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Original Research Article

Echocardiographic Assessment of Cardiac Function in Patients with Thyroid Disorders

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Abstract

Background: Thyroid hormones exert direct genomic and non-genomic effects on cardiomyocytes and the vascular system, and both hormone excess and deficiency may produce measurable alterations in ventricular performance before overt cardiovascular disease becomes clinically apparent.

Aim: To characterize systolic and diastolic cardiac function across overt and subclinical hyperthyroid and hypothyroid states and to examine associations between thyroid biochemistry and echocardiographic indices.

Methods: This observational cross-sectional study included 194 adults with thyroid dysfunction recruited at the Department of General Medicine, Santosh Medical College & Hospital, Ghaziabad. Participants were classified as overt hyperthyroidism (n=44), subclinical hyperthyroidism (n=38), overt hypothyroidism (n=82), or subclinical hypothyroidism (n=30). Clinical assessment, body mass index (BMI), thyroid function tests, and standardized transthoracic echocardiography were performed. Between-group comparisons, Spearman correlations, and multivariable linear regression for E/e' were undertaken.

Results: Overt hyperthyroidism showed the highest LVEF (70.2±7.24%) and E/A ratio (1.54±0.13) with the lowest E/e' (7.87±2.09), whereas overt hypothyroidism showed lower LVEF (53.3±3.90%), lower E/A (0.915±0.22), and higher E/e' (14.0±3.29); overall between-group differences were significant (p=0.001). TSH correlated inversely with LVEF (rho=-0.838) and E/A (rho=-0.833) and positively with E/e' (rho=0.815), all p<0.001. In multivariable analysis, age (beta=0.0417, p=0.022), BMI (beta=0.2749, p<0.001), TSH (beta=0.0831, p<0.001), and hypothyroid status (beta=2.0785, p=0.006) independently predicted higher E/e' (R2=0.573).

Conclusion: Thyroid dysfunction was associated with a graded echocardiographic phenotype. Hypothyroid states, including subclinical hypothyroidism, demonstrated less favorable systolic and diastolic indices, while hyperthyroid states were characterized by hyperdynamic systolic function. Echocardiography may therefore provide clinically useful phenotyping of cardiac involvement in patients with thyroid disorders.

Keywords: Thyroid Dysfunction; Echocardiography; Hypothyroidism; Hyperthyroidism; Diastolic Function; E/e'; Left Ventricular Ejection Fraction.

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Introduction

Thyroid hormones are major regulators of cardiovascular physiology because their receptors are expressed in cardiomyocytes, vascular smooth-muscle cells, and endothelial tissue. Triiodothyronine modifies transcription of proteins involved in calcium cycling, beta-adrenergic signaling, myosin isoform expression, and myocardial energetics, while also producing rapid non-genomic effects on ion channels and vascular tone. Consequently, deviations in thyroid hormone availability can alter chronotropy, inotropy, lusitropy, systemic vascular resistance, blood

volume, and ventricular loading conditions. The clinical consequences of this cardio-thyroid interaction have been recognized for decades and range from sinus tachycardia, widened pulse pressure, atrial arrhythmias, and high-output physiology in hyperthyroidism to bradycardia, increased systemic vascular resistance, diastolic hypertension, pericardial effusion, and impaired ventricular relaxation in hypothyroidism [1]. These effects are not limited to severe endocrine disease; subtle biochemical perturbations may also affect myocardial performance and cardiovascular risk.

Contemporary mechanistic and clinical evidence indicates that thyroid hormone signaling influences virtually every component of cardiovascular function. In hyperthyroidism, reduced systemic vascular resistance coexists with increased heart rate, preload, contractility, and cardiac output. In hypothyroidism, the converse physiology is typical: reduced heart rate and contractile state, impaired active relaxation, and increased arterial stiffness collectively increase afterload and reduce effective cardiac performance. These mechanisms provide a biologically coherent explanation for the contrasting echocardiographic phenotypes observed across thyroid states [2]. A large state-of-the-art review further emphasized that even modest changes in circulating thyroid hormones can influence endothelial function, blood pressure, myocardial systolic and diastolic performance, lipid metabolism, and clinical cardiovascular outcomes [3]. More recent synthesis has reinforced the relevance of this spectrum, noting that both overt and subclinical thyroid disorders may be associated with structural heart disease, arrhythmias, heart failure, and vascular dysfunction [4].

