None of the patients had serious complications such as neurological deterioration or death. Twenty of the thirty-four patients had general weakness and headache-like complaints, while six patients had nausea. These symptoms were seen in all hyponatremia patients, regardless of sodium levels.

DISCUSSION

Hyponatremia has been associated with poor outcomes in a variety of clinical settings. This situation can have negative consequences on patients with HF, coronary artery disease, heart valve disease, elderly patients, patients who have undergone cardiac surgery, and patients with renal dysfunction. The pathogenesis of sinus arrest or cardiac conduction defects caused by hyponatremia has yet to be elucidated, and there is insufficient information in the literature regarding this condition. Clinical observations have shown that a low extracellular sodium concentration can decrease the depolarization of the cardiac action potential and reduce its amplitude in the atrioventricular (AV) node. Furthermore, low sodium levels in the fluid perfusing isolated cardiac muscle can reduce contraction frequency, reduce excitability, and reduce conduction velocity.[6]

Hyponatremia caused by fluid retention increases atrial wall tension, which can lead to atrial arrhythmias.[7] In our study, supraventricular tachycardia was found in one patient. However, the patient had previously experienced palpitations similar to those described. There was insufficient evidence to directly link this condition with hyponatremia.

Severe symptomatic hyponatremia (serum sodium of ≤120 mEq/L) can lead to life-threatening or fatal complications, such as cerebral edema and permanent neurological disability, due to osmotic demyelination.[8] In our study, 20 patients (58.8%) had severe hyponatremia (serum sodium of ≤120 mEq/L). There were no neurological events reported in any patient.

In patients with congestive HF, the number of ventricular premature beats was found to be equally related to the severity of hyponatremia and hypokalemia. Mouallem et al.[9] described three patients who developed reversible cardiac conduction defects associated with transient hyponatremia. During hyponatremia, two of these patients had complete or second-degree AV block (both patients had preexisting heart disease and were taking antiarrhythmic drugs). The other patient developed first-degree AV block during hyponatremia. Second-degree AV block improved with treatment, while first-degree AV block persisted after treatment of hyponatremia. Kottwitz et al.[10] reported a first-degree AV block due to hyponatremia in a 76-year-old male patient, who complained of malnutrition, dysarthria, disorientation, weakness, and fatigue, and had a history of alcohol use and dementia. It was shown that a patient who developed first-degree AV block, followed by second and third-degree AV block, during an attack of severe hyponatremia (serum sodium 98 mmol/L), experienced AV block conditions that were completely reversed once the serum sodium level was corrected.

Previous studies have shown that changes similar to the Brugada pattern can occur in patients with hyponatremia. Brugada syndrome is a genetic condition that increases the risk of ventricular fibrillation and sudden cardiac death in a structurally normal heart. The Brugada type 1 ECG pattern can occur independently of the actual syndrome, and this clinical phenomenon is commonly known as the Brugada phenocopy. A 49-year-old male patient admitted to the emergency department with delirium (due to his use of thiazide diuretics and angiotensin-converting enzyme inhibitors for hypertension) was diagnosed with antihypertensive drug-induced hypovolemic hyponatremia (serum sodium 108 mmol/L, low serum osmolality, high urine osmolality, and urine sodium <20 mmol/L). On admission, the patient's ECG revealed a sloping ST-segment elevation in leads V1 and V2, with high J-dots indicating a Brugada type 1 pattern. The patient improved after treatment. It was believed that his findings were the result of antihypertensive medication. He was discharged after his antihypertensive medications were changed, and the incident did not occur again.[11] In our study, no changes in this pattern were observed in any patient.

The most common causes of hyponatremia in patients with HF are autonomic imbalance caused by diuretic use, sympathetic nervous system response, and renin-angiotensin

