ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 6, Issue – 3, June - 2023, Page No. : 277 - 281

Pulmonary hypertension in hypothyroidism and hyperthyroidism

[1]Dr. Snehanshu Kumar, Junior Resident, Department of Internal Medicine, Patna Medical College and Hospital, Patna.

[2]Dr. Ganesh Prasad, Associate Professor, Department of Internal Medicine, Patna Medical College and Hospital, Patna.

[3]Dr. Kumar Gaurav, Senior Resident, Department of Internal Medicine, Patna Medical College and Hospital, Patna.

Corresponding Author: Dr. Snehanshu Kumar, Junior Resident, Department of Internal Medicine, Patna Medical College and Hospital, Patna.

How to citation this article: Dr. Snehanshu Kumar, Dr. Ganesh Prasad, Dr. Kumar Gaurav, "Pulmonary hypertension in hypothyroidism and hyperthyroidism", IJMACR- June - 2023, Volume – 6, Issue - 3, P. No. 277 – 281.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Introduction: Pulmonary Arterial Hypertension (A subgroup of Pulmonary Hypertension) is characterized by a persistent rise in Pulmonary Artery Pressure and a progressive rise in Pulmonary Vascular Resistance giving rise to Right Ventricular insufficiency and premature death.

Objective: To study the occurrence of Pulmonary Hypertension in Hypothyroidism and Hyperthyroidism and reversibility of Pulmonary Hypertension after treatment of Hypothyroidism and Hyperthyroidism.

Materials and Methods: The present study was a cohort (Prospective) study conducted from October 2020 to March 2022 in the Department of Internal Medicine at PMCH, Patna.

Result: Pulmonary Hypertension was detected in both Hypothyroidism (20.51 %) and Hyperthyroidism (27.27%) groups. The mean Pulmonary Artery systolic pressure (by Doppler ECHO) was 37.72 mm of Hg (±5.49) in pretreatment group and mean Pulmonary Artery systolic pressure during the follow up (after 18 months) was 31 mm of Hg (± 5.20).

Conclusion: Hypothyroidism/Hyperthyroidism may cause Pulmonary Hypertension which can be reversed after correction of Hypothyroidism/Hyperthyroidism.

Keywords: Pulmonary Hypertension, Hypothyroidism, Hyperthyroidism.

Introduction

"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" [1]. The definition of PAH is based on hemodynamic criteria: MPAP > 20 mmHg at rest with pulmonary capillary wedge pressure or left atrial pressure < 15 mmHg and pulmonary vascular resistance > 3 wood units. Mean pulmonary artery pressure (MPAP), under physiological conditions is < 20

(approximately 14) mmHg, and pulmonary artery systolic pressure (PASP) is < 30mmHg.[2]

MPAP = (CO x PVR) + LA Pressure (PCWP)

"The World Health Organization has periodically offered classifications of PH, the current classification being the result of a 6th world symposium held in 2018 at NICE, France which recognizes five groups of pulmonary hypertensions of which PAH is a sub-group." [3]

Group – 1: PAH (due to increased PVR)

Group – 2: PH (due to left heart disease)

Group – 3: PH (due to lung disease)

Group – 4: CTEPH

Group – 5: PH with unclear mechanism

PH v/s PAH

PH - MPAP > 20 mmHg

PAH - Type 1 PH

MPAP > 20 mmHg

PVR > 3 wood units

PCWP < 15 mmHg + specific (small vessel) vasculopathy

"The principal alterations seen in PAH are Vaso construction, 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 Vaso constrictors and mitogens, such as endothelin 1" [1].

Yani-Landau et al found a 30% prevalence of antithyroglobulin antibodies in 40 patients with PPH. This is important because the likelihood of progression of subclinical hypothyroidism to clinical hypothyroidism increases if thyroid antibodies are present [4].

"In one retrospective study, the prevalence of hypo thyroidism in 41 patients with PAH (MPAP > 25 mmHg at rest, as estimated by right heart catheterization) was found to be 22.5% [5]. In another study, a sample of 356 patients with PAH and 698 gender-matched controls without PAH were retrospectively evaluated. Of the patients with PAH, 85 (24%) had thyroid disease, as did 107 (15%) of the controls. Most patients had mild thyroid disease, predominantly hypothyroidism" [6].

"The association between hyperthyroidism and pulmonary hypertension has been described since the early 1980s [7]. Nakchbandi et al suggested that pulmonary hypertension in a patient with hyper thyroidism was probably caused by a high cardiac output, endothelial damage/dysfunction, or increased metabolism of intrinsic pulmonary vasodilating substances. Also, acetylcholine which induces pulmonary vasodilator response, [8] plays a role. In the presence of hyperthyroidism, it is possible that the cholinergic output is decreased and the vasodilator response is diminished thus increasing pulmonary vascular resistance".

Some studies have shown the prevalence of PAH in patients with hyperthyroidism. In a study that evaluated patients recently diagnosed with hyperthyroidism, the prevalence of PAH was found to be 35% [9]. "In another study, involving 114 patients with hyperthyroidism (47 with Graves' disease and 67 with multinodular goiter), the prevalence of PAH was found to be 43% [10].

In these two studies, a diagnosis of PAH was made when PASP, as estimated by echocardiography, was > 30 mmHg. In other studies, a diagnosis of PAH was made when PASP was > 35 mmHg. In a study involving 39 consecutive patients recently diagnosed with hyper thyroidism, the prevalence of PAH was found to be 41 %" [11].

