Original Article

Correlational Analysis of Electrolytes Imbalance Between Subclinical Hypothyroidism (SHO) And Subclinical Hyperthyroidism (SHE) at a Tertiary Care Hospital

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Abstract

Background: Thyroid hormones are essential in keeping the metabolism and electrolytes at the right level. The condition is known as subclinical thyroid dysfunction, characterised by abnormal serum thyroid-stimulating hormone (TSH) levels despite normal circulating thyroid hormone levels. Even though overt thyroid disorders are well understood to result in electrolyte imbalances, there is limited information on electrolyte imbalance in the case of tertiary care cases of subclinical hypothyroidism (SHO) and subclinical hyperthyroidism (SHE). These delicate biochemical changes should be recognised, as even minor changes in electrolytes can contribute to morbidity and affect clinical practice. The study aims to correlate and analyse electrolyte imbalance between subclinical hypothyroidism (SHO) and subclinical hyperthyroidism (SHE) at a tertiary care hospital. **Material and Methods:** The analytical cross-sectional study was conducted at the Department of Biochemistry, IMS and SUM Hospital, Campus - II, Bhubaneswar, Odisha (India), and included 28 adult patients presenting with subclinical thyroid dysfunction. The identification of patients into SHO (n = 66) and SHE (n = 62) was based on thyroid function tests. Measurement of serum TSH, free thyroxine (FT4), and free triiodothyronine (FT3) was done by automated immunoassays. Standard automated electrolyte analysers were used to measure the serum electrolytes, sodium, potassium, chloride, calcium, and magnesium. Demographic and clinical records were collected, and patients with underlying conditions or medications that could alter their electrolyte balance were excluded. **Results:** Subclinical hypothyroidism showed significantly lower mean serum sodium (134.62 ± 3.18 mEq/L), chloride (100.84 ± 3.56 mEq/L), calcium (8.68 ± 0.54 mg/dL), and magnesium (1.74 ± 0.26 mg/dL) compared to subclinical hyperthyroidism ($p < 0.001$). SHO showed a highly significant difference in serum potassium, whereas SHE showed a higher incidence of hypokalaemia (29.03%). In SHO, hyponatremia, hypocalcemia, and hypomagnesemia was common. The correlations between TSH and the concentrations of sodium, calcium, and magnesium showed strong negative associations, whereas the correlation with potassium showed a positive association. **Conclusion:** Subclinical thyroid dysfunction relates to major and electrolyte imbalances. Thyroid disorders with subclinical conditions can be identified and managed at an early stage by routine electrolyte evaluation in patients.

Keywords: Subclinical Hypothyroidism; Subclinical Hyperthyroidism; Electrolyte Imbalance; Thyroid-Stimulating Hormone.

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Introduction

Thyroid hormones have pleiotropic actions on cellular metabolism and organ system physiology, and slight changes in thyroid activity can affect homeostatic processes that are not readily apparent until overt disease develops. Subclinical thyroid dysfunction represents an early biochemical stage in which serum thyroid-stimulating hormone (TSH) is abnormal while circulating free thyroxine (FT4) (and typically free triiodothyronine [FT3]) remains within the laboratory reference range.[1] This entity is clinically important because it is common in routine practice, frequently detected during evaluation for nonspecific symptoms or comorbidity workups, and may progress to overt thyroid disease or contribute to systemic complications if unrecognized.[2] Electrolyte balance is a foundational determinant of cardiovascular stability, neuromuscular excitability, renal function, and acid–base equilibrium. Minor changes in serum sodium, potassium, chloride, calcium, or magnesium can precipitate symptoms ranging from fatigue and cramps to arrhythmia, seizures, and altered sensorium. Thyroid disorders have long been linked with metabolic and electrolyte derangements through changes in tissue oxygen demand, renal perfusion, tubular transporter expression, and hormonal cross-talk involving the renin–angiotensin–aldosterone system and vasopressin-mediated water handling.[3] Although pronounced electrolyte abnormalities are

