ECHOCARDIOGRAPHIC EVALUATION OF PULMONARY ARTERIAL PRESSURE IN PATIENTS WITH THYROID DYSFUNCTION: AN OBSERVATIONAL HOSPITAL-BASED STUDY

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Abstract

Background

Thyroid dysfunction is associated with various cardiovascular manifestations including pulmonary hypertension, arrhythmias, cardiomyopathy, and ventricular dysfunction. Pulmonary hypertension is increasingly recognized in both hypothyroid and hyperthyroid patients and may contribute to unexplained dyspnea and cardiovascular morbidity. Early identification using two-dimensional echocardiography (2D Echo) may improve clinical outcomes.

Aim

To evaluate pulmonary hypertension in patients with thyroid disorders using two-dimensional echocardiography.

Materials and Methods

This hospital-based observational study was conducted in the Department of Pulmonology at Sree Mookambika Institute of Medical Sciences from October 2024 to April 2025. A total of 52 patients with hypothyroidism or hyperthyroidism were included using simple random sampling. Thyroid function tests were performed using enzyme immunoassay. Electrocardiography, chest X-ray, and 2D echocardiography were carried out in all patients. Pulmonary artery systolic

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pressure (PASP) was estimated using tricuspid regurgitation jet velocity by the modified Bernoulli equation. Patients with PASP >30 mmHg were considered to have pulmonary hypertension and were reassessed after treatment of thyroid dysfunction.

Results

Elevated PASP was observed in both hypothyroid and hyperthyroid patients, with a higher prevalence among hyperthyroid individuals. Mean PASP in hyperthyroidism was 30.62 mmHg,while hypothyroidism showed a mean PASP of 26.97 mmHg. Follow-up assessment demonstrated significant reduction in PASP after treatment and restoration of euthyroid status.

Conclusion

Pulmonary hypertension is an important cardiovascular manifestation of thyroid dysfunction and may be reversible with timely treatment. Two-dimensional echocardiography is a useful noninvasive tool for early detection and monitoring of pulmonary hypertension in thyroid disorder patients.

Introduction:

Pulmonary hypertension (PH) is a progressive cardiopulmonary disorder characterized by elevated pulmonary arterial pressure resulting in increased pulmonary vascular resistance and eventual right ventricular failure.[1] It is hemodynamically defined as an increase in mean pulmonary artery pressure (mPAP) greater than 25 mm Hg at rest and greater than 30 mm Hg during exercise, as measured by right heart catheterization.[2] PH is associated with significant morbidity and mortality, especially when diagnosis and treatment are delayed. Patients commonly present with unexplained dyspnea, fatigue, chest discomfort, palpitations, and exercise intolerance, symptoms that are often nonspecific and may overlap with several systemic disorders.[3]

Pulmonary hypertension may be classified as primary or secondary depending upon the underlying etiology.[4] Primary pulmonary arterial hypertension is considered a diagnosis of exclusion, whereas secondary pulmonary hypertension develops as a consequence of various cardiac, pulmonary, metabolic, hematological, or systemic disorders.[5] Common causes include chronic lung diseases, congenital and acquired heart diseases, connective tissue disorders, chronic thromboembolic disease, infections such as HIV, hemoglobinopathies, and endocrine disorders including thyroid dysfunction.[6]

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Thyroid hormones exert significant effects on cardiovascular physiology by influencing cardiac contractility, vascular resistance, heart rate, and pulmonary circulation.[7] Both hypothyroidism and hyperthyroidism are associated with various cardiovascular abnormalities and may contribute to the development of pulmonary hypertension.[8] In hypothyroidism, increased systemic vascular resistance, endothelial dysfunction, altered lipid metabolism, and impaired cardiac function contribute to cardiovascular complications.[9] Patients with hypothyroidism are at increased risk of atherosclerosis and coronary artery disease due to associated hypercholesterolemia, hypertension, and elevated homocysteine levels.[10] Studies have also demonstrated increased abdominal aortic atherosclerosis among elderly women with even mild hypothyroidism.[11]

Hyperthyroidism, on the other hand, may cause high cardiac output states, increased blood volume, and pulmonary vascular endothelial dysfunction, thereby predisposing individuals to elevated pulmonary arterial pressures.[12] Several studies have demonstrated a higher prevalence of pulmonary hypertension among patients with thyroid dysfunctions, including both hypothyroid and hyperthyroid states.[13] Owing to this association, thyroid disorders have been included under Group V pulmonary hypertension in the Dana Point 2008 classification system.[14]