In another study, involving 23 patients with hyperthyroidism (Grave's disease or multinodular

goiter), the authors found a prevalence of PAH of 65% [12].

A study shows that treatment of patients with hyperthyroidism (Graves' disease and toxic multinodular goiter) with radioactive iodine or ethionamides has decreased mean PASP from 40 ± 11 mmHg to 25 ± 6 mmHg [13].

Materials and methods

Study population

Cases

A total of 50 patients visiting opd or being admitted in department of internal medicine ward having hyper thyroidism/ hypothyroidism at PMCH, Patna

Type of study: This is a cohort study.

Study period: This study was conducted from October 2020 to March 2022.

Inclusion criteria

Clinically screened patients with biochemical evidence of hypothyroidism or hyperthyroidism.

Exclusion criteria

Patients with/who are

1. Smokers
2. Taking anorectics, contraceptives, chemotherapeutics or vasoactive drugs
3. Clinical features of pulmonary diseases
4. Clinicals features and markers of connective tissue diseases
5. Underlying cardiac diseases like VSD, Cardio myopathies, Myocarditis etc.
6. Cirrhosis or chronic liver disease
7. Chronic hypoxemia
8. On treatment for hypothyroidism/hyperthyroidism
9. HIV

Result and discussion

Total 50 patients were enrolled in the study, of them 78 % patients had Hypothyroidism and remaining 22 % patients had hyperthyroidism. Average age of the patients with hypothyroidism was 45.48 yrs and that of patients with hyperthyroidism was 37.91 years.

"In a study by Marvisi M et al, that evaluated patients recently diagnosed with hyperthyroidism, the prevalence of PAH was found to be 35% [9]. In another study by Marvisi M et al, involving 114 patients with hyper thyroidism (47 with Graves' disease and 67 with multinodular goiter), the prevalence of PAH was found to be 43% [10]. In those two studies, a diagnosis of PAH was made when PASP, as estimated by echo cardio graphy, was > 30 mmHg".

"In other study by Mercé J et al of 39 consecutive patients recently diagnosed with hyperthyroidism, the prevalence of PAH was found to be 41% [11], a diagnosis of PAH was made when PASP, as estimated by echocardiography, was > 35 mmHg".

"In a retrospective study by Curnock AL et al, the prevalence of hypothyroidism in 41 patients with PAH (MPAP > 25 mmHg at rest, as estimated by right heart catheterization) was found to be 22.5% which was definitely higher than the incidence that we found in our study" [5].

"In a study by Li JH et al, a sample of 356 patients with pulmonary hypertension and 698 gender-matched controls without PAH were retrospectively evaluated. Of the patients with pulmonary hypertension, 85 (24%) had thyroid disease, as did 107 (15%) of the controls. Most patients had mild thyroid disease, predominantly hypothyroidism" [6].

The mean TSH, T3, T4, and PASP (pre-treatment) values among the hypothyroidism group's follow-up patients were 22, 18, 0, 5.6, and 36.20, respectively. Mean TSH, T3, T4, and PASP post-treatment levels

were 5.20, 0.99, 10.91, and 30.71, respectively.

After 18 months of follow-up, the mean TSH value fell, although the values of T3 and T4 had increased. Mean PASP value dropped in the follow-up 18 months after therapy as well.

The mean TSH, T3, T4, and PASP (pre-treatment) values among the follow-up patients in the hyperthyroidism group were 0.22, 2.28, 15.88, and 42.5, respectively. Mean TSH, T3, T4, and PASP post-treatment levels were 2.93, 1.14, 8.78, and 29.5, respectively.

While the values of T3 and T4 had reduced after 18 months of follow-up, the mean TSH value had increased. Mean PASP levels dropped in the 18-month follow-up following therapy as well. The results of other research's findings were comparable to the PASP change following the treatment.

Conclusion

Pulmonary hypertension was detected in both hypothyroidism group (20.51%) and in the hyperthyroidism group (27.27%). It was concluded that hypothyroidism/hyperthyroidism may cause pulmonary hypertension and Pulmonary hypertension can be reversed after correction of hypothyroidism/ hyper thyroidism.

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Legend Tables

Table 1: PASP comparison between pre- and post-treatment in the follow-up group

Variable	"Mean± Sd"		P value
	"Before"	"After follow up"	
"2D-ECHO PASP mm Hg"	37.72±5.49	31±5.20	0.001

Table 2: Comparison of pre- and post-treatment TSH, T3, T4, and PASP levels in hypothyroid patients in the follow-up group.

"Variable"	"Hypothyroidism (Mean± Sd)"		"P value"
	"Before"	"After follow up"	
"TSH (uIU/ml)"	22.18±20.41	5.20±1.63	0.01
"T3 (ng/ml)"	0.76±0.57	0.99±0.36	0.05
"T4 (ug/dl)"	5.55±3.33	10.91±1.48	0.004
"2D-ECHO PASP mm Hg"	36.20±4.20	30.71±5.8	0.006

Table 3: Comparison of TSH, T3, T4 and PASP among patients of Hyperthyroidism in the follow up group, pre and post treatment.

"Variable"	"Hyperthyroidism (Mean± Sd)"		"P value"
	"Before"	"After follow up"	
"TSH (uIU/ml)"	0.22±0.10	2.93±1.56	0.07
"T3 (ng/ml)"	2.28±0.54	1.14±0.38	0.018
"T4 (ug/dl)"	15.88±2.57	8.78±1.40	0.013
"2D-ECHO PASP mm Hg"	42.5±5.75	29.5±5.3	0.023