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classically associated with overt thyroid states, accumulating evidence indicates that electrolyte shifts may occur even in subclinical disease—often subtly, but with potentially meaningful clinical consequences in predisposed individuals such as older adults, patients with comorbid hypertension or diabetes, or those exposed to medications that alter renal solute handling. Sodium and water homeostasis is among the most discussed interfaces between thyroid physiology and electrolyte regulation. Hypothyroid states have been associated with impaired free-water excretion and altered renal sodium handling, mechanisms that can predispose to dilutional hyponatremia under certain clinical contexts. The biologic rationale includes reduced cardiac output and renal blood flow, diminished glomerular filtration, and neurohormonal adaptations that favour water retention. Importantly, the causal strength of the association between hypothyroidism and hyponatremia has been debated, but mechanistic pathways—particularly those involving renal tubular function and water clearance—support the plausibility that thyroid status can modulate serum sodium, especially in settings where additional stressors are present (e.g., reduced solute intake, diuretics, intercurrent illness).[4] Within SHO, where FT4 remains normal, the magnitude of sodium disturbance may be smaller than in overt disease. Still, it remains clinically relevant because "borderline" sodium values may become overtly abnormal with minor additional insults. Thyroid-mediated renal and cellular effects may also influence potassium and chloride. Thyroid hormones regulate Na^+/K^+-ATPase activity and contribute to transmembrane ionic gradients that influence serum potassium distribution. Changes in renal tubular flow and aldosterone activity—often increased in hyperthyroid physiology—can promote kaliuresis and, in susceptible individuals, lower potassium levels.[3] In SHE, hemodynamic changes (including increased renal perfusion and altered vascular resistance) may modify tubular solute delivery and fractional excretion patterns, providing a pathway for measurable electrolyte differences even when FT4 remains within range.[5,6] These physiologic shifts underscore the importance of directly comparing SHO and SHE populations rather than assuming symmetrical or negligible effects across subclinical conditions. Mineral electrolytes, such as calcium and magnesium, are closely intertwined with thyroid activity through body turnover, calcitonin activity, abnormal reabsorption, interplay with parathyroid hormone, and vitamin D metabolism. Bone turnover and renal balance can change in hypothyroid states, and a decrease in circulating calcium has been observed in some cohorts. In contrast, magnesium, as a cofactor in enzyme activity and membrane integrity, may demonstrate clinically interesting variability. Cross-sectional assessment suggests that mineral alterations may occur in subclinical hypothyroidism. It may be related to TSH dynamics, indicating that even subtle dysregulation of the thyroid axis may represent extensive metabolic restructuring. In addition, biomarkers linked to fluid–ion homeostasis (including peptides reflecting water balance signaling) have been shown to change in hypothyroid states and relate to sodium and thyroid parameters, reinforcing that endocrine signalling networks relevant to electrolytes can shift even over relatively short physiologic intervals.[7] The kidney serves as a principal effector organ linking thyroid status to electrolyte profiles. Thyroid hormones influence renal development, renal blood flow, and glomerular filtration, while thyroid dysfunction has been associated with reduced renal function and bidirectional endocrine–renal interactions.[5] In subclinical hyperthyroidism, renal and vascular changes have been described and postulated to contribute to broader clinical associations such as blood pressure variation, with accompanying implications for sodium and potassium handling.[6] These observations strengthen the rationale for studying electrolyte patterns alongside thyroid indices in subclinical disease, particularly because tertiary care populations may include individuals in whom renal reserve is limited and electrolyte abnormalities can amplify morbidity. The present study was conducted to correlate-and analyse electrolyte imbalance between subclinical hypothyroidism (SHO) and subclinical hyperthyroidism (SHE) at a tertiary care hospital.

Materials and Methods

A hospital-based analytical cross-sectional study was conducted at the Department of Biochemistry, IMS and SUM Hospital, Campus -II, Bhubaneswar, Odisha (India). It included 128 adult patients with subclinical thyroid. study was conducted according to standardised institutional testing protocols and uniform sample-handling procedures. Patients were enrolled through consecutive sampling from outpatient and inpatient services of the tertiary care hospital. After enrolment, patients were categorised into two groups—SHO and SHE—based on thyroid function test results. Demographic and clinical profiles were recorded for all participants to allow appropriate group comparisons and correlation analyses.