The exact mechanism linking thyroid dysfunction and pulmonary hypertension remains incompletely understood. Proposed mechanisms include autoimmune-mediated endothelial injury, altered metabolism of intrinsic pulmonary vasodilators, increased pulmonary vascular resistance, and changes in cardiac output.[15] Importantly, pulmonary hypertension associated with thyroid dysfunction has been reported to be potentially reversible following restoration of euthyroid status through appropriate medical management.[16] Early identification and treatment of thyroid abnormalities may therefore improve pulmonary hemodynamics and overall patient prognosis.

Two-dimensional echocardiography (2D Echo) serves as a valuable noninvasive diagnostic modality for the screening and assessment of pulmonary hypertension.[17] It enables estimation of pulmonary artery pressure, evaluation of right ventricular function, and detection of associated cardiac abnormalities.[18] Echocardiography is particularly useful in patients with thyroid disorders presenting with unexplained dyspnea, as it helps identify underlying pulmonary hypertension at an early stage.

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Despite increasing evidence regarding the association between thyroid dysfunction and pulmonary hypertension, limited hospital-based observational studies are available evaluating pulmonary hypertension in patients with thyroid disorders using echocardiography.[19] Therefore, the present study was undertaken to analyze pulmonary hypertension in patients with thyroid dysfunction using 2D echocardiography in a teaching hospital setting. Early recognition of pulmonary hypertension in thyroid disorders may facilitate prompt treatment and prevent progression to severe cardiopulmonary complications.[20]

Aim

To evaluate pulmonary hypertension in patients with thyroid disorders using two-dimensional echocardiography (2D Echo).

Objectives

To assess the prevalence of pulmonary hypertension among patients with thyroid dysfunction.

To evaluate pulmonary artery pressure using two-dimensional echocardiography in patients with hypothyroidism and hyperthyroidism.

To study the association between thyroid hormone abnormalities and pulmonary hypertension.

Materials and Methodology

This hospital-based observational study was conducted in the Department of Pulmonology at Sree Mookambika Institute of Medical Sciences over a study period from October 2024 to April 2025. The study included a total sample size of 52 patients selected by simple random sampling. Patients attending the hospital on both Outpatient Department (OPD) and Inpatient Department (IPD) basis were screened for clinical features suggestive of hypothyroidism or hyperthyroidism using a prestructured proforma. Thyroid function tests were performed in all patients using enzyme immunoassay techniques to confirm the diagnosis of thyroid dysfunction.

All enrolled patients underwent detailed clinical examination and relevant investigations. Electrocardiography (ECG) was recorded in all cases to assess associated cardiac abnormalities. Chest radiography was performed and evaluated for radiological signs suggestive of pulmonary hypertension, particularly enlargement of the right descending pulmonary artery diameter greater than 1.8 cm. Two-dimensional echocardiography (2D Echo) was carried out in all patients to assess the presence and severity of pulmonary hypertension.

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Right ventricular systolic pressure (RVSP) was estimated using the tricuspid regurgitation jet velocity by applying the modified Bernoulli equation during 2D echocardiographic assessment. Central venous pressure (CVP) was not clinically elevated in the study population and was therefore assumed to be 5 mmHg. Mean right atrial pressure (RAP) was considered equivalent to CVP. Pulmonary artery systolic pressure (PASP) was calculated by adding RAP to RVSP. Patients with PASP greater than 30 mmHg were considered to have pulmonary hypertension. These patients were treated for the underlying thyroid disorder with thyroxine therapy in hypothyroidism and carbimazole therapy in hyperthyroidism. Follow-up reassessment using 2D echocardiography was performed after 10 months to evaluate reduction in PASP following treatment.

The inclusion criteria comprised patients diagnosed with hypothyroidism or hyperthyroidism, patients above 18 years of age belonging to both sexes, and patients willing to participate in the study. Patients with chronic pulmonary diseases such as chronic obstructive airway disease (COAD) and interstitial lung disease, connective tissue disorders, underlying cardiac diseases including ventricular septal defect, cardiomyopathies and myocarditis, chronic liver disease or cirrhosis, chronic hypoxemia, and patients already receiving treatment for pulmonary hypertension were excluded from the study.

