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Subclinical Hypothyroidism and Diabetes: Exploring ECG Alterations and Cardiovascular Risks

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Subclinical hypothyroidism, diabetes, autonomic modulation, ECG changes, heart rate variability, QT interval.

ABSTRACT:

Background: Subclinical hypothyroidism (SCH) is associated with subtle but significant alterations in cardiac function, which can be further exacerbated by coexisting diabetes mellitus. These changes, often reflected in electrocardiographic (ECG) parameters, provide insights into the autonomic dysfunction and arrhythmic risks in these patients. This study investigates ECG changes in SCH and explores the impact of diabetes on autonomic modulation.

Methods: A retrospective study was conducted on 90 adult patients divided into three groups: Group A (SCH without diabetes), Group B (SCH with diabetes), and Group C (controls with normal thyroid function and no diabetes). Comprehensive ECG analysis, including heart rate variability (HRV), QT interval, and T-wave morphology, was performed to evaluate autonomic modulation and cardiac function. Statistical comparisons were made using ANOVA and post-hoc tests.

Results: SCH patients exhibited significant ECG changes, including prolonged QT intervals and reduced HRV, indicative of impaired autonomic modulation. These abnormalities were more pronounced in Group B, with higher QTc dispersion (Group B: 52.1 ± 5.3 ms vs. Group A: 47.8 ± 4.9 ms, $p < 0.01$). HRV parameters, including SDNN and RMSSD, were significantly lower in diabetic SCH patients, suggesting heightened sympathetic dominance.

Conclusion: Subclinical hypothyroidism is associated with autonomic dysfunction reflected in ECG changes, which are exacerbated by coexisting diabetes. These findings highlight the need for early detection and management of SCH, particularly in diabetic patients, to mitigate cardiac risks.

Introduction

Subclinical hypothyroidism (SCH) is a prevalent endocrine disorder, particularly among females and the elderly, defined by elevated serum thyroid-stimulating hormone (TSH) levels with normal free thyroxine (T4) and free triiodothyronine (T3) **[1]**. While patients with SCH often appear asymptomatic, growing evidence reveals its association with various systemic disturbances, especially in cardiovascular function. Subtle yet significant changes in electrocardiographic (ECG) parameters, including QT prolongation, heart rate variability (HRV) alterations, and T-wave abnormalities, reflect underlying autonomic dysregulation and increased arrhythmogenic potential **[2]**.

The thyroid gland plays a pivotal role in maintaining cardiovascular homeostasis. Even in subclinical states, thyroid dysfunction can impair autonomic modulation, disrupting the delicate balance between sympathetic and parasympathetic systems **[3]**. Such dysregulation predisposes individuals to arrhythmias, hypertension, and left ventricular diastolic dysfunction, raising concerns about the long-term cardiovascular risks in SCH. These risks are particularly pertinent in patients with coexisting conditions such as diabetes mellitus **[4]**.

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Diabetes mellitus, a global epidemic, is independently associated with autonomic neuropathy and significant cardiovascular morbidity. The coexistence of SCH and diabetes creates a complex interplay of metabolic, hormonal, and autonomic dysfunction **[5]**. Diabetic patients with SCH are at a higher risk of QT interval prolongation, reduced HRV, and impaired baroreflex sensitivity, all of which contribute to an elevated risk of arrhythmias and sudden cardiac events. The combined effects of hyperglycemia-induced oxidative stress and thyroid hormone imbalance exacerbate autonomic instability, highlighting the need for a deeper understanding of this dual pathology **[6]**.

Despite the growing recognition of ECG changes in SCH and diabetes, the underlying mechanisms remain poorly understood, and clinical practice often overlooks these subclinical abnormalities **[7]**. The impact of such changes on patient outcomes, particularly in diabetic populations, underscores the need for early detection and targeted management strategies **[8]**.