Eligibility criteria

Adults (≥18 years) of either sex with biochemical evidence of subclinical thyroid dysfunction were included. Patients were excluded if they had overt thyroid disease (clinically and/or biochemically), acute systemic illness (including sepsis), pregnancy or postpartum state, known chronic kidney disease (moderate to advanced impairment), chronic liver disease, congestive heart failure, adrenal or parathyroid disorders, recent vomiting/diarrhoea or dehydration, or any condition likely to alter electrolyte levels independently. Patients receiving medications known to influence electrolytes significantly were excluded or appropriately documented and controlled for in analysis, including diuretics, systemic corticosteroids, lithium, amphotericin-B, and high-dose calcium/vitamin D supplementation; individuals on thyroid hormone replacement or anti-thyroid therapy were also excluded to avoid treatment-related shifts in thyroid function and electrolytes.

Operational definitions and grouping criteria

Subclinical hypothyroidism (SHO) was defined as elevated serum thyroid-stimulating hormone (TSH) with circulating free thyroxine (FT4) within the laboratory reference range. Subclinical hyperthyroidism (SHE) was defined as suppressed/low serum TSH with FT4 within the laboratory reference range (and free triiodothyronine [FT3] within reference range, where measured as part of routine evaluation). The laboratory-specific reference intervals used by the tertiary care hospital were applied uniformly for classification to ensure internal validity and consistency across participants.

Methodology

For each participant, a structured pro forma was used to record age, sex, body mass index (BMI), blood pressure, relevant symptoms, comorbidities (including diabetes mellitus and hypertension), and medication history, with particular emphasis on agents affecting sodium, potassium, calcium, and magnesium balance. The essential clinical examination results were recorded to aid case characterisation and to disclose the potential confounders. Renal function was assessed using serum creatinine and estimated glomerular filtration rate (eGFR) to adjust the statistical analysis for electrolyte variation in the kidney.

It involved collecting venous blood using standard aseptic technique and processing it in accordance with institutional laboratory guidelines. Thyroid function tests included serum TSH and FT4 (and FT3, where available/indicated), measured using an automated immunoassay platform routinely employed at the tertiary care hospital laboratory, with internal quality control procedures in place. Serum electrolytes comprised sodium (Na^+), potassium (K^+), and chloride (Cl^-) measured on an automated analyser using ion-selective electrode methodology; calcium (total calcium) and magnesium were measured using standardised colorimetric methods on the same or compatible automated chemistry analyser. Where clinically appropriate and available in routine workup, serum phosphate and serum albumin were recorded so that calcium values could be interpreted in context (including albumin-adjusted calcium when required by institutional practice). Additional parameters recorded to support confounder control included fasting/random plasma glucose and renal profile (urea, creatinine), as these may influence electrolyte distribution and renal handling.

The primary outcome was the difference in electrolyte levels (Na^+, K^+, Cl^-, total calcium, and magnesium) between SHO and SHE groups. As secondary outcome measures, the prevalence of electrolyte abnormalities in both groups, as defined by lab reference ranges, and the relationship of thyroid parameters (TSH, FT4, and FT3, where available), were measured. Further exploratory tests determined the existence of electrolyte changes following the correction of clinically vital covariates, including age, sex, BMI, comorbidities, exposure to electrolyte-impacting medicines, and renal function (eGFR).

Statistical analysis: IBM SPSS Statistics version 27.0 was used to input and analyse the data. The normality of continuous variables was evaluated using the Shapiro-Wilk test and visually by examining histograms and Q-Q plots. Continuous data that were normally distributed were reported in the expected standard way as mean and standard deviation, and non-normally distributed data were expressed in a clear, typical format as median and interquartile range. The frequencies and percentages were used to indicate the presence of categorical variables. Continuous variables (SHO vs SHE) were compared using an independent samples t-test (normally distributed data) or the Mann-Whitney U test (skewed data), and categorical variables were compared using a chi-square test or Fisher's exact test, respectively. Pearson's correlation (parametric) and Spearman's rank correlation (nonparametric) have been used to determine correlations between thyroid function parameters and electrolyte values. Multivariate linear regression models were developed with electrolytes as the dependent variable and thyroid category (SHO/SHE) and thyroid hormone parameters as predictors, after controlling for age, sex, BMI, eGFR, comorbidities, and any appropriate medication exposure. The study presented results with 95% confidence intervals for the latter when available, and a two-tailed p-value of less than 0.05 was considered statistically significant. Complete data were confirmed before proceeding to the analysis; any cases of missing data were handled using appropriate procedures, depending on the extent and pattern of missing data.