Table 1: Demographic, laboratory, and ECG findings of the patients

Case	Gender/age	Serum/urine Na	Serum K	Thyroid hormone/ cortisol	Additional diagnosis	Hyponatremia etiology	ECG finding
1	M/64	102/171	4.5	N/N	COPD	Inappropriate ADH	NSR, 75/min
2	M/69	124	5.1	N/N	HT	Diuretic use (Thiazide)	NSR, 1mm ST in d2-d3-avf
3	M/68	121	5.5	N/N	DM, CKD	Hypervolemic hyponatremia	NSR, p mitrale, 50/min
4	F/70	108/22	3.8	N/N	DM, HT	Diuretic use (Loop), dehydration	NSR, left bundle branch block, frequent VES (Quadrigemine)
5	F/84	124/34	5.1	N/N	Heart failure, HT, AF	Hypervolemic hyponatremia	AF, d1-avl T (-), 72/min
6	M/64	124/12	3.8	N/N	DM, HT, CKD	Hypervolemic hyponatremia	NSR, 90/min, P mitrale
7	F/58	104/10	3.6	N/N	No features	Dehydration	NSR, 106/min
8	M/48	124	4.2		CKD	Hypervolemic hyponatremia	NSR, 71/min, right bundle branch block, d1-d2 T (-), p mitrale
9	F/63	122/31	4.3	N/N	HT, kidney transplant	Diuretic use (thiazide)	NSR, 86/min
10	M/63	113	4.5		AKI	Dehydration	NSR, 100/min, d3-avf T (-)
11	M/61	119	4.8	N/N	Heart failure, CKD	Hypervolemic hyponatremia	NSR, Left bundle branch block, p mitrale, d1-avl T (-)
12	F/80	128/27	4.9	N/N	HT	Diuretic use (thiazide)	NSR, T (-) in V1, 1 mm ST depression in V6
13	F/72	111/23	5.2	N/N	DM, HT	Dehydration	NSR, 75/min, T (-) at V6
14	F/80	116/54	3.8	N/N	DM	Inappropriate ADH	NSR, 62/min, T (-) in V4-V6
15	F/94	111	2.8	N/N	DM, HT	Diuretic use (loop)	NSR, 85/min
16	M/67	127	4.3	N/N	DM, HT, CKD, AF	Hypervolemic hyponatremia	AF, ıncomplete left bundle branch block, VES
17	F/53	116/87	4.7	N/N	HT	Diuretic use (thiazide)	NSR, 71/min
18	F/52	105/38	4.2	N/N	Bronchiectasis, CKD	Diuretic use (loop)	NSR, 70/min
19	F/52	119/18	4.3	N/N	Anorexia nervosa	Dehydration	NSR, T (-) in V1
20	F/71	123/50	4.2	N/-	HT	Diuretic use (thiazide)	NSR, p mitrale
21	M/84	123/38	3.6	N/N	HT, gallbladder cancer	Inappropriate ADH	NSR, right bundle branch block, p mitrale
22	F/32	125	4.2	N/N	ABH	Dehydration	NSR, diffuse T flattening
23	M/79	119/12	5.0	N/N	HT, CKD	Diuretic use (loop), gastroenteritis	NSR, 52/min
24	F/60	119/41	4.0		HT, CKD	Diuretic use (loop)	NSR, 72/min, d1-avL T flattening
25	M/72	114/13	5.5	N/-	CKD, colon cancer	Malnutrition	NSR, 75/min, p mitrale, aVL'de T (-)
26	F/75	123/33	4.0	N/N	HT, CKD	Diuretic use (loop)	NSR, 75/min, p mitrale, T (-) in aVL
27	F/73	105/29	2.7	N/N	HT, COPD	Malnutrition	NSR, 80/min, p mitrale, T (-) in d1-aVL

Table 1: Continued

Case	Gender/age	Serum/urine Na	Serum K	Thyroid hormone/ cortisol	Additional diagnosis	Hyponatremia etiology	ECG finding
28	F/50	118	4.7	N/-	CKD	Hypervolemic hyponatremia	NSR, 90/min, p mitrale, T (-) in d1-aVL
29	F/65	113	4.7	N/-	Heart failure, HT, CKD	Hypervolemic hyponatremia	SVT, 114/min, right bundle branch block, 1 mm ST depression in v4-v6, T(-) in v1-v3 and aVL
30	F/65	124/65	4.8	N/-	DM, HT, CKD, hypothyroidism	Diuretic use (thiazide)	NSR, SVES, 65/min
31	M/84	119	4.2	N/-	Heart failure, CKD	Hypervolemic hyponatremia	NSR, left bundle branch block, T (-) in D1-aVL, q(+) in the inferior
32	F/82	118	4.7	N/-	Heart failure	Hypervolemic hyponatremia	NSR, Diffuse ST depression, ST elevation in V1
33	M/68	128/32	4	N/-	No features	Inappropriate ADH	NSR, 98/min, T flattening in aVL
34	M/82	111/90	4.0	N/N	Lung cancer	Inappropriate ADH	NSR, 82/min, T flattening in aVL