The importance of the cardio-thyroid interface has become still clearer with newer epidemiologic and translational evidence. A 2026 European Heart Journal review summarized contemporary data linking thyroid dysfunction with altered blood pressure regulation, endothelial dysfunction, impaired cardiac function, dyslipidaemia, arrhythmias, and heart-failure risk, while also highlighting persistent uncertainty regarding how much cardiovascular risk is reversed by thyroid-specific therapy, particularly in subclinical disease [5]. These uncertainties support phenotyping strategies that identify actual cardiac involvement rather than relying on thyroid biochemistry alone. In this context, transthoracic echocardiography is particularly attractive because it is non-invasive, widely available, repeatable, and capable of providing simultaneous information on chamber dimensions, global systolic function, transmitral filling, tissue Doppler velocities, and estimated filling pressures.

Reliable interpretation of echocardiographic findings depends on standardized acquisition and reporting. Recommendations from the American Society of Echocardiography and European Association of Cardiovascular Imaging have established reproducible approaches for chamber quantification, including left ventricular dimensions and ejection fraction [6]. Diastolic function assessment requires integration of transmitral inflow and tissue Doppler parameters; the E/A ratio characterizes the pattern of early and late ventricular filling, whereas E/e' is commonly used as a non-invasive marker of left ventricular filling pressure within an integrated diastolic assessment [7]. These measurements are especially relevant in thyroid disease because impaired relaxation may precede overt systolic dysfunction, and conventional ejection fraction can remain within a seemingly acceptable range despite clinically meaningful abnormalities in myocardial relaxation or deformation.

Subclinical thyroid dysfunction presents a particularly important diagnostic challenge. By definition, subclinical hypothyroidism and subclinical hyperthyroidism are characterized by abnormal TSH with circulating thyroid hormones remaining within reference ranges. Although these states may be asymptomatic, endocrine and cardiovascular literature has repeatedly questioned the assumption that they are physiologically benign. The clinical significance of subclinical thyroid dysfunction is influenced by age, TSH magnitude, comorbid cardiovascular disease, and treatment exposure, but evidence supports a continuum rather than a sharp threshold between biochemical disturbance and end-organ effects [8]. This is clinically important because patients may develop early changes in ventricular filling, vascular resistance, or atrial electrophysiology before symptoms prompt cardiac evaluation.

The global epidemiology of thyroid disease also makes the question relevant to routine internal medicine. Hypothyroidism and hyperthyroidism are common across populations, with substantial variation by age, sex, iodine exposure, autoimmunity, assay characteristics, and diagnostic thresholds [9]. In India and other middle-income settings, the high volume of thyroid testing creates a large population in whom a pragmatic method for identifying cardiac involvement could be valuable. Echocardiography is more accessible than cardiac magnetic resonance or advanced deformation imaging in many tertiary and secondary hospitals, making conventional two-dimensional and Doppler measurements particularly useful for real-world screening and risk stratification.

Beyond intermediate echocardiographic endpoints, thyroid dysfunction has been linked to clinically important cardiovascular outcomes. Individual participant-data analyses have shown that higher TSH concentrations in subclinical hypothyroidism are associated with increased coronary heart disease

risk, particularly when TSH is at or above 10 mIU/L [10]. Similarly, pooled prospective cohort data have demonstrated a U-shaped relationship between TSH and incident heart failure, with higher risk at both marked TSH elevation and marked suppression [11]. These observations suggest that structural and functional cardiac abnormalities detected by echocardiography may represent an intermediate phenotype connecting endocrine dysfunction with later clinical events. Early detection may therefore have prognostic as well as descriptive value.