Results

[Table 1] summarises the distribution of the 128 enrolled participants by subclinical thyroid status. Out of the total study population, 66 participants (51.56) were determined to be of subclinical hypothyroidism (SHO), and 62 subjects (48.44) of subclinical hyperthyroidism (SHE).

[Table 2] shows the initial demographic and clinical features of the members of both groups. The age of patients in the SHO group was 42.18 ± 11.36 years, and the age of patients in the SHE group was 39.74 ± 10.92 years, with a difference of 0.214 ± 0.214, which is not statistically significant. The females were the majority in both groups, with 44 (66.67) in the SHO group and 38 (61.29) in the SHE groups, but the sex distribution was not significant ($p = 0.529$). In the same way, the BMI in the SHO group (24.96 ± 3.42 kg/m 2) was slightly more than in the SHE groups (23.88 ± 3.27 kg/m 2), though this difference was non-significant ($p = 0.083$). In terms of comorbidities, hypertension was found in 27.27% of SHE patients, and diabetes mellitus was found in 24.24% of SHO patients ($p= 0.533$ and $p= 0.661$).

[Table 3] compares thyroid function parameters between the SHO and SHE groups and confirms the expected biochemical distinction between the two subclinical conditions. Serum TSH levels were markedly higher in SHO (8.92 ± 2.14 mIU/L) than in SHE (0.19 ± 0.07 mIU/L), and the difference was highly statistically significant ($p < 0.001$), validating the group classification based on thyroid status. In contrast, FT4 levels remained within the reference range for both groups: SHO 1.18 ± 0.14 ng/dL and SHE 1.21 ± 0.16 ng/dL; the difference was not significant ($p = 0.267$). Likewise, FT3 (where available) was similar between groups (3.02 ± 0.41 pg/mL in SHO vs 3.11 ± 0.46 pg/mL in SHE), showing no statistically significant difference ($p = 0.241$).

[Table 4] compares the serum electrolyte profiles between the SHO and SHE groups and shows statistically significant differences across all measured electrolytes. Mean serum sodium was significantly lower in SHO (134.62 ± 3.18 mEq/L) compared with SHE (137.28 ± 2.94 mEq/L), and this difference was highly significant ($p < 0.001$), suggesting a greater tendency toward lower sodium levels in SHO. Mean serum potassium was significantly higher in SHO (4.18 ± 0.46 mEq/L) than in SHE (3.92 ± 0.41 mEq/L), with a significant difference ($p = 0.002$). Similarly, serum chloride was lower in SHO (100.84 ± 3.56 mEq/L) compared with SHE (102.91 ± 3.22 mEq/L), and this difference was statistically significant ($p = 0.001$). For mineral electrolytes, serum calcium was significantly lower in SHO (8.68 ± 0.54 mg/dL) than in SHE (9.12 ± 0.51 mg/dL) ($p < 0.001$), and serum magnesium was also significantly lower in SHO (1.74 ± 0.26 mg/dL) compared to SHE (1.92 ± 0.24 mg/dL) ($p < 0.001$).