F: Female, M: Male, ECG: Electrocardiogram, COPD: Chronic obstructive pulmonary disease, ADH: Antidiuretic hormone, NSR: Normal sinus rhythm, HT: Hypertension, DM: Diabetes mellitus, CKD: Chronic kidney disease, AF: Atrial fibrillation, AKI: Acute kidney injury, ABH: Acute bronchitis

Table 2: The table shows the accompanying ECG changes according to the levels of hyponatremia. The numbers in the columns indicate the number of patients

Serum Na (mEq/L)	T-wave alternans	Atrial fibrillation	LBBB	RBBB	P mitrale
120 and higher	7	2	1	2	6
110-119	13	None	2	1	3
100-109	1	None	1	None	1

ECG: Electrocardiogram, LBBB: Left bundle branch block, RBBB: Right bundle branch block

system activation, as well as the subsequent neurohormonal response. The primary cause of hyponatremia associated with HF is increased non-osmotic release of ADH as a result of the reduced circulating effective volume.[12]

Although the rate of AF was found to be higher in patients with hyponatremia than in patients with normonatremia, logistic regression analysis did not reveal a direct relationship between hyponatremia and AF.[13] However, for the first time in the literature, hyponatremia has been shown to be independently associated with an increased risk of AF.[14] Another study found a higher incidence of AF and diabetes mellitus in patients with hyponatremia. Fasting glucose, mineralocorticoid receptor antagonists, and digoxin use were found to be higher in the hyponatremia group than in the normonatremia group. However, no correlation was found between sodium levels and AF in patients with HF with preserved ejection fraction. The slowing of the sinoatrial nodal rate caused by low sodium and potassium levels may contribute to an increased risk of AF during hyponatremia or hypokalemia.[15] In our study, AF was found in two patients; however, these patients had pre-existing AF (as confirmed by reviewing their previous records), which was not associated with hyponatremia. In our study, five patients had hyponatremia associated with HF. Hypervolemic hyponatremia was found in each of them.

Hyponatremia may be associated with cardiac conduction abnormalities and some ECG changes. However, with current knowledge, direct ECG assessment cannot be used to determine whether a patient has hyponatremia. Recognizing diseases associated with hyponatremia and identifying conditions that may cause it, such as medication use, can provide early warning signs for the prevention and diagnosis of this electrolyte disorder.

Hyponatremia may prolong the hospital stay and increase mortality. Hyponatremia is especially common in patients with HF who are using diuretics and in the elderly, and therefore, more care should be taken in these patients. Although our study findings do not identify specifics to hyponatremia, they may be useful in taking precautions, especially in patients in the risk group. The heterogeneity of the findings

detected in ECGs supports the possibility that this condition may also occur due to other concomitant heart diseases. However, the retrospective nature of our study and the lack of echocardiographic evaluations of the patients make it difficult to evaluate this relationship. Therefore, it should be evaluated in larger patient groups to clarify its direct relationship with hyponatremia.

Early diagnosis with ECG can contribute to disease management when the possible risks associated with hyponatremia are evaluated. Therefore, methods other than laboratory measurements can be used to detect ECG changes that may occur due to hyponatremia. Similar to previous studies, the current study shows that physician evaluation with ECG alone is not sufficient to diagnose hyponatremia. In addition, the inadequacy of ECG in the diagnosis of hyponatremia emphasizes the need for the development of new methods. Machine learning-based approaches may increase the success of early diagnosis in patient groups where sufficient information about hyponatremia cannot be obtained through ECG evaluation. However, further studies are needed before they can be used in clinical settings.

Future research is needed to understand the complexities that constitute ECG changes in patients with hyponatremia. It is necessary to investigate how low sodium levels affect cardiac electrophysiology, particularly given the high prevalence of ECG abnormalities in these patients. The retrospective nature of the current study highlights the necessity of prospective, longitudinal studies that capture a broader demographic, allowing for more generalized conclusions about cardiac implications in different health conditions. Future studies with larger sample sizes may deepen the understanding of how hyponatremia affects cardiac health and guide clinical practice for monitoring high-risk populations.