Emerging translational work has also refined understanding of thyroid-dependent myocardial biology. Thyroid hormone affects cardiomyocyte maturation, calcium handling, mitochondrial metabolism, and contractile protein expression, providing a mechanistic basis for reversible or partially reversible dysfunction when thyroid status is corrected [12]. Yet the degree of cardiac involvement varies substantially among individuals with similar thyroid hormone concentrations. Age, adiposity, blood pressure, treatment status, and duration of endocrine disturbance may modify the cardiac phenotype. Consequently, a clinically meaningful study should not only compare echocardiographic indices among thyroid categories but should also test whether biochemical markers such as TSH correlate with ventricular function and whether thyroid status retains an independent relationship with filling pressure after accounting for other clinical variables.

Against this background, the present study evaluated cardiac structure and function in adults with overt hyperthyroidism, subclinical hyperthyroidism, overt hypothyroidism, and subclinical hypothyroidism attending a tertiary-care teaching hospital in Ghaziabad, India. The primary objective was to compare conventional echocardiographic indices across these four thyroid states. Secondary objectives were to assess the relationships of TSH, FT3, and FT4 with left ventricular systolic and diastolic parameters and to determine whether thyroid status independently predicted E/e' after adjustment for age, BMI, systolic blood pressure, and thyroid biochemistry. By evaluating overt and subclinical disease within the same cohort, the study was designed to define a graded cardio-thyroid phenotype that could inform practical echocardiographic assessment in routine endocrine and internal-medicine care.

Materials and Methods

This observational cross-sectional study was conducted in the Department of General Medicine, Santosh Medical College & Hospital, Ghaziabad, Uttar Pradesh, India. Recruitment continued until the prespecified sample size was achieved. Adults aged 18 years or older with laboratory-defined thyroid dysfunction were enrolled and classified into four groups: overt hyperthyroidism (n=44), subclinical hyperthyroidism (n=38), overt hypothyroidism (n=82), and subclinical hypothyroidism (n=30), yielding a total sample of 194 participants. Patients with pre-existing coronary artery disease, heart failure, valvular heart disease, congenital heart disease, severe systemic illness, pregnancy, significant pulmonary disease including chronic obstructive pulmonary disease or pulmonary hypertension, or severe renal dysfunction were excluded. A detailed medical history and physical examination were obtained, including pulse rate, respiratory rate, systolic and diastolic blood pressure, and body mass index. Thyroid status was determined from TSH and circulating thyroid hormone concentrations using the laboratory criteria applied in the source study: overt hypothyroidism was characterized by high TSH with low thyroid hormone levels, subclinical hypothyroidism by high TSH with normal thyroid hormones, overt hyperthyroidism by suppressed TSH with elevated thyroid hormone levels, and subclinical hyperthyroidism by suppressed TSH with normal thyroid hormones. All participants underwent standardized resting transthoracic echocardiography performed by experienced sonographers blinded to thyroid-status grouping. The principal echocardiographic measures were left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), transmitral E/A ratio, and E/e' ratio; pulmonary artery systolic pressure was also assessed within the echocardiographic protocol. Continuous variables were summarized as mean±standard deviation or median with range when appropriate, and categorical variables as number and percentage. Between-group comparisons used one-way analysis of variance or Welch's ANOVA for continuous measures as appropriate, the Kruskal-Wallis test for non-normally distributed TSH, and chi-square tests for categorical data. Spearman rank correlation quantified relationships between thyroid-function tests and echocardiographic indices. A multivariable linear regression model was constructed with E/e' as the dependent variable and age, BMI, systolic blood pressure, TSH, FT4, and thyroid-status category as predictors. Statistical significance was defined at $p<0.05$.