[Table 5] presents an assessment of electrolyte abnormalities based on reference-range cutoffs, along with an explanation of the clinical distribution of electrolyte imbalance between groups. Hyponatremia was significantly more prevalent in SHO (24 patients, 36.36% of the total) than in SHE (8 patients, 12.90% of the total), with a statistically significant difference ($p = 0.002$). The incidence of hypocalcemia was also significantly higher in SHO, with 22 patients (33.33%) having hypocalcemia, compared to 6 patients (9.68%) in SHE; the difference was quite significant ($p = 0.001$). On the same note, only 19 SHO patients (28.79) had hypomagnesemia, compared with 7 SHE patients (11.29), with a significant difference ($p = 0.013$). On the contrary, hypokalaemia was more common in SHE (18 patients, 29.03%) than in SHO (10 patients, 15.15%), and this difference was statistically significant ($p = 0.048$).

The results of the correlation analysis between TSH and serum electrolytes in the entire study population (n = 128) are presented in Table 6 and show statistically significant relationships. TSH was moderately negatively correlated with sodium ($r = -0.42$, $p < 0.001$), suggesting that high TSH was associated with low sodium levels. TSH also showed a moderate negative correlation with calcium ($r = -0.47$, $p = 0.001$) and a negative correlation with magnesium ($r = -0.38$, $p = 0.001$), indicating that an increase in TSH levels is associated with decreases in calcium and magnesium concentrations. On the other hand, TSH was positively correlated with potassium ($r = 0.29$, $p = 0.001$), indicating that TSH values were positively correlated with potassium.

Table 1: Distribution of study participants according to subclinical thyroid status (n = 128)

Thyroid status	Number of patients (n)	Percentage (%)
Subclinical hypothyroidism (SHO)	66	51.56
Subclinical hyperthyroidism (SHE)	62	48.44
Total	128	100.00

Table 2: Baseline demographic and clinical characteristics of study participants

Variable	SHO (n = 66)	SHE (n = 62)	p-value
Age (years), mean ± SD	42.18 ± 11.36	39.74 ± 10.92	0.214
Female, n (%)	44 (66.67)	38 (61.29)	0.529
Male, n (%)	22 (33.33)	24 (38.71)	
BMI (kg/m²), mean ± SD	24.96 ± 3.42	23.88 ± 3.27	0.083
Hypertension, n (%)	18 (27.27)	14 (22.58)	0.533
Diabetes mellitus, n (%)	16 (24.24)	13 (20.97)	0.661

Table 3: Comparison of thyroid function parameters between SHO and SHE groups

Parameter	SHO (n = 66) Mean ± SD	SHE (n = 62) Mean ± SD	p-value
TSH (mIU/L)	8.92 ± 2.14	0.19 ± 0.07	<0.001
FT4 (ng/dL)	1.18 ± 0.14	1.21 ± 0.16	0.267
FT3 (pg/mL) *	3.02 ± 0.41	3.11 ± 0.46	0.241

*FT3 values included where available as part of routine evaluation.

Table 4: Comparison of serum electrolyte levels between SHO and SHE groups

Electrolyte	SHO (n = 66) Mean ± SD	SHE (n = 62) Mean ± SD	p-value
Sodium (mEq/L)	134.62 ± 3.18	137.28 ± 2.94	<0.001
Potassium (mEq/L)	4.18 ± 0.46	3.92 ± 0.41	0.002
Chloride (mEq/L)	100.84 ± 3.56	102.91 ± 3.22	0.001
Calcium (mg/dL)	8.68 ± 0.54	9.12 ± 0.51	<0.001
Magnesium (mg/dL)	1.74 ± 0.26	1.92 ± 0.24	<0.001

Table 5: Prevalence of electrolyte abnormalities in SHO and SHE groups

Electrolyte abnormality	SHO (n = 66) n (%)	SHE (n = 62) n (%)	p-value
Hyponatremia	24 (36.36)	8 (12.90)	0.002
Hypokalaemia	10 (15.15)	18 (29.03)	0.048
Hypocalcemia	22 (33.33)	6 (9.68)	0.001
Hypomagnesemia	19 (28.79)	7 (11.29)	0.013

p-value calculated using chi-square test.

Table 6: Correlation of TSH with serum electrolytes (overall study population, n = 128)

Electrolyte	Correlation coefficient (r)	p-value
Sodium	−0.42	<0.001
Potassium	0.29	0.001
Calcium	−0.47	<0.001
Magnesium	−0.38	<0.001

Pearson’s correlation test applied.