Identification and management of ECG changes in patients with hyponatremia is important, especially given their potential to affect cardiac function. In this retrospective study, a number of ECG abnormalities were noted in patients with serum sodium levels less than 135 mEq/L, highlighting their risk for future arrhythmias and poor clinical outcomes. Clinicians should be vigilant in monitoring sodium levels, especially in high-risk populations such as those with preexisting heart conditions or CKD. Furthermore, the association of severe hyponatremia with cardiac conduction defects highlights the need to include comprehensive electrolyte assessments in routine cardiovascular assessments. By recognizing the interaction between hypoosmolality and cardiac health, healthcare providers can implement early interventions that can significantly improve patient prognosis and prevent adverse events related to arrhythmias.

The findings of this retrospective study underscore the critical interplay between electrolyte balance and cardiac function, specifically focusing on instances of hyponatremia. A notable percentage of the 34 patients observed displayed ECG abnormalities, with conditions such as P mitrale and T-wave changes being present in those with serum sodium levels less than 135 mEq/L. While no severe neurological events were directly linked to these changes, the connection between electrolyte imbalances and arrhythmias remains a compelling area for further exploration, particularly given that hyponatremia is prevalent in individuals with other cardiac conditions, as highlighted in the study. It is crucial to consider that, despite the documented impacts of hyponatremia on cardiac action potentials, the study's data suggest, a lack of definitive correlation with arrhythmogenic events, which should be further examined in larger cohorts. The necessity for ongoing monitoring and evaluation of ECG changes in patients at risk of hyponatremia cannot be overstated, as it could significantly affect clinical outcomes and management strategies.

The investigation into ECG changes in patients suffering from hyponatremia yielded several significant findings that underscore the condition's impact on cardiac function. In a sample population averaging 67.5 years old, a notable prevalence of comorbidities, such as CKD and HF, was observed. Key ECG abnormalities included P mitrale, bundle branch blocks, and various T-wave changes, indicating that hyponatremia can interfere with cardiac action potentials and provoke arrhythmias. Despite earlier studies suggesting a link between severe hyponatremia and cardiac conduction disturbances, this study found no direct association between severe hyponatremia and neurological events in patients with significant electrolyte imbalances. Moreover, among 20 patients with serum sodium levels at or below 120 mEq/L, ECG results did not reflect expected conduction defects, which resonates with findings that the ECG alone may not suffice in diagnosing hyponatremia-associated changes. Overall, these results highlight the complexity of hyponatremia's arrhythmogenic potential and call for increased awareness in clinical practice.

The relationship between electrolyte imbalances and cardiac function is critical for understanding the ECG changes observed in patients with hyponatremia. In this context, hyponatremia, defined as a serum sodium level below 135 mEq/L, can produce significant alterations in the cardiac electrical conduction system, which are reflected in the ECG patterns. Notably, key abnormalities such as P mitrale, bundle branch blocks, and T-wave changes were prevalent among patients studied, corroborating findings from (Table 2). Moreover, while severe hyponatremia has been implicated in leading to various arrhythmias, the study emphasizes the absence of direct links between these instances and AF in

affected patients. Such findings align with previous research suggesting that ECG may not consistently reflect electrolyte levels, heightening the importance of vigilant monitoring and diagnostic clarity. Thus, the comprehensive interpretation of ECG changes emerges as essential for the management of hyponatremia and its potential cardiac consequences.

Study Limitations

The findings of the current study on ECG changes in patients with hyponatremia highlight significant limitations that must be considered when interpreting the data. First, the retrospective design inherently limits the ability to establish causative relationships between observed ECG changes and hyponatremia levels. Furthermore, the study's small sample size, comprising only 34 participants, restricts the generalizability of its conclusions, particularly when considering the diverse clinical backgrounds of the patients, as described in. Additionally, the lack of follow-up ECG data post-treatment diminishes the understanding of potential reversibility, of the observed changes. Lastly, while the analysis does contribute valuable insights, it must be contextualized within a broader scope of research, which includes studies on cardiac conduction abnormalities. A more comprehensive approach involving larger, prospective studies is necessary to validate the findings.

CONCLUSION

In reviewing the implications of the findings from this study, it becomes evident that abnormal sodium levels can have significant cardiac effects, warranting further clinical attention. The correlations highlight the need for thorough monitoring of ECGs in patients exhibiting signs of hyponatremia, particularly those with underlying cardiovascular conditions. While our study did not identify direct links between severe hyponatremia and significant arrhythmias, existing literature suggests that electrolyte imbalances may contribute to conduction abnormalities, underscoring the importance of proactive management. Clinicians must remain vigilant in recognizing the potential for long-term consequences associated with these cardiac changes, as even mild hyponatremia can precipitate serious health issues. This research reinforces the necessity for larger, prospective studies to enhance our understanding and improve patient outcomes effectively.