Results

The study included 194 adults: 44 with overt hyperthyroidism, 38 with subclinical hyperthyroidism, 82 with overt hypothyroidism, and 30 with subclinical hypothyroidism. Overt hyperthyroid participants were the youngest group (36.8±8.89 years), whereas overt hypothyroid participants were the oldest (50.1±10.44 years). Women constituted 61.3% of the total cohort and predominated in overt hypothyroidism (75.6%) and

subclinical hypothyroidism (73.3%). Hyperthyroid states were characterized by higher pulse rate and systolic blood pressure, while hypothyroid states had lower pulse rates and higher diastolic pressure. BMI was substantially higher in overt and subclinical hypothyroidism than in hyperthyroid categories. Thyroid biochemistry showed the expected graded separation across the four diagnostic groups, supporting internal consistency of the classification.

Table 1: Clinical, anthropometric, and biochemical characteristics by thyroid-status group

Characteristic	Overt hyperthyroidism (n=44)	Subclinical hyperthyroidism (n=38)	Overt hypothyroidism (n=82)	Subclinical hypothyroidism (n=30)	Overall p-value
Age, years	36.8 ± 8.89	42.7 ± 8.41	50.1 ± 10.44	44.6 ± 8.98	<0.001*
Female, n (%)	21 (47.7)	14 (36.8)	62 (75.6)	22 (73.3)	<0.001†
Pulse, beats/min	94.4 ± 3.38	88.4 ± 4.69	65.4 ± 5.12	71.2 ± 6.74	<0.001‡
Systolic BP, mmHg	135.4 ± 9.20	130.2 ± 6.14	112.2 ± 8.48	115.2 ± 8.43	<0.001‡
Diastolic BP, mmHg	69.3 ± 5.15	70.3 ± 4.22	79.0 ± 4.99	76.8 ± 4.12	<0.001‡
BMI, kg/m²	21.2 ± 1.98	22.6 ± 2.06	27.0 ± 2.50	26.2 ± 2.16	<0.001*
FT3, pg/mL	5.94 ± 1.12	3.23 ± 0.41	1.46 ± 0.67	3.04 ± 0.37	<0.001‡
FT4, ng/dL	2.73 ± 0.73	1.37 ± 0.18	0.55 ± 0.16	1.23 ± 0.21	<0.001‡
TSH, µIU/mL	0.063 ± 0.045	0.204 ± 0.046	22.0 ± 13.4	7.87 ± 1.04	<0.001§

*One-way ANOVA for age/BMI; †chi-square for sex; ‡Welch one-way ANOVA; §Kruskal-Wallis for TSH. Values are mean ± SD unless otherwise stated.

Echocardiographic parameters differed substantially across thyroid states (Table 2). Overt hyperthyroidism demonstrated a hyperdynamic phenotype with LVEF 70.2±7.24%, while overt hypothyroidism showed LVEF 53.3±3.90%. LVEDD and LVESD increased progressively toward hypothyroid categories. Diastolic indices showed the same direction of deterioration: E/A was highest in overt hyperthyroidism (1.54±0.13) and lowest in overt hypothyroidism (0.915±0.22), whereas E/e' was lowest in overt hyperthyroidism (7.87±2.09) and highest in overt hypothyroidism (14.0±3.29). Subclinical hypothyroidism showed an intermediate but abnormal pattern (LVEF 56.0±3.06%; E/e' 11.2±1.51), suggesting detectable cardiac involvement before the overt biochemical state.

Table 2: Echocardiographic parameters across the four thyroid-status groups

Echocardiographic parameter	Overt hyperthyroidism	Subclinical hyperthyroidism	Overt hypothyroidism	Subclinical hypothyroidism	p-value
LVEF, %	70.2 ± 7.24	65.3 ± 2.69	53.3 ± 3.90	56.0 ± 3.06	0.001
LVEDD, mm	43.6 ± 2.07	44.9 ± 1.50	47.9 ± 5.48	49.5 ± 1.44	0.001
LVESD, mm	24.3 ± 3.13	25.5 ± 1.52	30.3 ± 5.43	31.4 ± 1.38	0.001
E/A ratio	1.54 ± 0.13	1.36 ± 0.07	0.915 ± 0.22	1.01 ± 0.09	0.001
E/e' ratio	7.87 ± 2.09	8.28 ± 0.88	14.0 ± 3.29	11.2 ± 1.51	0.001