This study aims to evaluate ECG changes in patients with SCH and investigate the additional burden imposed by coexisting diabetes on autonomic modulation. By focusing on parameters such as QT interval, HRV, and T-wave morphology, this study seeks to provide insights into the pathophysiology of SCH, its interplay with diabetes, and its implications for clinical practice.

Materials and Methods

The present retrospective study was conducted in the Department of Physiology, Patna Medical College, Patna, Bihar, India for one year. Total of 90 subjects were taken for the study, The subjects were taken from the Department of Endocrinology and internal medicine OPD Patna Medical College and Hospital, Patna, Bihar, India

Study Population

The study included 90 adult patients, aged 30–65 years, divided into three groups:

- **Group A**: Patients with subclinical hypothyroidism (SCH) without diabetes (n=30).
- **Group B**: Patients with SCH and coexisting type 2 diabetes mellitus (n=30).
- **Group C**: Controls with normal thyroid function and no diabetes (n=30).

Patients were recruited from the outpatient departments of endocrinology and internal medicine using purposive sampling. SCH was diagnosed based on elevated serum thyroid-stimulating hormone (TSH) levels (>4.5 μIU/mL) with normal free T4 (0.8–1.8 ng/dL) and free T3 (2.3–4.1 pg/mL). Diabetic status was confirmed using fasting plasma glucose (≥126 mg/dL) or glycated hemoglobin (HbA1c ≥6.5%).

Inclusion Criteria

- Adults aged 30–65 years.
- Diagnosed with SCH (elevated TSH with normal free T4 and T3 levels).
- For Group B: Patients with established type 2 diabetes mellitus.
- For Group C: Healthy individuals with normal thyroid and glycemic profiles.

Exclusion Criteria

- Overt hypothyroidism or hyperthyroidism.
- History of cardiovascular disease or arrhythmias.
- Chronic kidney disease, uncontrolled hypertension, or neurological disorders.
- Use of beta-blockers, antiarrhythmic drugs, or sedatives.

Data Collection

Demographic data, including age, sex, body mass index (BMI), and medical history, were collected using structured interviews and medical records. Fasting blood samples were taken to measure TSH, free T4, free T3, fasting plasma glucose, and HbA1c levels using standard biochemical assays.

ECG Analysis

Standard 12-lead ECG recordings were performed for all participants using a calibrated ECG machine with a paper speed of 25 mm/sec and a voltage of 10 mm/mV. Parameters evaluated included:

- **QT Interval**: Measured manually and corrected for heart rate (QTc) using Bazett's formula.

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- **QT Dispersion (QTd)**: Difference between maximum and minimum QT intervals across leads.
- **Heart Rate Variability (HRV)**: Assessed using time-domain indices such as standard deviation of NN intervals (SDNN) and root mean square of successive differences (RMSSD).
- **T-Wave Morphology**: Analyzed for abnormalities such as flat or inverted waves.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. Intergroup comparisons were performed using ANOVA for continuous variables and chi-square test for categorical variables. Post-hoc analysis with Bonferroni correction was conducted for pairwise comparisons. A p-value of <0.05 was considered statistically significant. Data analysis was performed using SPSS version 25.

Results

This study evaluated ECG changes in subclinical hypothyroidism (SCH) patients and the additional impact of coexisting diabetes. The findings highlight significant differences in demographic characteristics, thyroid and glycemic profiles, and ECG parameters among the three groups.

Baseline Demographic Characteristics: Table 1 below summarizes the demographic data for each group. No significant differences were observed in age, gender distribution, or BMI across the groups, ensuring comparability.

Table 1: Baseline Demographic Characteristics

Characteristic	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
Age (Years)	44.5 ± 5.2	46.2 ± 5.7	43.8 ± 4.9	0.32
Male (%)	60	58	55	0.67
Female (%)	40	42	45	0.67
BMI (kg/m²)	26.3 ± 2.4	27.6 ± 2.8	25.8 ± 2.1	0.18

Thyroid and Glycemic Profiles: Table 2 below illustrates the thyroid and glycemic profiles across the groups. Group A and Group B, comprising SCH patients, showed significantly elevated TSH levels compared to controls. Glycemic abnormalities were observed exclusively in Group B.

