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ORIGINAL ARTICLE

Assessment of thyroid function in idiopathic pulmonary hypertension

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ABSTRACT:

Background: Pulmonary artery hypertension is a hemodynamic and pathophysiological condition defined as an increase in mean pulmonary artery pressure (mPAP) > 25 mm Hg at rest and > 30 mm Hg during exercise. The present study was conducted to assess thyroid function in idiopathic pulmonary hypertension. **Materials & Methods:** 82 patients of idiopathic pulmonary hypertension of both genders were enrolled. The evaluation of RV dysfunction was done by both a thorough physical examination and echocardiographic examination using a 2 MHz GE S4 Probe. The thyroid markers (TSH, thyroxine [T4], and triiodothyronine [T3]) were assessed by electrochemiluminescence. The functional capacity was studied using the SMWT. **Results:** Out of 82 patients, males were 50 and females were 32. The meanT3 was 123.8n/dl, T4 was 8.9mcg/dl, TSH was 4.1mIU/ml, PAP was 50.4mmHg, CVP was 9.5mmHg, RVEDD was 3.7 cm, TAPSE was 1.9 mm and SMWT was 370.4 m. The mean T3 level of TAPSE >15 mm was 125.2, T4 was 8.5 and TSH was 4.1, <15 mm of T3 was 113.2, T4 was 8.3 and TSH was 4.8. The difference was significant in T3 (P< 0.05). The mean T3, T4 and TSH level of RVEDD > 3cm and < 3 cm was 121.4 and 124,7, 8.7 and 9.9 and 4.5 and 3.4 respectively. The difference was significant in RVEDD (P< 0.05). The mean T3, T4 and TSH level of SMWT > 200 m and < 200 m was 130.4 and 119.2, 9.0 and 8.6 and 3.6 and 5.2 respectively. The difference was significant in SMWT (P< 0.05). The mean T3, T4 and TSH level of CVP >5 mm Hg and < 5 mm Hg found to be 125.7 and 130.2, 8.7 and 10.6 and 4.5 and 3.9 respectively. **Conclusion:** Idiopathic pulmonary hypertensionis associated with subclinical hypothyroidism and low patient functional capacity.

Key words: Pulmonary artery hypertension, vascular resistance, Thyroid

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INTRODUCTION

Pulmonary hypertension is a complex, multidisciplinary disorder defined as an increase in the mean pulmonary arterial pressure (mPAP) of 25 mmHg at rest, assessed by right heart catheterization (RHC). It refers to the presence of high pulmonary vascular resistance, and can be the end result of a variety of different underlying disorders.[1] It is not very common, but with the new developments taking place in diagnostic and management techniques, pulmonary arterial hypertension (PAH) patients are identified earlier every day. PAH is more common in women (1.7:1) with a mean age of 36 years and chronic right ventricular (RV) pressure overload and RV failure can lead to end-stage organ damage (including the kidneys, liver, and thyroid gland), as well as many other hemodynamic changes that could be fatal. Early diagnosis and management can be critical and lifesaving in such cases.[2]

The principal alterations seen in PAH are vasoconstriction, remodeling and in situ thrombosis. There is endothelial dysfunction, which leads to impaired production of vasodilators, such as NO and prostacyclin, and increased expression of vasoconstrictors and mitogens, such as endothelin-1.[3] Thyroid hormones may play an important role in regulating PASP. However, the association of thyroid hormones with PH remains controversial.[4]Cardiovascular manifestations in hyperthyroidism occur frequently with various phenotypes. These include sinus tachycardia, atrial fibrillation, dilated cardiomyopathy, and highoutput congestive heart failure.[5] Recent studies have suggested a possible association between hyperthyroidism and pulmonary arterial hypertension. Although the exact mechanism of this combination has not yet been established, it has been hypothesized that thyroid hormones and autoimmunity have an important influence.[6]The present study was conducted to assess thyroid function in idiopathic pulmonary hypertension.

MATERIALS & METHODS

The present study comprised of 82 patients of idiopathic pulmonary hypertensionof both genders. The consent was obtained from all patients.

Data such as name, age, gender etc. was recorded. The evaluation of RV dysfunction was done by both a thorough physical examination and echocardiographic examination using a 2 MHz GE S4 Probe. The thyroid markers (TSH, thyroxine [T4], and triiodothyronine [T3]) were assessed by electrochemiluminescence. The functional capacity was studied using the SMWT.

Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 82		
Gender	**Males**	**Females**
Number	50	32

Table I shows that out of 82 patients, males were 50 and females were 32.

Table II Baseline characteristics

Parameters	Mean	SD
T3 (n/dl)	123.8	11.7
T4 (mcg/dl)	8.9	2.3
TSH (mIU/ml)	4.1	1.1
PAP (mmHg)	50.4	32.1
CVP (mmHg)	9.5	4.8
RVEDD (cm)	3.7	1.2
TAPSE (mm)	1.9	0.7
SMWT (m)	370.4	28.5

Table II, graph I shows that meanT3 was 123.8n/dl, T4 was 8.9mcg/dl, TSH was 4.1mIU/ml, PAP was 50.4mmHg, CVP was 9.5mmHg, RVEDD was 3.7 cm, TAPSE was 1.9 mm and SMWT was 370.4 m.

Graph I Baseline characteristics

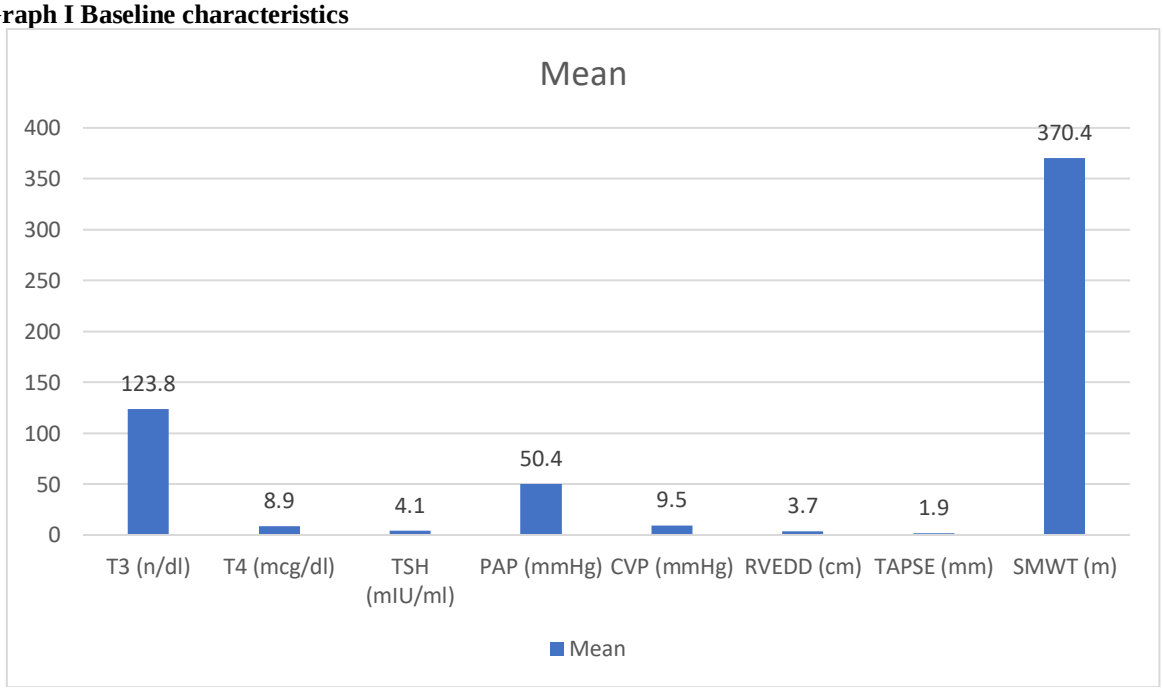

Table III Comparisons of thyroid hormone levels with Echocardiographic, hemodynamicand functional parameters

Parameters	Variables	T3 (n/dl)	T4 (mcg/dl)	TSH (mIU/ml)
TAPSE	>15 mm	125.2	8.5	4.1
	<15 mm	113.2	8.3	4.8
P value		0.05	0.91	0.94
RVEDD	>3 cm	121.4	8.7	4.5
	< 3 cm	124.7	9.9	3.4
P value		0.93	0.97	0.05
SMWT	>200 m	130.4	9.0	3.6
	<200 m	119.2	8.6	5.2
P value		0.14	0.95	0.01
CVP	>5 mm Hg	125.7	8.7	4.5
	<5 mm Hg	130.2	10.6	3.9
P value		0.17	0.07	0.06