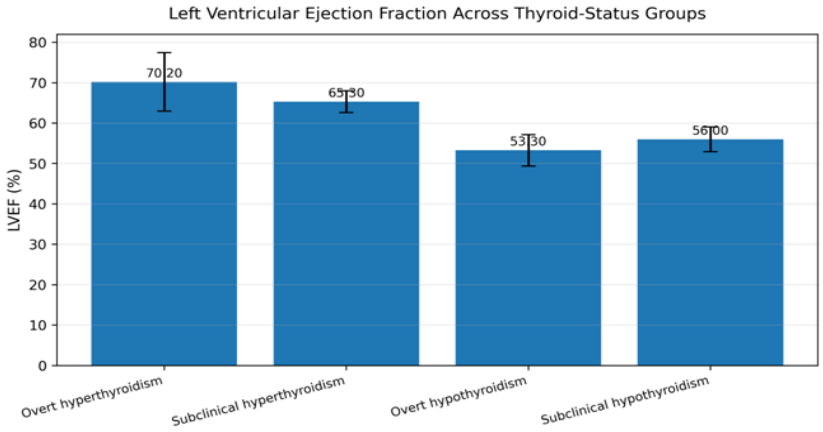


Figure 1: Mean left ventricular ejection fraction by thyroid-status group. Error bars indicate standard deviation

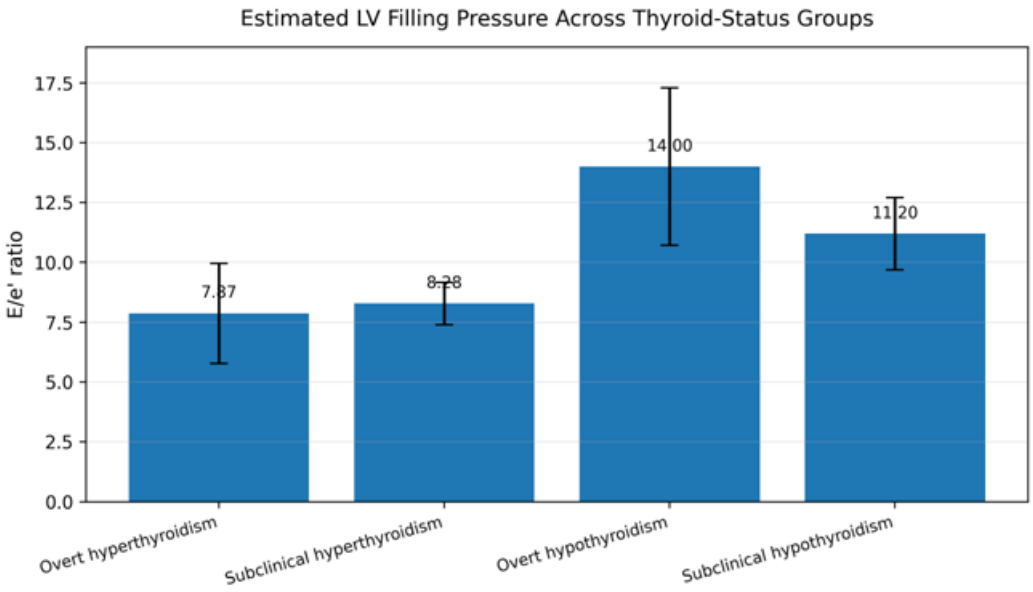


Figure 2: Mean E/e' ratio by thyroid-status group. Error bars indicate standard deviation

Correlation analysis demonstrated strong, directionally coherent relationships between thyroid biochemistry and cardiac function. TSH correlated inversely with LVEF (ρ=-0.838) and E/A (ρ=-0.833) and positively with LVEDD (ρ=0.480), LVESD (ρ=0.556), E/e' (ρ=0.815), and PASP (ρ=0.607), with all p<0.001. FT3 and FT4 showed the opposite pattern, correlating positively with LVEF and E/A and inversely with E/e'. In the multivariable model for E/e', higher age, BMI, TSH, and hypothyroid status remained independent predictors, while systolic blood pressure and FT4 were not independently significant. The model explained 57.3% of variability in E/e'.