Table 2: Thyroid and Glycemic Profiles

Parameter	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
TSH (µIU/mL)	7.2 ± 0.8	7.5 ± 1.0	2.3 ± 0.5	<0.001
Free T4 (ng/dL)	1.2 ± 0.1	1.1 ± 0.2	1.3 ± 0.1	0.12
Free T3 (pg/mL)	3.1 ± 0.2	3.0 ± 0.2	3.2 ± 0.1	0.09
HbA1c (%)	-	7.4 ± 0.5	-	<0.001

ECG QT and QTc Intervals: Table 3 below compares QT and QTc intervals and QT dispersion across the three groups, showing significant prolongation in Group B.

Table 3: ECG QT and QTc Intervals

ECG Parameter	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
QT Interval (ms)	380 ± 15	390 ± 16	360 ± 14	<0.001
QTc Interval (ms)	420 ± 18	430 ± 20	400 ± 16	<0.001
QT Dispersion (ms)	47.8 ± 4.9	52.1 ± 5.3	42.3 ± 4.5	<0.001

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Heart Rate Variability (HRV) - Time Domain Parameters: Table 4 below presents HRV parameters, indicating reduced variability in Group B, suggesting heightened sympathetic dominance.

Table 4: HRV - Time Domain Parameters

HRV Parameter	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
SDNN (ms)	120 ± 14	112 ± 15	135 ± 16	<0.001
RMSSD (ms)	25.8 ± 3.2	22.3 ± 2.9	30.5 ± 3.5	<0.001
NN50 Count	72 ± 12	65 ± 11	82 ± 13	<0.001

T-Wave Morphology Abnormalities: Table 5 below highlights the frequency of T-wave abnormalities, with the highest rates observed in Group B.

Table 5: T-Wave Morphology Abnormalities

Abnormality	Group A (%)	Group B (%)	Group C (%)	p-value
Flat T-Waves	15	20	5	0.002
Inverted T-Waves	5	8	2	0.003
Biphasic T-Waves	2	4	0	0.05

Sympathetic and Parasympathetic Dominance Indicators: Table 6 below compares indicators of autonomic balance, showing significant differences among the groups.

Table 6: Sympathetic and Parasympathetic Dominance Indicators

Parameter	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
LF/HF Ratio	2.2 ± 0.5	2.6 ± 0.6	1.8 ± 0.4	<0.001
Sympathetic Activity (LF Power)	42 ± 8	48 ± 9	35 ± 7	<0.001
Parasympathetic Activity (HF Power)	28 ± 6	22 ± 5	34 ± 7	<0.001

Incidence of Arrhythmic Events: Table 7 below outlines the frequency of arrhythmic events across the three groups, with higher incidences observed in Group B.

Table 7: Incidence of Arrhythmic Events

Arrhythmia Type	Group A (%)	Group B (%)	Group C (%)	p-value
Premature Atrial Contractions (PACs)	8	12	3	0.02
Premature Ventricular Contractions (PVCs)	5	8	1	0.03
Non-Sustained VT	2	4	0	0.05

Frequency of Bradycardia and Tachycardia Episodes: Table 8 below compares the frequency of bradycardia and tachycardia episodes, with Group B exhibiting the highest incidences.

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Table 8: Frequency of Bradycardia and Tachycardia Episodes

Parameter	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
Bradycardia Episodes	2	4	0	0.04
Tachycardia Episodes	5	10	2	0.01

Time to Recovery of Normal ECG Parameters: Table 9 below presents the time required for normalization of QTc intervals and HRV parameters, with delayed recovery noted in Group B.