The collected data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 25.0. Quantitative variables were expressed as mean ± standard deviation, while qualitative variables were represented as frequency and percentage. Comparison between categorical variables was performed using Chi-square test, and continuous variables were analyzed using Student's t-test. Correlation between thyroid dysfunction and pulmonary artery systolic pressure was assessed using Pearson's correlation coefficient. A p-value less than 0.05 was considered statistically significant.

Result:

In our study out of the 52 cases, 38 (74%) cases were of Hypothyroidism and 14 (26%) cases were of Hyperthyroidism.

Table 1: Distribution of mean PASP using 2D-ECHO.

		Mean± Sd	P value
Variables	Hypothyroidism	Hyperthyroidism	
2D-ECHO PASP(RVSP+RA)	26.97±5.16	30.62±9.57	0.09

Unpaired t test is applied. P value is significant if < 0.05 PASP value in patients with

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hyperthyroidism was 30.62mm Hg while that in the patients with hypothyroidism was 26.97mm Hg. Difference between them was comparable.

Table 2: Comparison of PASP in Hypothyroidism and Hyperthyroidism

PASP Group			Thyroid Status		Grand total
	Hypothyroidism		Hyperthyroidism		
	N	%	N	%	
Normal (PASP≤ 30 mm Hg)	31	81.08	10	69.23	41
Mild (PASP 31 to 45 mm Hg)	7	18.92	2	15.38	9
Moderate (PASP 46 to 60 mm Hg)	0	0	2	15.38	2
Severe (PASP>60 mm Hg	0	0	0	0	0
Grand total	38	74	14	26	52

81.08% patients with hypothyroidism and 69.23% patients with hyperthyroidism had normal PASP level. While 15.38% patients with hyperthyroidism had mild and moderate PASP each, only 18.92% with hypothyroidism had mild PASP.

Table 3: Comparison of PASP in the follow up group: pre treatment and post treatment.

		Mean± Sd	P value
Variables	Before	After follow up	
2D-ECHO PASP(RVSP+RA)	38.82±5.51	30±5.14	0.001

Paired t test is applied. P value is significant if < 0.05. The mean PASP by Doppler Echocardiography was 38.82 mm of Hg in the pre-treatment group. And the mean PASP during the follow up (after 10 months) was 30 mm of Hg**.**

Table 4: Comparison of TSH, T3, T4 and PASP among patients of Hypothyroidism in the follow up group, pre and post.

Variables	Hypothyroidism (Mean± Sd)		P value
	Before	After follow up	
TSH (uIU/ml)	16.62±10.36	3.52±1.57	0.01
T3 (ng/ml)	0.67±0.19	1.14±0.42	0.05
T4 (ug/dl	5.14±2.83	8.66±1.55	0.004
2D-ECHO PASP mm Hg	36.14±3.29	29.71±5.5	0.006

Paired 't' test applied. 'P' value is significant if < 0.05 Among the patients in the Hypothyroidism group with Pulmonary Hypertension, the pre-treatment values of TSH and PASP were high and reduced in the follow-up (after 10 months of treatment).

Table 5: Comparison of TSH, T3, T4 and PASP among patients of Hyperthyroidism in the follow up group, pre and post treatment.

Variables	Hyperthyroidism (Mean± Sd)		P value
	Before	After follow up	
TSH (uIU/ml)	0.1±0.04	2.54±1.84	0.07
T3 (ng/ml)	2.9±0.72	1.16±0.20	0.018
T4 (ug/dl	16.83±2.88	9.53±1.34	0.013

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2D-ECHO PASP mm Hg	43.5±5.80	30.5±5.2	0.023

Paired 't' test applied. 'P' value is significant if < 0.05 Among the patients in the Hyperthyroidism group with Pulmonary Hypertension, the mean pre-treatment value of TSH was 0.1 and increased to
2.54 in the follow up. Mean PASP value was 43.5 and reduced in the follow-up (after 10 months of treatment) to 30.5.