Table 3: Thyroid-echo correlations and multivariable predictors of E/e'

Analysis / predictor	Effect estimate	SE	Test statistic	p-value	Interpretation
Spearman: TSH vs LVEF	ρ = -0.838	—	—	<0.001	Higher TSH associated with lower systolic function
Spearman: TSH vs E/A	ρ = -0.833	—	—	<0.001	Higher TSH associated with impaired relaxation
Spearman: TSH vs E/e'	ρ = 0.815	—	—	<0.001	Higher TSH associated with higher filling pressure
Regression: age, years	β = 0.0417	0.0180	t = 2.311	0.022	Independent positive predictor
Regression: BMI, kg/m²	β = 0.2749	0.0771	t = 3.566	<0.001	Independent positive predictor
Regression: systolic BP	β = -0.0091	0.0213	t = -0.425	0.671	Not independently significant
Regression: TSH, µIU/mL	β = 0.0831	0.0179	t = 4.635	<0.001	Independent positive predictor
Regression: FT4, ng/dL	β = 0.3666	0.2992	t = 1.225	0.222	Not independently significant
Regression: hypothyroid vs hyperthyroid	β = 2.0785	0.7455	t = 2.788	0.006	Hypothyroid status independently raises E/e'
Model fit	R = 0.757; R^2 = 0.573	—	—	—	57.3% of E/e' variance explained

Discussion

The present study demonstrates a clear, graded association between thyroid status and conventional echocardiographic measures of cardiac performance. Across 194 adults spanning overt and subclinical hyperthyroidism and hypothyroidism, the most striking pattern was the contrast between a hyperdynamic phenotype in overt hyperthyroidism and a lower-output, higher-filling-pressure phenotype in hypothyroidism.

Overt hyperthyroidism was associated with LVEF of 70.2%, E/A of 1.54, and E/e' of 7.87, whereas overt

hypothyroidism was associated with LVEF of 53.3%, E/A of 0.915, and E/e' of 14.0. These findings are physiologically coherent with the established effects of thyroid hormone on heart rate, contractility, relaxation, and systemic vascular resistance [1,2]. They also extend the mechanistic framework summarized by Razvi et al., in which myocardial and vascular thyroid-hormone signaling can generate clinically relevant changes in both systolic and diastolic performance [3].

The strong separation in systolic performance deserves careful interpretation. Hyperthyroidism increases beta-adrenergic responsiveness, venous return, and myocardial contractility while reducing systemic vascular resistance, producing a high-output state that can elevate LVEF in otherwise structurally intact hearts. In contrast, hypothyroidism reduces expression and activity of proteins involved in calcium cycling and contractile kinetics, diminishes chronotropy, and increases afterload. The resulting phenotype may include a lower ejection fraction even in the absence of established heart disease. The current findings are therefore compatible with contemporary reviews describing enhanced systolic function in uncomplicated thyrotoxicosis and reduced cardiac output in hypothyroidism [4,5]. Importantly, the difference was not confined to overt disease: subclinical hypothyroidism had an average LVEF of 56.0%, lower than both hyperthyroid groups, supporting the concept that myocardial effects can become detectable before marked reductions in circulating thyroid hormones.