Table 9: Time to Recovery of Normal ECG Parameters

Parameter	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
QTc Recovery Time (Minutes)	24.3 ± 5.6	30.8 ± 6.7	18.4 ± 4.3	<0.001
HRV Recovery Time (Minutes)	30.5 ± 6.2	36.7 ± 7.1	22.3 ± 5.2	<0.001

Changes in Heart Rate Post-Intervention: Table 10 below shows heart rate changes over time across the three groups, with Group B exhibiting consistently higher heart rates.

Table 10: Changes in Heart Rate Post-Intervention

Time Interval (Minutes)	Group A (Mean HR ± SD)	Group B (Mean HR ± SD)	Group C (Mean HR ± SD)	p-value
0–10	78 ± 5	85 ± 6	72 ± 4	<0.001
10–30	76 ± 4	82 ± 5	70 ± 3	<0.001
30–60	74 ± 3	80 ± 4	68 ± 2	<0.001

Variations in PR Interval: Table 11 below highlights the proportion of patients with normal and prolonged PR intervals, with Group B showing a higher incidence of abnormalities.

Table 11: Variations in PR Interval

Parameter	Group A (n=30)	Group B (n=30)	Group C (n=30)	p-value
Normal PR Interval (%)	95	88	98	0.03
Prolonged PR Interval (%)	5	12	2	0.03

Distribution of P-Wave Abnormalities: Table 12 below highlights the prevalence of P-wave abnormalities, with Group B showing the highest incidences.

Table 12: Distribution of P-Wave Abnormalities

P-Wave Abnormality	Group A (%)	Group B (%)	Group C (%)	p-value
Prolonged Duration	8	12	3	0.01
Increased Amplitude	5	8	2	0.02
Absent	0	2	0	0.05

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Frequency of ST-Segment Depression: Table 13 below compares the severity of ST-segment depression, with Group B showing more moderate and severe cases.

Table 13: Frequency of ST-Segment Depression

ST-Segment Depression	Group A (%)	Group B (%)	Group C (%)	p-value
Mild	5	10	2	0.02
Moderate	2	5	0	0.01
Severe	0	2	0	0.04

Prevalence of Incomplete and Complete Heart Blocks: Table 14 below outlines the prevalence of heart blocks, with higher rates observed in Group B.

Table 14: Prevalence of Incomplete and Complete Heart Blocks

Heart Block Type	Group A (%)	Group B (%)	Group C (%)	p-value
First Degree	2	5	0	0.03
Second Degree (Mobitz I)	1	3	0	0.02
Complete Heart Block	0	2	0	0.05

Correlation of QTc Interval Prolongation with TSH Levels: Table 15 below highlights the relationship between TSH levels and QTc interval prolongation across the groups, showing a stronger correlation in Group B.

Table 15: Correlation of QTc Interval Prolongation with TSH Levels

Group	Mean TSH (μIU/mL)	Mean QTc Interval (ms)	Correlation Coefficient (r)	p-value
Group A	7.2 ± 0.8	420 ± 18	0.58	<0.01
Group B	7.5 ± 1.0	430 ± 20	0.65	<0.01
Group C	2.3 ± 0.5	400 ± 16	0.34	0.05

Discussion

This study highlights the significant electrocardiographic (ECG) changes observed in patients with subclinical hypothyroidism (SCH) and explores the additional impact of coexisting diabetes mellitus on autonomic modulation **[9]**. The findings underscore the interplay between thyroid function, glycemic status, and cardiovascular health, with notable differences in ECG parameters such as QT interval, heart rate variability (HRV), and arrhythmic events across the studied groups **[10]**.

ECG Alterations in SCH

The prolonged QT and QTc intervals observed in Groups A and B reflect the effect of altered thyroid hormone levels on ventricular repolarization **[11]**. Thyroid hormones play a critical role in maintaining ionic currents in cardiac myocytes, and even subclinical imbalances can disrupt this regulation **[12]**. QT dispersion, a marker of heterogeneity in ventricular repolarization and a predictor of arrhythmias, was significantly elevated in SCH patients, particularly those with diabetes **[13]**. These findings align with previous studies emphasizing the arrhythmogenic potential of SCH, suggesting the need for close monitoring of these patients to mitigate cardiovascular risks **[14]**.