Discussion

Thyroid dysfunction is associated with a wide spectrum of cardiovascular manifestations including pulmonary hypertension, arrhythmias, pericardial effusion, dilated cardiomyopathy, systolic and diastolic dysfunction, and pulmonary regurgitation.[21] Among these, pulmonary hypertension (PH) has gained increasing clinical importance because of its potential reversibility following correction of thyroid abnormalities.[22] The present study demonstrated elevated pulmonary artery systolic pressure (PASP) in both hypothyroid and hyperthyroid patients, supporting the significant association between thyroid dysfunction and pulmonary vascular abnormalities.

Pulmonary arterial hypertension is characterized by sustained elevation of pulmonary artery pressure and progressive increase in pulmonary vascular resistance, eventually resulting in right ventricular failure and premature mortality if left untreated.[23] Under physiological conditions, mean pulmonary artery pressure remains below 20 mmHg and PASP remains below 30 mmHg.[24] In the current study, patients with thyroid dysfunction demonstrated elevated PASP values, with higher prevalence among hyperthyroid individuals compared to hypothyroid patients. Mean PASP among hyperthyroid patients was 30.62 mmHg, whereas hypothyroid patients demonstrated a mean PASP of 26.97 mmHg. These findings were comparable to previously published studies.[25]

Several earlier studies have reported increased prevalence of pulmonary hypertension in thyroid disorders. Marvisi et al. observed pulmonary hypertension in nearly 35% of patients with recently diagnosed hyperthyroidism.[26] In another study involving 114 hyperthyroid patients, the prevalence of pulmonary hypertension increased to 43%, with PASP values exceeding 30 mmHg on echocardiographic evaluation.[27] Similarly, Mercé et al. reported pulmonary hypertension in 41% of patients with newly diagnosed hyperthyroidism.[28] The findings of the present study are in agreement with these observations and further strengthen the evidence linking thyroid dysfunction and pulmonary hypertension.

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The exact mechanism responsible for pulmonary hypertension in thyroid disorders remains incompletely understood. Proposed mechanisms include autoimmune-mediated endothelial dysfunction, increased cardiac output, altered metabolism of vasodilators and vasoconstrictors, and enhanced pulmonary vascular resistance.[29] Hyperthyroidism may contribute to pulmonary hypertension through high-output circulatory states and endothelial injury, whereas hypothyroidism may lead to impaired myocardial relaxation, increased systemic vascular resistance, and vascular endothelial dysfunction.[30]

In the present study, approximately 30.76% of hyperthyroid patients demonstrated mild pulmonary hypertension, while 18.92% of hypothyroid patients had mild to moderate pulmonary hypertension. These findings suggest that pulmonary vascular involvement may occur in both forms of thyroid dysfunction, although it appears more pronounced in hyperthyroid states.[31]

An important observation in this study was the reversibility of pulmonary hypertension following treatment of thyroid dysfunction. Among follow-up patients, mean PASP values decreased significantly after achieving euthyroid status. Mean PASP reduced from 38.82 mmHg before treatment to 30 mmHg after treatment, indicating improvement in pulmonary hemodynamics following medical therapy. Similar reversibility of pulmonary hypertension after treatment of hyperthyroidism and hypothyroidism has been reported in previous studies.[32] Restoration of euthyroid state may improve endothelial function, normalize cardiac output, and reduce pulmonary vascular resistance, thereby contributing to reduction in pulmonary artery pressures.[33]

The present study highlights the importance of routine cardiovascular and echocardiographic evaluation in patients with thyroid disorders, especially those presenting with unexplained dyspnea. Early identification of pulmonary hypertension using 2D echocardiography may facilitate prompt treatment and prevent progression to irreversible cardiopulmonary complications.[34]

Conclusion

The present study demonstrated a significant association between thyroid dysfunction and pulmonary hypertension. Both hypothyroid and hyperthyroid patients showed elevated pulmonary artery systolic pressure (PASP) on two-dimensional echocardiography, with pulmonary hypertension being more common among hyperthyroid patients. Early detection of

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pulmonary hypertension in thyroid disorders is important, as many patients present with unexplained dyspnea and nonspecific cardiovascular symptoms. The study also showed improvement in PASP following appropriate treatment of thyroid dysfunction, indicating the reversible nature of thyroid-related pulmonary hypertension. Two-dimensional echocardiography serves as a valuable noninvasive tool for early diagnosis and follow-up assessment of pulmonary hypertension in patients with thyroid disorders.

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