Discussion

In this tertiary-care cohort (n=128), subclinical hypothyroidism (SHO) accounted for 51.56% (66/128) and subclinical hyperthyroidism (SHE) for 48.44% (62/128), with a clear female predominance in both groups (SHO: 66.67% female; SHE: 61.29% female). This sex pattern is consistent with population-level evidence showing that thyroid dysfunction and thyroid autoimmunity are more frequent among women; Hollowell et al (2002), analysing NHANES III, reported substantial sex-related differences in thyroid indices and thyroid antibodies in the general population, which supports the observation that women are disproportionately represented among patients presenting with biochemical thyroid dysfunction in clinical settings.[8]

The age distribution in the present study (SHO: 42.18±11.36 years; SHE: 39.74±10.92 years; p=0.214) and the broadly comparable background burden of cardiometabolic comorbidities (hypertension 27.27% vs 22.58%, diabetes 24.24% vs 20.97%) suggest that the electrolyte differences observed are less likely to be explained by major baseline demographic imbalance. Longitudinal community follow-up data have shown that thyroid dysfunction accumulates across adult life and is not confined to late age; Vanderpump et al (1995), in the 20-year Whickham follow-up, documented ongoing incident thyroid disease through adulthood, which provides epidemiological context for why many "subclinical" cases in hospital practice cluster in mid-life rather than only in older age strata.[9]

The thyroid hormone profile in this work appropriately reflected subclinical disease, with TSH as the principal discriminator between groups: TSH 8.92±2.14 mIU/L in SHO versus 0.19±0.07 mIU/L in SHE (p<0.001), while FT4 remained similar and within reference range (1.18±0.14 vs 1.21±0.16 ng/dL; p=0.267). These findings align with hospital-based data in subclinical hypothyroidism where TSH is elevated but FT4 is preserved; Jat et al (2021) reported mean TSH around the same order of magnitude (subclinical hypothyroid TSH approximately 9.41±4.10 μIU/mL) with FT4 within the normal range (about 1.22±0.36 ng/dL), supporting that the biochemical phenotype in our SHO arm is representative of typical subclinical hypothyroidism.[10]

In this study, the significant difference in extracellular ions between SHO and SHE is particularly sodium and chloride. We observed lower sodium in SHO (134.62±3.18 mEq/L) compared with SHE (137.28±2.94 mEq/L; p<0.001) and lower chloride in SHO (100.84±3.56) compared with SHE (102.91±3.22 mEq/L; p=0.001), indicating a tendency toward relatively reduced sodium–chloride values in SHO. Similar directionality has been reported in other tertiary-care comparisons; Saxena et al (2023) found sodium to be lower in SHO than SHE (approximately 137.3 vs 140.8 mEq/L) and chloride lower in SHO than SHE (about 108.7 vs 115.3 mEq/L), reinforcing that subclinical thyroid status can be associated with measurable, group-level differences in these electrolytes, even though absolute means vary across laboratories, diet patterns, and analytical platforms.[11]

In the present work, potassium showed a contrasting pattern: SHO had a higher mean potassium (4.18±0.46 mEq/L) than SHE (3.92±0.41 mEq/L; p=0.002), and hypokalaemia was more frequent in SHE (29.03%) than SHO (15.15%; p=0.048). This is biologically plausible given prior evidence that hyperthyroid physiology can predispose to clinically significant potassium shifts. Although our SHE cohort was "subclinical," the higher hypokalaemia burden parallels the broader thyrotoxic spectrum described in neuromuscular presentations; Kalita et al (2012), in an Indian experience with thyrotoxic periodic paralysis, documented profound hypokalaemia in thyrotoxic cases (mean potassium around 2.21±0.49 mmol/L), illustrating the direction of potassium vulnerability in hyperthyroid states, against which our milder but significant SHE hypokalaemia frequency can be interpreted as a less severe expression on the same physiological continuum.[12]