Future research should focus on elucidating the precise mechanisms by which hyponatremia affects ECG changes, as current studies have emphasized correlations rather than causal relationships. In particular, examining the interaction between varying degrees of hyponatremia and specific ECG findings may provide valuable information regarding patient prognosis and management strategies. Furthermore, longitudinal studies evaluating the effects of treatment interventions on both sodium levels and ECG findings may increase our understanding of potential synergies in therapeutic approaches. Consequently, comprehensive multicenter studies are necessary to validate these emerging hypotheses and guide clinical practice.

Ethics

Ethics Committee Approval: The study protocol was reviewed and approved by the Ethics Committee of Hatay Mustafa Kemal University (decision number:19, date:12.10.2023).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Concept: U.I., C.H., Design: U.I., C.H., Data Collection or Processing: U.I., C.H., Analysis or Interpretation: U.I., C.H., Literature Search: U.I., C.H., Writing: U.I., C.H.

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REFERENCES

1. Rondon H, Badireddy M: Hyponatremia. In. StatPearls. StatPearls Publishing, Treasure Island; 2023.
2. Christopoulou E, Liamis G, Naka K, Touloupis P, Gkartzonikas I, Florentin M. Hyponatremia in patients with heart failure beyond the neurohormonal activation associated with reduced cardiac output: a holistic approach. Cardiology. 2022;147:507-20.
3. De las Peñas R, Escobar Y, Henao F, Blasco A, Rodríguez CA; Spanish Society for Medical Oncology. SEOM guidelines on hydroelectrolytic disorders. Clin Transl Oncol. 2014;16:1051-9.
4. Nakazone MA, Otaviano AP, Machado MN, Bestetti RB. Does left ventricular reverse remodeling influence long-term outcomes in patients with Chagas cardiomyopathy? Cardiol J. 2022;29:44-52.
5. Kapłon-Cieślicka A, Balsam P, Ozierański K, Tymińska A, Peller M, Galas M, *et al.* Resting heart rate at hospital admission and its relation to hospital outcome in patients with heart failure. Cardiol J. 2014;21:425-33.
6. Rosner MH. Hyponatremia in heart failure: the role of arginine vasopressin and diuretics. Cardiovasc Drugs Ther. 2009;23:307-15.
7. Zou S, Zhang Q, Gao S, Luo M, Gan X, Liang K. Electrocardiogram manifestations of hyponatraemia. Cardiovasc J Afr. 2022;33:98-100.
8. Sterns RH. Treatment of severe hyponatremia . Clin J Am Soc Nephrol. 2018;13:641-9.
9. Mouallem M, Friedman E, Shemesh Y, Mayan H, Pauzner R, Farfel Z. Cardiac conduction defects associated with hyponatremia. Clin Cardiol. 1991;14:165-8.
10. Kottwitz J, Akdis D, Duru F, Heidecker B. Severe hyponatremia leading to complete atrioventricular block. Am J Med. 2016;129:243-4.
11. Ramsaroop K, Seecheran R, Seecheran V, Persad S, Giddings S, Mohammed B, *et al.* Suspected hyponatremia-induced Brugada phenocopy. Int Med Case Rep J. 2019;12:61-5.

12. Rodriguez M, Hernandez M, Cheungpasitporn W, Kashani KB, Riaz I, Rangaswami J, *et al*. Hyponatremia in heart failure: pathogenesis and management. Curr Cardiol Rev. 2019;15:252-61.

13. Akyüz A, Baykız D, Gür DÖ, Gökçek S, Efe MM, Alpsoy Ş. Role of sodium levels on atrial fibrillation in heart failure: active player or a bystander? Arq Bras Cardiol. 2022;118:712-8.

14. Cavusoglu Y, Kaya H, Eraslan S, Yilmaz MB. Hyponatremia is associated with occurrence of atrial fibrillation in outpatients with heart failure and reduced ejection fraction. Hellenic J Cardiol. 2019;60:117-21.

15. Rafaqat S, Rafaqat S, Khurshid H, Rafaqat S. Electrolyte's imbalance role in atrial fibrillation: pharmacological management. Int J Arrhythm. 2022;23:1-10.