Table III shows that the mean T3 level of TAPSE >15 mm was 125.2, T4 was 8.5 and TSH was 4.1, <15 mm of T3 was 113.2, T4 was 8.3 and TSH was 4.8. The difference was significant in T3 (P< 0.05). The mean T3, T4 and TSH level of RVEDD > 3cm and < 3 cm was 121.4 and 124,7, 8.7 and 9.9 and 4.5 and 3.4 respectively. The difference was significant in RVEDD (P< 0.05).The mean T3, T4 and TSH level of SMWT > 200 m and < 200 m was 130.4 and 119.2, 9.0 and 8.6 and 3.6 and 5.2 respectively. The difference was significant in SMWT (P< 0.05).The mean T3, T4 and TSH level of CVP >5 mm Hg and < 5 mm Hg found to be 125.7 and 130.2, 8.7 and 10.6 and 4.5 and 3.9 respectively.

DISCUSSION

Pulmonary arterial hypertension (PAH) is characterized by a sustained increase in pulmonary artery pressure and a progressive increase in pulmonary vascular resistance, leading to right ventricular insufficiency and premature death. Classically, pulmonary hypertension was divided into primary (idiopathic) and secondary forms.[7]Idiopathic PAH is more common in women than in men (ratio, 1.7:1), and the mean age at diagnosis is 36 years.[8] In many cases, the diagnosis of PAH is delayed, since the symptoms are nonspecific and can be confused with those of other, more common diseases. Dyspnea is the initial symptom in 90% of patients. Less common symptoms include fatigue, chest pain, syncope, peripheral edema and palpitations.[9]However, within the secondary PAH category, there are conditions that are similar to those of primary PAH, in terms of histopathological characteristics as well as response to treatment. The World Health Organization has periodically offered classifications of PAH.[10]The present study was conducted to assess pulmonary artery hypertension withechocardiography in thyroid dysfunction.

In present study, out of 82 patients, males were 50 and females were 32.Marvisi et al[11] demonstrated that in population with recently diagnosed hyperthyroidism without antithyroid treatment, PASP was associated with thyroid-stimulating hormone (TSH) and free thyroxine (FT4) levels. The prevalence of PAH in recently diagnosed hyperthyroidism was found to be 35% 6 while in his other study, involving 114 patients with hyperthyroidism (47 with Graves' disease and 67 with multinodular goiter), the prevalence of PAH was 43%. Here PASP, as estimated by echocardiography, was > 30 mmHg.

The effects of thyroid hormones on myocardium and vascular system are well known, but the mechanisms underlying pulmonary arterial hypertension associated with hyperthyroidism have not been clearly identified. Possible mechanisms include direct effects of thyroid hormone and immunemediated endothelial damage and/or dysfunction.[12]

We found that meanT3 was 123.8n/dl, T4 was 8.9mcg/dl, TSH was 4.1mIU/ml, PAP was 50.4mmHg, CVP was 9.5mmHg, RVEDD was 3.7 cm, TAPSE was 1.9 mm and SMWT was 370.4 m. There have been few studies that have evaluated thyroid function in IPAH. Since subclinical hypothyroidism can be seen in chronic diseases like heart failure, RV failure in IPAH can contribute to increased TSH levels and low functional capacity in these patients. Immunological disorders can sometimes be the cause for thyroid disease, and "auto-antibodies" can also contribute to the physiological pathway of PAH in these patients.[13]

Curnock et al[14] characterized the prevalence of hypothyroidism in a population with primary pulmonary hypertension (PPH).Of the 40 patients with PPH included in the study (11 men and 29 women), ages ranged from 11 to 76 years (mean 43.5 years). The mean pulmonary artery pressure was 58.7 mm Hg. Thirty-three patients had normal serum TSH levels (3 of whom were on levothyroxine supplement); 1 had low TSH; 5 had high TSH (range, 6.8-9.9 U/L, mean 8.4 U/L), and 1 had low thyroxine (T4 < 1.0 microg/dL). Nine of 40 patients (22.5%) had evidence of hypothyroidism, which is much more than expected in the general population of similar age range (2.8% in men, 7.5% in women).The prevalence of hypothyroidism in patients with PPH is high (22.5%). Patients with PPH should be investigated for the possibility of coexisting hypothyroidism.

CONCLUSION

Authors found that Idiopathic pulmonary hypertensionis associated with subclinical hypothyroidism and low patient functional capacity.

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