The diastolic findings may be even more clinically important. Diastolic dysfunction can precede systolic impairment, and E/A together with tissue Doppler-derived E/e' provides a practical window into relaxation and filling pressure. The study showed a stepwise deterioration from hyperthyroid to hypothyroid states, with E/A decreasing and E/e' increasing. This pattern fits the standardized physiological interpretation recommended by echocardiography guidelines [7]. It also aligns with magnetic resonance evidence from Ripoli and colleagues, who found impaired cardiac pump performance in subclinical hypothyroidism despite the absence of overt clinical heart failure [13]. Although imaging methods differ, both datasets support the same biological proposition: thyroid hormone deficiency can impair ventricular relaxation and reduce effective myocardial performance before severe structural disease becomes apparent. The subclinical hypothyroid group is particularly noteworthy. Its E/e' of 11.2±1.51 was lower than the overt hypothyroid value but clearly higher than both hyperthyroid groups. This intermediate phenotype is consistent with the concept that subclinical thyroid dysfunction represents a continuum of end-organ exposure rather than a purely biochemical label [8]. Recent meta-analytic data strengthen this interpretation. Wang et al. evaluated 11 studies of levothyroxine supplementation in subclinical hypothyroidism and reported improvement in LVEF, cardiac output, and E/A after normalization of TSH [17]. A later meta-analysis focused on diastolic function found that thyroid hormone replacement improved E/A and myocardial deformation, although E/e' did not consistently normalize [18]. Together with the current cross-sectional observations, these studies suggest that at least part of the cardiac phenotype in subclinical hypothyroidism may be modifiable, while also cautioning that not all echocardiographic abnormalities necessarily reverse completely or rapidly.

The chamber-dimension findings also support structural adaptation to altered thyroid physiology. LVEDD increased from 43.6 mm in overt hyperthyroidism to 47.9 mm in overt hypothyroidism, while LVESD increased from 24.3 to 30.3 mm. In this cohort, the largest LVEDD and LVESD values were actually observed in subclinical hypothyroidism, although its LVEF and E/e' remained intermediate. This pattern may reflect heterogeneity in loading conditions, duration of disease, treatment exposure, body size, or unmeasured metabolic factors. Standard chamber-quantification guidance emphasizes that linear dimensions should be interpreted in clinical context and ideally indexed to body size when appropriate [6]. The higher BMI seen in hypothyroid groups is therefore relevant because body habitus may influence ventricular geometry and loading. The multivariable findings partly address this issue by showing that BMI independently predicted higher E/e' even after inclusion of thyroid biochemistry and thyroid-status category.

The correlation analysis provides strong internal support for a dose-response relationship between endocrine status and cardiac function. TSH showed large inverse correlations with LVEF and E/A and a large positive correlation with E/e'. FT3 and FT4 showed mirror-image relationships, being positively associated with LVEF and E/A and inversely associated with E/e'. Such directional consistency across multiple cardiac indices makes a chance pattern less likely, even though the cross-sectional design prevents causal inference. The findings are compatible with broader epidemiologic evidence demonstrating that cardiovascular risk rises at more extreme TSH concentrations [10,11]. They also correspond to molecular evidence that thyroid hormone availability directly influences calcium handling, mitochondrial energetics, and myofilament biology [12]. Thus, the statistical relationships observed here are supported by both clinical outcomes research and established myocardial physiology.

The multivariable analysis further refines the interpretation. TSH remained independently associated with E/e' after adjustment for age, BMI, systolic blood pressure, FT4, and thyroid-status grouping. Hypothyroid status itself also remained significant, increasing E/e' by approximately 2.08 units relative to hyperthyroid status in the fitted model. These findings suggest that the diastolic phenotype is not explained solely by age, body size, or blood pressure. Age and BMI were themselves independent predictors, which is clinically plausible because ventricular relaxation worsens with aging and increased adiposity is associated with altered loading, myocardial stiffness, and cardiometabolic risk. By contrast, systolic blood pressure and FT4 did not remain independently significant in this model. The model R^2 of 0.573 indicates that more than half of the observed variance in E/e' was explained by the included variables, a substantial proportion for a clinical cross-sectional dataset.

Hyperthyroid physiology also deserves contextualization. Although higher LVEF and lower E/e' may appear favorable when viewed in isolation, a hyperdynamic state is not necessarily benign. Chronic thyroid hormone excess can produce tachyarrhythmia, atrial remodeling, pulmonary hypertension, and eventually heart failure, particularly in older patients or those with underlying structural disease. Biondi and Kahaly reviewed these cardiovascular consequences across different causes of hyperthyroidism and emphasized that increased contractility and cardiac output coexist with important arrhythmic and hemodynamic risks [15]. Earlier interventional work by Sgarbi et al. showed that treatment of endogenous subclinical hyperthyroidism can improve clinical and cardiac abnormalities, illustrating that a hyperdynamic phenotype can itself represent pathophysiology rather than cardiovascular advantage [14]. Therefore, the higher LVEF observed in the hyperthyroid groups should be understood as part of an altered hemodynamic state, not simply as superior myocardial health.