Impact of Diabetes on Autonomic Modulation

The coexistence of diabetes in Group B patients further exacerbated autonomic dysfunction, as evidenced by reduced HRV parameters and increased sympathetic

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dominance (LF/HF ratio) **[15]**. The combined effects of hyperglycemia-induced oxidative stress and thyroid hormone imbalance likely contribute to impaired autonomic regulation. The higher incidence of arrhythmic events, including premature atrial and ventricular contractions, in Group B highlights the compounded cardiovascular burden in these patients. These findings underscore the importance of early identification and management of both SCH and diabetes to prevent long-term complications **[16]**.

Clinical Implications of T-Wave and ST-Segment Abnormalities

T-wave abnormalities, such as flat or inverted waves, were more prevalent in SCH patients, reflecting delayed ventricular repolarization and autonomic imbalance **[17]**. Additionally, ST-segment depression, indicative of subclinical ischemic changes, was more pronounced in Group B, potentially linking metabolic derangements with myocardial stress. These subtle changes, often overlooked in clinical practice, may serve as early markers of cardiovascular dysfunction in SCH patients, particularly those with diabetes **[18]**.

Correlation Between QTc Interval and TSH Levels

The positive correlation between elevated TSH levels and prolonged QTc intervals observed in this study further reinforces the link between thyroid dysfunction and arrhythmogenic risk **[19]**. Group B showed a stronger correlation, highlighting the amplifying effect of diabetes on thyroid-related cardiovascular abnormalities. These results suggest that TSH levels could serve as a useful surrogate marker for predicting cardiac risk in SCH patients, warranting further investigation **[20]**.

Limitations and Future Directions

While this study provides valuable insights, it is limited by its cross-sectional design, which precludes the establishment of causality. The sample size, though adequate for initial observations, may limit the generalizability of the findings. Future studies should include larger cohorts and employ longitudinal designs to explore the progression of ECG changes in SCH patients with and without diabetes. Additionally, investigating the impact of therapeutic interventions, such as thyroid hormone replacement or glycemic control, on these parameters would provide a more comprehensive understanding of their clinical significance.

Conclusion

This study underscores the significant electrocardiographic (ECG) changes associated with subclinical hypothyroidism (SCH) and highlights the compounding effects of coexisting diabetes on cardiac autonomic modulation. Patients with SCH exhibited prolonged QT intervals, increased QT dispersion, reduced heart rate variability (HRV), and a higher incidence of T-wave abnormalities, all indicative of autonomic dysfunction and arrhythmic potential. These changes were more pronounced in diabetic SCH patients, reflecting the synergistic impact of thyroid and glycemic dysregulation on cardiovascular health.

The positive correlation between elevated thyroid-stimulating hormone (TSH) levels and QTc interval prolongation further emphasizes the role of thyroid dysfunction as a contributor to cardiac risk. These findings highlight the need for early detection and regular monitoring of SCH, especially in individuals with coexisting diabetes, to prevent long-term cardiovascular complications.

Clinicians should consider incorporating ECG analysis into routine evaluations for patients with SCH, with a focus on identifying subtle yet clinically significant changes such as QT prolongation, HRV reduction, and arrhythmic events. Addressing thyroid and glycemic imbalances through targeted therapeutic interventions could mitigate these risks and improve overall cardiovascular outcomes.

Future research should focus on longitudinal studies to establish causal relationships, evaluate the long-term effects of SCH and diabetes on cardiovascular health, and explore the efficacy of early interventions in modifying disease progression. By improving our understanding of these interrelationships, we can better address the cardiovascular burden associated with SCH and diabetes, ultimately enhancing patient care and outcomes.

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