Our correlation analysis further supported a thyroid–electrolyte association: TSH correlated negatively with sodium (r = -0.42, p < 0.001) and positively with potassium (r = 0.29, p = 0.001), indicating that as TSH increased, sodium tended to decrease while potassium tended to increase. Large retrospective datasets also identify statistically detectable thyroid–electrolyte relationships, albeit often with smaller effect sizes. Schwarz et al (2012) analysed a broad emergency-department dataset and concluded that an association between thyroid function and electrolyte disorders exists, but is typically most clinically relevant in more marked thyroid dysfunction; compared with their conclusions, our moderate correlations may reflect a more selected hospital cohort with protocol-based exclusions of alternative electrolyte drivers, thereby amplifying the detectable thyroid contribution.[13]

The categorical analysis of sodium abnormalities is particularly notable: hyponatremia was present in 36.36% of SHO compared with 12.90% of SHE (p=0.002), consistent with the lower SHO mean sodium. When placed against large cross-sectional hyponatremia research, our prevalence appears higher—reflecting clinical selection within a tertiary-care setting and the specific inclusion of subclinical thyroid disease. Nagata et al (2018), in a large retrospective analysis, reported that overt hypothyroidism prevalence increased with hyponatremia severity and provided category-level data (e.g., overt hypothyroidism prevalence approximately 1.2% at sodium ≥136 mEq/L, rising to about 3.5% at sodium ≤129 mEq/L), supporting the concept that thyroid dysfunction and sodium derangements are linked. However, the magnitude and clinical visibility of that link vary across population mixes and study designs.[14]

Differences in clinical context also help explain variability in reported hyponatremia frequencies across studies. In our SHO cohort, the mean sodium was 134.62±3.18 mEq/L, and 36.36% had hyponatremia. In contrast, hyponatremia prevalence is often lower in cohorts in which hypothyroidism is transiently induced for therapeutic reasons and in which competing factors (e.g., isolation protocols, diet, fluid intake) influence sodium. Dayrit et al (2016), examining hypothyroid patients undergoing radioactive iodine ablation preparation, reported hyponatremia prevalence of 6.7% pre-RAI and 26.7% post-RAI, with no significant correlation between TSH and sodium in that specific setting; compared with those figures, our higher SHO hyponatremia rate may reflect differences in patient selection, comorbidity patterns, and real-world inpatient/outpatient exposures typical of tertiary-care practice.[15]

Mineral electrolytes in our study showed significant group separation and clinically meaningful categorical abnormalities: calcium was lower in SHO (8.68±0.54 mg/dL) than SHE (9.12±0.51 mg/dL; p<0.001), magnesium was lower in SHO (1.74±0.26 mg/dL) than SHE (1.92±0.24 mg/dL; p<0.001), and hypocalcemia (33.33% vs 9.68%) and hypomagnesemia (28.79% vs 11.29%) were both more common in SHO. Importantly, TSH correlated negatively with calcium (r=−0.47) and magnesium (r=−0.38) (both p<0.001), suggesting that higher TSH states in this cohort tracked with lower mineral levels. Prior work has documented thyroid-related mineral disturbances, though patterns may differ by overt vs subclinical disease, nutrition, renal handling, and assay methodology; Abdel-Gayoum et al (2014) reported altered mineral profiles across thyroid dysfunction states (including reductions in serum calcium and phosphate in hypothyroidism and reductions in magnesium in hyperthyroidism), highlighting that mineral shifts can occur across the thyroid spectrum and that inter-study differences in direction (particularly for magnesium) may reflect differences in patient characteristics, disease severity distribution, and contextual confounding.[16]

Conclusion

This paper emphasises that even subclinical thyroid dysfunction is associated with significant changes in serum electrolyte levels. Subclinical hypothyroidism was demonstrated by the decreased levels of sodium, calcium, and magnesium at a higher rate of related electrolyte imbalances, whereas subclinical hyperthyroidism was more inherently inclined to hypokalaemia. The acute links between TSH and several electrolytes also accentuate the role of thyroid status on the equilibrium of electrolytes. Frequent checks of electrolytes in patients with subclinical thyroid disorders can help them identify imbalances sooner and thereby correct them as and when they occur, thereby achieving better clinical outcomes.

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Conflicts of interest

There are no conflicts of interest.

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