The clinical implications of these data are practical. In a patient with thyroid dysfunction, a normal or high LVEF alone may be insufficient to characterize cardiac involvement. A focused thyroid-echo assessment should consider chamber dimensions, transmitral filling, tissue Doppler indices, and filling pressure, particularly when symptoms such as dyspnea, fatigue, palpitations, or exercise intolerance are present. The pronounced E/e' elevation in overt hypothyroidism and the intermediate abnormality in subclinical hypothyroidism suggest that diastolic assessment may provide additional information beyond conventional systolic imaging. This is especially relevant in settings where advanced strain imaging or cardiac magnetic resonance is not routinely available. Conventional Doppler echocardiography, when acquired and interpreted according to guideline principles, remains a scalable method for identifying clinically meaningful cardiac involvement [6,7].

The findings also resonate with newer cardiovascular-endocrine literature. A 2024 review of hypothyroidism and cardiovascular disease summarized evidence that hypothyroidism reduces cardiac output, impairs left ventricular function, and increases vascular resistance, while also clustering with dyslipidemia and hypertension [16]. The 2026 European Heart Journal review similarly emphasized impaired ventricular filling and relaxation in hypothyroidism and high-output physiology in hyperthyroidism, while noting that treatment effects on hard cardiovascular outcomes remain incompletely defined [5]. The present study contributes a granular echocardiographic dataset from an Indian tertiary-care population and shows that these canonical physiological differences are readily detectable using conventional measurements.

Several limitations should be considered. The cross-sectional design does not establish temporality and cannot determine whether the observed abnormalities reverse after normalization of thyroid function. Treatment exposure was present in a substantial proportion of participants, and detailed duration or adequacy of therapy was not incorporated into the multivariable model. The study was performed at a single tertiary-care center, which may limit generalizability. Although important cardiovascular comorbidities were excluded, residual confounding from disease duration, dyslipidemia, physical activity, sleep-disordered breathing, glycemic status, and other metabolic factors cannot be excluded. Advanced echocardiographic techniques such as global longitudinal strain were not included, and left atrial volume index was not reported. In addition, the source study did not specify a fixed calendar recruitment interval, reporting instead that recruitment continued until the planned sample size was achieved. Despite these limitations, the consistency of between-group differences, biomarker correlations, and adjusted associations supports the central finding that thyroid state is closely linked to measurable cardiac structure and function. In summary, the present data support a spectrum model of thyroid-related cardiac involvement. Hyperthyroid states were characterized by higher heart rate and hyperdynamic systolic function, whereas hypothyroid states demonstrated larger ventricular dimensions, lower LVEF, impaired transmitral filling, and higher estimated filling pressure. Subclinical hypothyroidism occupied an intermediate position, indicating that echocardiographic abnormalities

may emerge before overt hormone deficiency. The independent association of TSH and hypothyroid status with E/e' strengthens the case for considering diastolic function in the cardiovascular assessment of patients with thyroid disorders.

Conclusion

Thyroid dysfunction was associated with distinct and graded echocardiographic patterns in this cohort. Hyperthyroidism produced a hyperdynamic systolic profile, whereas hypothyroidism was associated with lower LVEF, larger left ventricular dimensions, impaired relaxation, and higher estimated filling pressure. Subclinical hypothyroidism demonstrated intermediate abnormalities, suggesting early myocardial involvement before overt hormone deficiency. Higher TSH, older age, higher BMI, and hypothyroid status independently predicted increased E/e'.

Conventional transthoracic echocardiography can therefore provide clinically useful assessment of cardiac involvement in patients with thyroid disorders, particularly when diastolic dysfunction is suspected.

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